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DIAGNOSIS OF TIGHT MITRAL STENOSIS

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Recent advances in the surgical treatment of mitral stenosis have made the diagnosis of tight mitral stenosis important. Evidence is accumulating that mitral commissurotomy performed in patients with tight mitral stenosis is of benefit and can be performed with a comparatively low mortality in selected cases. Patients with mild mitral stenosis and with severe mitral insufficiency or aortic valvular lesions may present somewhat similar pictures, but would not be candidates for surgical treatment until such time as operative approaches to the correction of mitral insufficiency or aortic insufficiency and stenosis have proved their worth. Although there is a tendency, at present, to limit the operation to those patients who show tight mitral stenosis without significant associated lesions, it seems possible that correction of the mitral stenosis may be of value whenever it is severe, regardless of associated lesions. The purpose of this presentation is to describe the factors that are important in the diagnosis of tight mitral stenosis. Seventeen patients with this diagnosis were carefully studied clinically and by means of cardiac catheterization. These patients were subsequently operated on, and the presence of a tight mitral stenosis demonstrated. None of the mitral valves would admit the tip of the surgeon's index finger. Since the area of the finger tip was approximately 1 sq. cm., the valve areas were all less, often much less, than 1 sq. cm.

Other valvular lesions of minor degree were present in these patients but influenced the general picture little, if at all. Mitral insufficiency was frequently present but, in view of the small valve opening, was necessarily slight. Faint diastolic murmurs along the left border of the sternum were considered as indicating some aortic insufficiency, but no other evidences of this lesion were present. Some of these murmurs may have been due to pulmonary insufficiency. Tricuspid lesions were recognized in at least two cases and, in minor degree, may have been present in others. The important findings in these patients are shown in table 1 and are discussed below.

AUSCULTATION

The auscultatory findings (table 1, columns 4 through 8) are of major importance, and the diagnosis of tight mitral stenosis can be made in many cases on the basis of auscultation alone.

Murmurs.—For the diagnosis of mitral stenosis the characteristic mid-diastolic murmur, whether loud or faint, should be present. The murmur of mitral stenosis has the following characteristics:

1. It is best heard at the apex and when faint may be confined to a very small area. As the murmur becomes more intense, it is heard over a wider area; however, even when very intense, it may be confined to a relatively small area.
2. The murmur is best heard when the patient is recumbent and in the left lateral position. Even when it is very loud in this position, it may be heard poorly or not at all in the sitting position. Listening to a patient only in the sitting or standing position does not constitute a complete examination. When faint, the murmur may be best heard for several beats just after the patient has assumed the left lateral position or just after the patient returns to the recumbent position.
3. The murmur in its early stages is rather low-pitched and rumbling in character and therefore best heard with the bell of the stethoscope. To hear faint murmurs, one must hold the bell lightly against the skin because the intensity of the murmur is decreased by pressure. As the intensity of the murmur increases, it becomes harsher and may be heard equally well with the bell or the diaphragm.

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4. In the early stages the murmur of mitral stenosis is entirely mid-diastolic. As the stenosis progresses or the heart rate increases, the murmur, which begins in mid-diastole, now continues to the first heart sound, often with a presystolic accentuation. Rarely does a presystolic murmur occur without a mid-diastolic murmur. These cases must be regarded with suspicion, because various changes occurring about the time of the first heart sound may give the impression of a presystolic murmur. A faint mid-diastolic murmur associated with a loud presystolic murmur may indicate a valve that is moderately stenotic but not as tight as in those cases in which the murmur is loud from its onset in mid-diastole. In the presence of auricular fibrillation the duration of the murmur may be an index of the severity of the stenosis.

5. Tachycardia due to exertion or other causes makes the murmur more evident.

In tight mitral stenosis the mid-diastolic murmur is usually loud or moderately loud. Occasionally it may be faint, and rarely it may be absent. The position of the [illegible] onary emphysema may at

degrees of right heart failure. Thus a patient who has a moderately loud mid-diastolic murmur before failure occurs may have only a faint murmur with the onset of fibrillation and/or failure. In patients with known rheumatic heart disease who are in failure and do not have evidence of active rheumatic fever or a significant aortic valvular lesion to account for the failure, the diagnosis of tight mitral stenosis may tentatively be made on the basis of the other findings described below, even if the mid-diastolic murmur is faint.

When a loud mid-diastolic murmur is heard, the systolic murmur of mitral insufficiency is usually absent or faint, and only occasionally is it moderately loud. The murmur of slight or moderate mitral insufficiency is high-pitched and blowing and is best heard at the apex with the diaphragm of the stethoscope. If the diastolic murmur is loud and the systole is short, the systolic murmur may be difficult to hear. In general, it is true that the louder the systolic murmur the less intense the diastolic murmur, and vice versa. In the patients of our series the stenosis was so marked that there could not have been [illegible] fficiency, and in no instance was there a loud [illegible]

TABLE 1.—*Results of a Study of Seventeen Patients with Tight Mitral Stenosis*

			Findings on Auscultation					Roentgen Findings			Cardiac Output (Index)		Pulmonary Arterial Pressure			
Patient	Age	Sex	Mitral Diastolic Murmur *	Apical Systolic Murmur *	First Sound at Apex †	Second Pulmonic Sound †	Early Diastolic Murmur, Third Left Interspace *	Left Atrium ‡	Pulmonary Artery ‡	Left Ventricle ‡	Rest	Exercise	Rest	Exercise	Functional Status (Class) §	Hemoptysis ‖
F. B.	[illegible]	F	++++	++	N	A	0	+++	+	+	3.84 (2.46)	3.54 (2.27)	40/13 26	46/23 31	4	0
T. B.	[illegible]	M	++++	0	MA	N	0	++	+++	+	2.54 (1.39)	3.29 (1.80)	44/22 31	95/50 65	3	++++
D. A. B.	37	F	+++	++	A	..	0	++	++	++	3.80 (2.54)	5.40 (3.62)	58/26 42	109/73 87	3	0
M. D.	47	M	++ to +++	++	A	N	0	+++	++	N	3.40 (2.06)	4.44 (2.70)	40/16 24	48/24 32	3	0
E. J. D.	[illegible]	M	++++	0	MA	MA	++	+++	++	N	4.17 (2.24)	5.62 (3.06)	41/22 28	99/55 70	3	0
F. C. D.	[illegible]	M	+++++	++	N	A	++	++++	+++	+	2.87 (1.73)	3.42 (2.07)	53/24 37	102/54 70	4	+
N. D.	[illegible]	F	+++	++	A	A	0	++	++	N	3.41 (2.05)	3.68 (2.20)	64/24 33	132/48 76	3	++++
E. R. H.	[illegible]	F	++ to +++	++	N	N	0	+++	N	N	2.56 (1.64)	3.78 (2.42)	21/ 6 15	50/26 34	4	0
E. M.	[illegible]	F	+++	++	A	A	+++	+++	++++	+	....	....	90/42 60	....	3	++
E. L. P.	[illegible]	F	++++	++	A	N	++	+	+	+	3.26 (1.98)	3.60 (2.18)	48/18 29	60/38 45	3	++++
C. L. R.	[illegible]	M	+++	++	A	A	+	+++	++	N	2.87 (1.70)	3.97 (2.35)	48/22 33	83/58 66	3	+
G. R.	[illegible]	M	++++	+	A	N	0	++++	+++	N	4.74 (2.84)	5.18 (3.10)	54/31 40	83/47 62	4	++
H. S.	42	M	++++	++	MA	MA	++	+++	+++	N	4.10 (2.40)	3.71 (2.18)	72/32 42	90/46 64	4	+++
M. S.	[illegible]	F	++++	++	MA	MA	0	+++	+++	N	1.50 (1.08)	....	72/42 52	....	4	0
L. S.	[illegible]	F	+++	++	MA	A	0	+++	++	+	2.81 (1.84)	5.92 (3.87)	26/10 17	59/30 40	3	0
H. M. W.	[illegible]	F	++ to +++	+	N	MA	++	++	++	N	3.64 (2.32)	5.11 (3.25)	33/15 21	72/37 49	3	0
C. W.	26	M	++++	+	A	A	0	+++	++	+	4.10 (2.50)	3.90 (2.40)	58/28 40	84/44 60	3	+++

* No murmur, 0; very faint, +; faint, ++; moderately loud, +++; loud, ++++; very loud, +++++; extremely loud, ++++++.
† N, normal; A, accentuated; MA, markedly accentuated.
‡ Enlargement graded + to ++++. N, normal.
‖ Graded + to ++++ on the basis of number of episodes.
§ According to classification of the American Heart Association.

mild or moderate degree and the insufficiency is likely to be the more important lesion Since insufficiency is often aggravated by the operation, patients whose insufficiency is judged to be the more important lesion would not appear to be candidates for the operation at present.

The systolic murmur of tricuspid insufficiency often becomes evident as right heart failure develops and should not be confused with the murmur of mitral insufficiency. The murmur of tricuspid insufficiency is of maximum intensity just to the left of the lower end of the sternum and fades out laterally, so that it is usually faint at the point of maximum intensity of the mid-diastolic murmur, while the murmur of mitral insufficiency is loud at the apex and well heard laterally. When the tricuspid insufficiency is marked, cervical venous pulsation and liver pulsation may be of help in the diagnosis.

Many of the patients had a high-pitched, early diastolic murmur along the left border of the sternum, best heard with the diaphragm of the stethoscope. This murmur was usually considered to be indicative of aortic insufficiency, although in some instances it might have represented pulmonary insufficiency. This murmur, even when well heard at the apex, should not be confused with the murmur of mitral stenosis, because of the difference in timing, pitch, and points of maximum intensity.

Heart Sounds.—The intensity of the first heart sound is usually increased. It is occasionally diminished, but the presence of a markedly diminished first heart sound should be regarded with suspicion, for it is more likely to occur when mitral insufficiency is the important lesion. Accentuation of the second heart sound in the pulmonic area is of importance as an indication of increased pressure in the pulmonary artery. However, accentuation of the pulmonic second sound does not invariably occur when pulmonary arterial hypertension is present. It may be absent because of the position of the artery, the presence of pulmonary emphysema, or other causes. The opening click of the mitral valve was present in several of our patients, but its value as a diagnostic sign for tight mitral stenosis could not be evaluated.

FLUOROSCOPIC AND ROENTGENOGRAPHIC OBSERVATIONS

Next in importance to the auscultatory findings in the diagnosis of tight mitral stenosis is the roentgenologic evidence of the presence of left auricular enlargement and the absence of left ventricular enlargement. Left auricular enlargement was marked in almost every case and was well seen in both the posteroanterior view and in the left lateral esophogram. It was well seen during fluoroscopic examination when the points of opposite pulsation along the left cardiac contour could easily be determined. If orthodiascopic study is made, the exact length of the auricular segment may be determined and a measure of the degree of left auricular enlargement obtained. This segment normally does not exceed 2.2 cm. in length[1]; in our patients it was often 4.5 to 5 cm. in length. The enlargement of the left auricle is of importance as an indication of the degree of mitral stenosis only when taken in conjunction with the other criteria, especially those that rule out marked mitral insufficiency. Although the degree of auricular enlargement in mitral stenosis may often be pronounced, similar degrees of enlargement may occur in mitral insufficiency and the most pronounced enlargement is often seen in patients with fairly severe mitral insufficiency associated with moderate stenosis.

The left ventricle (as judged by cardiac contour) was considered to be not enlarged in nine cases and possibly slightly enlarged in eight cases. The absence of left ventricular enlargement is reflected in the absence of pronounced increases in transverse diameter in a group of patients all showing evidence of advanced heart disease. The cardiothoracic ratio was 50% or greater in only 10 patients. The transverse diameter of the heart was greater than the transverse diameter predicted on the basis of height and weight[2] in 16 patients, and the average increase was 25%. Although the absence of left ventricular enlargement is a factor against severe mitral insufficiency, its presence could at times be due to aortic valvular lesions or arterial hypertension.

The pulmonary artery and its major branches were considered to be enlarged in almost every instance. The prominence of the pulmonary artery served as a rough index of the pulmonary arterial pressure, since patients with the most prominent arteries had the highest pulmonary arterial pressures. As a result of the increased prominence of the left auricular appendage and pulmonary artery, all the patients showed straightening or even bulging of the left cardiac contour, the so-called mitral configuration.

Enlargement of the right ventricle was considered to be present in a majority of instances but was much more difficult to assess. The lung fields showed increased vascularity in all but two cases, with prominence of the vascular pattern, nodular densities, and, in several instances, multiple small calcifications.

ELECTROCARDIOGRAPHIC OBSERVATIONS

Since in the course of mitral stenosis an increased pressure develops in the left auricle and later in the right ventricle, these chambers of the heart often show hypertrophy and enlargement. In 16 patients we made a detailed study of the electrocardiogram in order to determine its value in the detection of these changes.

Left auricular hypertrophy and enlargement may be evidenced by P wave changes or by the presence of auricular fibrillation, for auricular fibrillation in rheumatic heart disease usually indicates the presence of a large left auricle under strain. Auricular fibrillation was present in 7 of the 16 electrocardiograms, and there was one instance of persistent auricular flutter. P wave changes occurred in all the remaining eight patients, with regular rhythm. These changes consisted of broad notched P waves, most clearly seen in leads 1 and 2. Increased amplitude of the P waves occurred at times but was not striking. This is in accord with the observation that tall P waves are more likely to occur with right auricular hypertrophy than with left.[3] The finding of either P wave changes or auricular fibrillation is in accord with the roentgenographic observation of enlargement of the left auricle in all 16 cases.

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[1] Kurtz, C. M.: Orthodiascopy: An Analysis of over Seventeen Hundred Cases, New York, The Macmillan Company, 1937.

[2] From the table of Ungerleider and Clark.

[3] Berliner, K., and Master, A. M.: Mitral Stenosis: A Correlation of Electrocardiographic and Pathologic Observations, Arch. Int. Med. 61: 39 (Jan.) 1938.

Electrocardiographic criteria for right ventricular hypertrophy have been defined by Sokolow and Lyon,[4] who used the unipolar and precordial limb leads, and by Schack, Rosenman, and Katz,[5] who employed only the unipolar limb leads. These criteria are listed in table 2, together with the data obtained from the electrocardiograms of our patients. A, B, C, and D are the criteria given by Sokolow. Under A are given the measurements for the voltages of the R and S waves in the precordial and unipolar limb leads and the various sums and ratios of these voltages. It can be seen that in only a few of our cases was the presence of abnormalities shown in these measurements, the greatest number being 5 patients out of the 16 who showed an R:S ratio in V_1 greater than 1 (criterion A-9). Four of these five patients also showed delayed ventricular activation time in V_1 or V_2 (criterion B). Changes in the RS-T segment and T waves occurred in only two (criterion C).

TABLE 2.—*Evidence of Right Ventricular Hypertrophy in Patients with Mitral Stenosis*

	Total	
	+	0
A. Voltage of R and S waves		
1. R wave in V_1 7 mm. or greater	3	13
2. S wave in V_1 less than 2 mm.	3	13
3. S wave in V_5 or V_6 7 mm. or greater	3	13
4. R in V_1 + S in V_6 greater than 10.5 mm.	3	13
5. R in V_6 less than 5 mm.	0	16
6. Ratio of R:S in V_6 = 1 or less	2	14
7. R in aV_r = 5 mm. or more	0	16
8. $\frac{\text{Ratio R:S in } V_5}{\text{Ratio R:S in } V_1}$ = .4 or less	4	12
9. Ratio R:S in V_1 greater than 1	5	11
B. Delayed ventricular activation time between 0.04 — 0.07 sec.		
In V_1	3	13
In V_2	4	12
C. Depressed RS-T and inversion of the T wave		
In V_1	1	15
In V_2	3	13
In V_3	2	14
In aV_l	0	16
In aV_f	0	16
D. Right axis deviation greater than + 110 degrees or more	2	..
E. Sum of ratios R:S in aV_l and aV_f Less than 13	3	13
Ratio R:S in aV_r greater than .7	3	13
Total voltages in aV_r, aV_l and aV_f greater than 42	1	15

No. of positive criteria shown by a given patient	0	1	3	7	10	12
No. of patients showing this no. of criteria	5	6	1	2	1	1

The changes in the unipolar limb leads in right ventricular hypertrophy given by Schack, Rosenman, and Katz[5] are shown in table 2 (criterion E). Only six patients showed abnormal values in any one of the criteria presented by Schack and his co-workers, no patient having more than one abnormal value.

The electrocardiographic position of the heart in these patients was vertical in 11, semi-vertical in 2, and intermediate in 3. There was accordingly right axis deviation (axis greater than +90 degrees) in six patients, and a tendency to right axis deviation (+80 to +90 degrees) in three patients. The remaining seven showed a normal axis. Although a vertical electrocardiographic position of the heart is of no significance when the heart size is normal, its occurrence in patients with cardiac disease and enlarged hearts is much more significant. In mitral stenosis, whether as a result of greater electrical potentials arising from the hypertrophied right ventricle or as a result of changes in the anatomic position of the heart, the electrocardiographic position of the heart tends to be vertical. In the case in which the enlargement of the heart is mostly left ventricular, the electrocardiographic position tends to be horizontal. The occurrence of a horizontal electrocardiographic position of the heart and corresponding left axis deviation suggests the presence and functional importance of mitral insufficiency or aortic valvular lesions rather than tight mitral stenosis.

Table 2 shows that in only two patients did the electrocardiogram exhibit abnormalities in over one-half of the criteria that are said to be of value in the detection of right ventricular hypertrophy. Only four (25%) of the patients had abnormalities in five or more of the criteria. One must assume either that very little right ventricular hypertrophy existed in these patients or that the electrocardiogram is of limited value in the detection of right ventricular hypertrophy until it is severe.

Postmortem findings in two patients showed right ventricular walls 4 to 6 mm. and 5 to 6 mm., respectively, in thickness, approximately twice that of the normal heart; yet the electrocardiograms met only 3 and 1, respectively, of the 20 criteria. All these patients had a long history of rheumatic heart disease with mitral stenosis, and the mean pulmonary arterial pressures were increased in all but three at rest and in all during mild exercise. It is reasonable, therefore, to conclude that right ventricular hypertrophy existed but that it apparently has to be severe in order to be detectable by electrocardiography.

CLINICAL DATA

Functional Status.—The occurrence of no limitation or slight limitation of physical activity would seem unlikely in the presence of a tight mitral stenosis of the type considered here. Although these patients get along remarkably well at rest, the inability to increase the cardiac output to any significant degree prevents their doing well on exertion. Some of them may for a while be able to increase their cardiac output by rather pronounced increases in pulmonary arterial pressure and thus apparently are in class 2.[6] They will, however, show a gradual deterioration in their ability to do physical activity and soon are clearly in class 3, that is, less than ordinary physical activity will cause discomfort.

Many patients with mitral stenosis get along well for years, for example, women who bear many children and patients who lead active lives and die at an advanced age of unrelated causes. These patients usually have mitral stenosis of only moderate degree. A valve area of 1.5 sq. cm. or more permits a cardiac output compatible with a rather active life.[7] Such valve areas are considerably less than the normal 4 to 6 sq. cm., yet greater than the mitral valve areas of our patients.

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[4] Sokolow, M., and Lyon, T. P.: The Ventricular Complex in Right Ventricular Hypertrophy as Obtained by Unipolar Precordial and Limb Leads, Am. Heart J. 38: 273 (Aug.) 1949.

[5] Schack, J. A.; Rosenman, R. H., and Katz, L. N.: The aV Limb Leads in the Diagnosis of Ventricular Strain, Am. Heart J. 40: 696 (Nov.) 1950.

rt Association classification.

Attacks of Paroxysmal Auricular Fibrillation or Tachycardia.—Although these occur in many types of cardiac disease, patients with mitral stenosis and enlarged left auricles are particularly prone to have them and are often more ill with these attacks than are other cardiac patients. Persons with mitral stenosis tolerate tachycardia very poorly, because of their dependence on a diastolic filling period adequate for left ventricular filling. As the tachycardia shortens diastole, left ventricular filling decreases markedly and the cardiac output, low initially, falls to even lower levels. It is not uncommon for marked hypotension or anginal pain to develop during these attacks in such patients.

Episodes of Pulmonary Edema and Congestive Failure.—In a patient with the auscultatory findings of mitral stenosis and with only moderate cardiac enlargement, the occurrence of episodes of pulmonary edema or congestive failure suggests that the mitral stenosis is tight. These episodes are less significant when there is pronounced cardiac enlargement. Pulmonary edema is often precipitated by tachycardia.

Hemoptysis.—Hemoptysis is a classical sign of mitral stenosis, but its value as an index of the severity of mitral stenosis depends on the size of the heart. Hemoptysis in a patient with a heart that is only moderately enlarged suggests the presence of a tight mitral stenosis. Arterial embolization is not necessarily suggestive of tight mitral stenosis.

CARDIAC CATHETERIZATION

This procedure is widely used at present, and its importance in selection of patients for mitral commissurotomy has been emphasized. Valuable physiological data are certainly obtained by catheterization, for example, the cardiac output and index at rest and during exercise, the pulmonary arterial pressures at rest and during exercise, and the pulmonary "capillary" pressure. It is our opinion, however, that the information obtained from cardiac catheterization is of limited value in the determination of whether a tight mitral stenosis is present. Where the diagnosis is in doubt before catheterization, it is rarely made by catheterization.

Cardiac Output.—The resting cardiac output and cardiac index are decreased, often markedly so, in tight mitral stenosis. The average cardiac index in our patients was 2.05. Inasmuch as two factors, the mitral stenosis and cardiac decompensation, may play a role in decreasing cardiac output, the relative importance of each factor must be assessed in any given case. When the heart is of nearly normal size and cardiac decompensation is not present to a significant degree, the cardiac output may reflect the severity of the mitral stenosis.

The resting cardiac output in patients with mitral insufficiency is also reduced and has in fact been reported to be lower than in those with mitral stenosis.[8] However, decrease in cardiac output due to cardiac decompensation may have been involved.

These patients show an inability to increase significantly the cardiac output during exercise. Whatever increase does result is due to a decided elevation of pulmonary arterial pressure. Tachycardia, by decreasing the diastolic filling period, tends to decrease the cardiac output. This effect may outweigh the effect of increased pulmonary arterial pressure, resulting in a decreased output during exercise. Inability to increase cardiac output is also present in patients with mitral insufficiency and in patients with cardiac decompensation due to other causes.

Pulmonary Arterial Pressure.—The pulmonary arterial pressure is usually increased at rest but may be within normal limits even in the presence of tight mitral stenosis with diminished cardiac output. Apparently patients show various methods of compensating for their handicap; in some the output is maintained by raising of the pulmonary arterial pressure, while others manage surprisingly well on very low cardiac outputs and low pulmonary arterial pressures.

The pulmonary arterial pressure rises rapidly and severely with slight exertion, the systolic pressure frequently doubling. The pressure increases shown in column 15 in table 1 resulted from exertion that was usually very mild, because of the functional status of the patients. An increase in pulmonary arterial pressure during exertion occurs in the presence of various cardiac conditions and is not diagnostic of mitral stenosis.

Pulmonary "Capillary" Pressure.—When the tip of the catheter is advanced through the pulmonary arterial tree until it occludes a branch, the pressure that may then be recorded has been shown to be a close approximation of the pulmonary capillary pressure.[9] This pressure also bears a definite relationship to left auricular and pulmonary venous pressures. Evidence of mitral regurgitation when this is severe may at times be seen in the pulmonary "capillary" pressure tracing. Such evidence would be of value in excluding the diagnosis of tight mitral stenosis.

Calculation of Mitral Valve Area by Formulas.—Gorlin and Gorlin have presented a formula for calculation of the mitral valve area in cases of mitral stenosis.[7] This formula states that the mitral valve area in square centimeters is equal to

$$\frac{\text{mitral valve flow in cubic centimeters per second}}{31\sqrt{\text{"PC"} - 5}}$$

where "PC" is the pulmonary "capillary" pressure. Mitral valve flow in cubic centimeters per second is calculated by dividing the cardiac output in cubic centimeters per minute by the diastolic filling period in seconds per minute. The diastolic filling period is estimated from the brachial arterial pressure-pulse contours. These authors find that the valve areas calculated from this formula correlate very well with the valve areas found at operation or autopsy.

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[7] Gorlin, R., and Gorlin, S. G.: Hydraulic Formula for Calculation of the Area of the Stenotic Mitral Valve, Other Cardiac Valves, and Central Circulatory Shunts, Am. Heart J. 41: 1 (Jan.) 1951.

[8] Draper, A., and others: Physiologic Studies in Mitral Valvular Disease, Circulation 3: 531 (April) 1951.

[9] Hellems, H. K.; Haynes, F. W., and Dexter, L.: Pulmonary "Capillary" Pressure in Man, J. Appl. Physiol. 2: 24 (July) 1949.

To derive their formula, Gorlin and Gorlin made a number of assumptions, some of which probably introduce little error, while others may introduce errors of a magnitude sufficiently great to make the formula of questionable value. By assuming a fixed value for "PC" and using cardiac output instead of mitral valve flow, a simplified formula may be derived that requires only the resting cardiac output. Thus,

$$\text{mitral valve area} = \frac{8 + (\text{C.O.} - 3)\,5}{14}$$

where C.O. is resting cardiac output in liters per minute.

In six of our patients in whom the pulmonary "capillary" pressure was determined, the mitral valve areas by the formula of Gorlin and Gorlin were calculated as 0.5, 0.7, 0.7, 0.9, 0.6, and 0.8 sq. cm. With the use of the simplified formula given above, the corresponding values were 0.5, 0.5, 0.7, 1.0, 0.4, and 0.8. The similarity is striking. Recalculating the data of Gorlin and Gorlin by means of the simplified formula given above, one finds that in 16 of their 21 cases the simplified formula gives a value within 0.2 sq. cm. of the value obtained with their formula.

The purpose of these comparisons is not to propose another formula for calculation of the mitral valve area, but rather to point out, first, that it may be incorrect to make several broad assumptions and then employ measurements that are too fine, and, second, that the important variable in the formula is apparently the cardiac output.

AMYL NITRITE AND THE VALSALVA TEST

Amyl nitrite inhalation has been suggested as a test for tight mitral stenosis. The test depends on the production of tachycardia, which, as has been mentioned, these patients tolerate poorly. Syncope will, therefore, frequently follow amyl nitrite inhalation in patients with mitral stenosis. Without other data the test, of course, means nothing. If the diagnosis is definite before the test is done, the test may be of a little value in confirmation.

decided rise in systemic arterial pressure, which lts in bradycardia, normally follows the Valsalva maneuver. This post-Valsalva hypertension depends in part on the increased venous return and cardiac output following the fall of the intrathoracic pressure to normal. Since the output of the left ventricle cannot increase to any degree in patients with tight mitral stenosis, the rise in arterial pressure and the resulting bradycardia do not occur or tend to be less pronounced. The Valsalva maneuver has therefore been suggested as a test for tight mitral stenosis.[10] Further study has shown that post-Valsalva failure of the arterial pressure to increase and failure of bradycardia to develop may be seen in other conditions and that the test has no great or specific value.

SUMMARY

The diagnosis of tight mitral stenosis can be made clinically on the basis of the following findings:

1. The presence of the typical diastolic murmur of mitral stenosis. This murmur is usually loud or moderately loud but may occasionally be faint.

2. The absence of a loud apical systolic murmur, indicative of mitral insufficiency. A faint systolic murmur is frequently present, and occasionally there may be a moderately loud systolic murmur.

3. The presence of an enlarged left atrium. This is usually evident on fluoroscopic or roentgenographic examination. It is also evident in the electrocardiogram as a change in the P waves or by the presence of auricular fibrillation.

4. The absence of left ventricular enlargement. This is determined by the fluoroscopy and roentgenogram. The absence of a horizontal electrocardiographic position of the heart and left axis deviation is corroborative. In the presence of a significant aortic valvular lesion or longstanding hypertension, there may be some left ventricular enlargement.

In the presence of the above findings, valuable corroborative findings are (1) a functional status of class 3 or 4, (2) evidence of right ventricular hypertrophy on fluoroscopic examination or in the roentgenogram or the electrocardiogram, and (3) episodes of pulmonary edema, congestive failure, or hemoptysis in the presence of a heart of only moderately increased size. Cardiac catheterization furnishes interesting data but is usually of little value in the diagnosis of tight mitral stenosis.

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[10] Goldberg, H.; Elisberg, E. I., and Katz, L. N.: The Effects of the Valsalva-Like Maneuver upon the Circulation in Normal Individuals and Patients with Mitral Stenosis, Circulation 5:38 (Jan.) 1952.

Presymptomatic Medicine.—Screening as a routine function of practicing physicians represents a departure in activities to which the profession as a whole has not completely oriented itself. . . . For centuries the public has looked to the medical profession for the cure of diseases and the relief of suffering, to tax-supported agencies for sanitation and preventive medicine. The search for diseases in their earliest stages is an extension of preventive medicine toward medical practice, or an extension of medical practice toward preventive medicine—dependent upon viewpoint. It represents a new form of activity which might be termed "diagnostic preventive medicine," "preventive diagnosis," or "presymptomatic medicine." The field of presymptomatic medicine has not been sharply delineated as a practical challenge for physicians. . . .

Adequate "screening" by the medical profession has been hampered (*a*) by the traditional role of physicians as healers, (*b*) by economic factors, (*c*) by lack of interest in the "well patient," (*d*) by the low yield of mass screening technics, (*e*) by the faulty logic of medical professors who have engendered confusion between "screening" and the diagnosis of a presenting complaint, and (*f*) by the let-George-do-it philosophy of therapeutic specialists. If "screening" is to be carried out in routine medical practice, the entire profession must face the issue, and each physician must be prepared to carry his share of the burden. Moreover, it is essential that the medical profession as a whole develop a new attitude toward each patient, (*a*) as an individual with a chief complaint, and (*b*) as a person to be "screened" for unsuspected chronic diseases. In his time-honored role as healer the physician must listen intently to the chief complaint of the sufferer and take whatever steps historical, physical or laboratory are required to arrive at the proper diagnosis. As a practitioner of presymptomatic medicine he must carry out such additional steps as are needed to exclude a reasonable likelihood of the existence of any threatening entity.—Benjamin V. White, M.D., Practical Diagnosis in Preventive Geriatrics, *Geriatrics*, April, 1952.

COMMISSUROTOMY FOR MITRAL STENOSIS

TECHNIQUE FOR PREVENTION OF CEREBRAL COMPLICATIONS

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One of the most tragic complications occurring in a case of mitral stenosis is cerebral embolization. In those cases in which the condition does not prove fatal, there is usually a long period of invalidism and not infrequently a permanent residual paralysis or personality change. With present generally accepted medical management, such vascular accidents are not completely avoidable, especially in cases with associated auricular fibrillation. The daily use of anticoagulants such as bishydroxycoumarin (dicumarol®) to maintain a persistent state of relative incoagulability of the circulating blood is an incomplete and none too satisfactory method of protection against embolization in cases of mitral stenosis. The patient is thereby converted into a "bleeder," with the constant risk of serious hemorrhage should he sustain a slight wound from any cause.

Logically, the best protective measure against the formation of auricular thrombi and the occurrence of embolization is the early surgical attack on the ultimate causal factor, mitral stenosis. Operation in the early stages of the disease would eliminate the possibility of blood stagnation, and the common site of thrombus formation, the atrial appendage, would be obliterated. There will probably be a considerable lapse of time before the medical profession generally accepts the philosophy of aggressive surgical attack in what has formerly been considered a chronic medical illness. Until the time comes when early surgery is universally accepted, we shall continue to have the problem of the thrombosed auricular appendage and its resulting emboli and must continue in our efforts to solve it by presently acceptable means.

Theoretically, amputation or ligation of the thrombosed auricular appendage would prevent peripheral emboli. Experimentally, this was proved to be an adequate solution by Hellerstein, Sinaiko, and Dolgin.[1] To us, this solution appears to be an oversimplification. While the majority of fresh, soft thrombi are located entirely within the left auricular appendage, there are many cases in which long streamers or eel-like extensions of the thrombi extend well into the main auricular cavity. These could easily be dislodged during ligation or amputation of the auricular appendage. The commoner type of thrombus encountered in the auricular appendage at operation is firm, well organized, and attached securely to the trabeculae and to other irregularities within the lumen. It is not unusual in such cases to find an extension of this organized thrombus in the form of a large flat mass, coating or lining the lateral wall of the auricle to which it is firmly adherent. The entire thrombus then assumes the shape of a mushroom, with the stem in the auricular appendage. While it would appear unlikely that embolization could be produced by such an old well-organized thrombus, auricular appendectomy has been practiced for such. In some cases a considerable portion of the auricular lumen is filled with a large thrombus, perhaps poorly organized (fig. 1).

When arterial embolization occurs during the course of medical treatment of mitral stenosis, it is generally accepted as unavoidable by the patient, the family, and the physician. However, when it occurs during or immediately after commissurotomy, it is inevitably and correctly attributed to surgical intervention. Commissurotomy can never attain general and complete acceptance until this complication is largely eliminated.

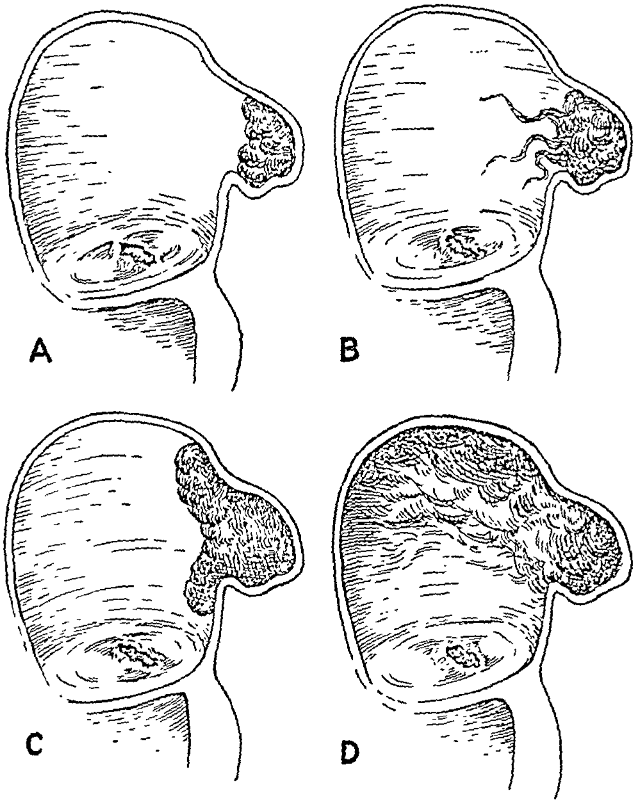

Fig. 1.—Thrombosis of the auricular appendage. *A*, limited to the appendage. *B*, associated with eel-like streamers of thrombus floating in the true auricular cavity. *C*, mushroom-like thrombus of appendage with cap lining the auricular wall (usually well organized). *D*, extensive intra-auricular thrombosis with varying degrees of organization.

While a large portion of arterial emboli occurring during medical treatment for mitral stenosis lodge in vessels of the extremities and abdominal viscera, we have seen no such localization occur in a series of 235 surgically treated patients. However, we know of several cases of arterial embolization to the lower extremities or to the bifurcation of the aorta occurring in the practice of other operators during the performance of commissurotomy.

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[1] Hellerstein, H. K.; Sinaiko, E. S., and Dolgin, M.: Amputation of Canine Atrial Appendages, Proc. Soc. Exper. Biol. & Med. 66:337 (Nov.) 1947.

This complication is evidently less common than cerebral embolism occurring during surgery, is a less serious threat to life and health, and is more easily treated. In this series, there were 12 instances of cerebral embolization. We do not wish to imply that most emboli released during the maneuvers of commissurotomy fail to traverse the aortic arch to enter the vessels beyond. Rather, we think that many of the probably more frequent emboli that do pass the aortic arch are of such small size that they usually remain unnoticed when they lodge peripherally. Particles of the same size coming to rest in the cerebral arterial pathways quickly make their effects evident.

It should be mentioned that thrombotic material in the atrium or its appendage is not the only source of these emboli. Some of them may be small pieces of calcium dislodged from the valve during commissurotomy. This calcium may exist as loosely attached sand-like fragments on the edges of the valve orifice, as a block-like mass at either commissure, or as a general infiltration of the valve substance. We feel that such calcium fragments were responsible for the embolism in at least two cases in this series. In one, the calcium was actually demonstrated in a cerebral artery at autopsy (fig. 2). Our difficulties because of cerebral embolization, occurring in 5.1% of 235 cases in which commissurotomy was done and contributing to a sizable proportion of our mortality and morbidity, have led us to undertake a study of the problem in regard to its avoidance and treatment.

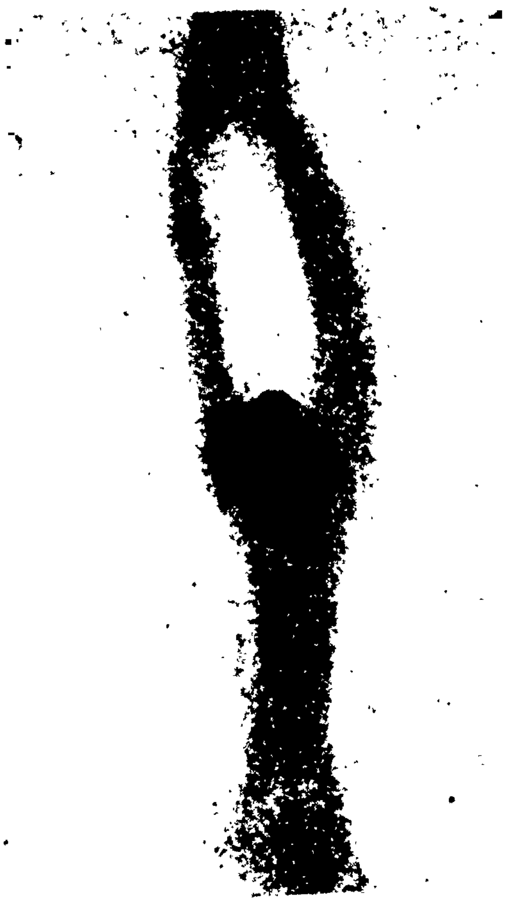

Fig. 2.—Cerebral embolism due to dislodgment of a bit of calicification from the stenotic valve orifice. Roentgenographic demonstration of calcium in brain post mortem (enlarged 4.7 diameters).

Of the 12 patients with this complication, 5 presented evidence of predominant involvement of the right side of the brain and 7 of the left. Three had bilateral involvement. All 12 patients presented definite neurological changes indicative of cerebral embolism, although 6 did not regain full consciousness postoperatively. The majority had signs and symptoms that suggested involvement of the middle cerebral artery or its branches. There was hemiplegia in nine cases and partial quadriplegia in three. There were eight deaths among the 12 cases, a mortality of 66.6%. In each case, we felt that death was directly related to this complication.

LOCALIZATION

The localization of the cerebral involvement corresponds essentially to that disclosed experimentally by Cone and Barrera.[2] The reason for this frequent involvement is not explained but may be assumed to be due to the more direct flow of blood into the middle cerebral artery than into the anterior or posterior artery. An embolus floating into the vertebral artery, if small, might well pass into one of the many branches of the basilar end and cause no trouble. If large enough to occlude the vertebral or basilar artery, death would rapidly ensue.

PATHOLOGICAL CHANGES

The pathological changes in the brains of the patients who were examined post mortem were extensive areas of softening with small hemorrhages interspersed. In no patient had the hemorrhagic areas coalesced to form a large discrete hematoma. The portion of brain involved in these middle cerebral emboli was typical. It demonstrated the reason for the more pronounced involvement of the arm, and, when the dominant hemisphere was affected, for the severe speech disturbance.

SYMPTOMS AND SIGNS

From a review of the anesthesia records we found that, during the course of surgery and anesthesia, the only early positive finding suggesting the incidence of cerebral embolization was a severe drop in systolic and diastolic blood pressures that rapidly (in 5 to 10 minutes) returned to a normal value for the patient during surgery.

It was the aim of the anesthesiologist to have the patients sufficiently awake at the completion of surgery so that simple commands for movement of the extremities might be obeyed. Therefore, prolongation of the unconscious state immediately after surgery, partial or complete paresis, aphasia, and pathological reflexes (Babinski sign and ankle clonus) were used for the clinical diagnosis of cerebral embolization. When seen shortly thereafter by the neurosurgeon, the patient usually had flaccid hemiplegia, with no motion in the arm and only flexor reflexes in the leg on painful stimulation. There was usually pronounced central facial weakness.

The diagnosis was established clinically in all cases. In three (25%), a spinal puncture was done to rule out the presence of intracranial bleeding. The pressure was found to be at the upper limits of normal. Death occurred in one case within five hours, and in the remaining fatal cases the patient survived 1, 1, 2, 5, 8, 40, and 53 days, respectively. In the last case, terminal subacute bacterial endocarditis developed.

Among the four patients who recovered, none became completely well. The use of the leg became essentially normal in all. The arm was definitely impaired, but still partially useful in all. The speech function became adequate, but impaired, in three. The fourth patient at no time exhibited aphasia (left-sided paresis).

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[2] Cone, W., and Barrera, S. E.: Brain and Cerebrospinal Fluid in Acute Aseptic Cerebral Embolism: Experimental and Pathologic Study, Arch. Neurol. & Psychiat. 25: 523-547 (March) 1931.

Our therapeutic efforts consisted, in the main, of the use of stellate ganglion blocks. Systemic heparinization was also carried out in one case, but the patient succumbed in 30 hours owing to rupture of a fulminating ulcer of the stomach. This case is to be reported in detail elsewhere.[3] The thought was entertained that it might be possible to pass a small catheter up the internal carotid artery and through it to aspirate the obstructing material. This proved impractical by cadaver studies, because of the S-shaped curve of the vessel before it communicates with the circle of Willis. It was further interdicted by the demonstration of Harvey and Rasmussen [4] that 30 minutes of ischemia produces irreparable brain damage.

Stellate ganglion blocks [5] were done if the patient was (1) unable to move all extremities, (2) aphasic, or (3) unconscious and unable to respond to painful stimuli. Either the anterior approach of Ochsner and DeBakey [6] or the anterolateral technique of Volpitto and Risteen [7] was used in each case. Horner's syndrome and/or warming of the upper extremity of the side on which the injection was made (opposite to the side of the paresis) constituted evidence of a satisfactory block. It became apparent early that, for adequate inhibition of sympathetic impulses from the stellate ganglion, the block would have to be repeated every two to four hours. To simplify the procedure, we adopted the following techniques.[8] A no. 15 Tuohy needle was inserted in the proper position, and through it a no. 4 radiopaque urethral catheter with stylet was threaded until it met resistance. The needle was then removed, leaving in place the catheter, which was secured by narrow strips of adhesive or by nonabsorbable suture. After removal of the stylet, a blunt no. 23 Leur-Lok needle was inserted into the distal end of the catheter. Five cubic centimeters of 1% intracaine® (beta-diethylaminoethyl-*p*-ethoxy benzoate hydrochloride) solution was then injected.

If a Horner's syndrome was not obtained within five minutes, administration of the dose was repeated. Failure of the second injection to inhibit sympathetic activity called for a bedside roentgenogram so that we might determine whether the catheter tip was in the proper position. If not, it was withdrawn and reinserted in the same manner and a roentgenogram was again taken. If the position was satisfactory, a three-way stopcock was attached to the no. 23 Leur-Lok needle. Connected to the stopcock were a flask containing 1% intracaine® and a 10 cc. Leur-Lok syringe. This allowed withdrawal of 5 to 10 cc. of the solution into the syringe for injection through the catheter every two to four hours as required to maintain the desired effect.

In addition to the ganglion blocks, an occasional dehydration regimen, mild sedation, and vasodilators were used. Eight of the patients were treated by stellate ganglion block on the side of the cerebral lesion. Two patients received one such block, four received multiple blocks at intervals of two to four hours, and two received continuous block. The mortality in the group treated by block was 62.5%, and that of the untreated group was 75%. However, it was our clinical impression that more rapid and satisfactory recovery from paralysis occurred in those treated by block than in those not so treated. It was obvious, nevertheless, that this form of therapy was not satisfactory, and our attention was therefore turned to the possible prevention of the complication.

PREVENTIVE MEASURES

Since most of the emboli were presumed to be related to dislodgment of the thrombotic material in the auricular appendage as the operator's finger was inserted into the left auricle, an attempt was first made to recognize thrombosis of the appendage before insertion of the finger. Then we thought it possible, after carefully clamping the appendageal base, to remove the thrombus from the excluded appendageal lumen. By intermittently releasing the basal clamp, we would provide a rapid gush of blood to expel any residual bits of loosened material. This technique has at times seemed to be highly effective. However, it is open to the objection that the clot may initially extend well into the auricular cavity and may, therefore, be divided by the pressure of the basal clamp. Also, some thrombi are completely or partially organized and well attached, so that they are not easily or completely extractable from the appendage or auricle. Having observed cerebral embolism develop in two patients when thrombi had apparently been completely removed from the appendage, we reluctantly abandoned this technique. We now feel that it is often impossible to recognize thrombosis in the auricular appendage before opening the structure.

Our next attempt was to devise an entrance into the left auricle that would avoid the thrombosed appendage. Such possible sites of entrance are provided by an incision in the auricular wall itself or by an incision in the wall of the left superior pulmonary vein. Entrance through the auricular wall, even though guarded by a purse-string suture about the area, carries the inherent risk of uncontrollable hemorrhage. The sometimes friable auricular wall may be easily lacerated by a too tight purse-string or mattress suture. Therefore, we have used this approach on only three occasions, fortunately without serious incident.

Entrance into the left auricle by way of the left superior pulmonary vein has been used on 21 occasions. There have been two deaths in this series, both of which could be considered as due to the choice of this method of entering the heart. This represents a mortality of 9.5%. While all 21 patients presented gross thrombosis of the auricular appendage and presumably would have had a high incidence of cerebral embolization, it is questionable whether we can justify entrance via the vein on the basis of these statistics. However, both deaths occurred relatively early in our use of vein approaches, and it is noteworthy that there were no instances of brain embolism in this series, in which there was undoubted predisposition to embolism.

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[3] Bolton, H.; Massey, F., and Maniglio, R.: To be published.

[4] Harvey, J., and Rasmussen, T.: Occlusion of the Middle Cerebral Artery, A. M. A. Arch. Neurol. & Psychiat. **66**: 20-29 (July) 1951.

[5] Volpitto, P. P., and Risteen, W. A.: The Use of Stellate Ganglion Block in Cerebral Vascular Occlusions, Anesthesiology **4**: 403-408 (July) 1943.

[6] Ochsner, A., and DeBakey, M.: Thrombophlebitis: Role of Vasospasm in Production of Clinical Manifestations, J. A. M. A. **114**: 117-123 (Jan. 13) 1940.

[7] Volpitto, P. P., and Risteen, W. A.: Stellate Ganglion Block: A Definite Anterolateral Approach, Anesthesiology **5**: 491-494 (Sept.) 1944.

[8] Tuohy, E. B.: Continuous Spinal Anesthesia: A New Method Utilizing a Urethral Catheter, Surg. Clin. North America **25**: 834-840 (Aug.) 1945.

The disadvantages of the auricular approach via the left superior pulmonary vein may be summarized as follows.

1. The finger is 2 to 4 cm. farther from the mitral valve than when entrance is effected through the auricle. This makes both the digital pressure technique and instrumental division of the valve technically more difficult and more likely to be imperfectly performed.

2. The vein is often insufficiently large to admit the average operator's finger, requiring extension of the vein incision partially into the auricular wall. Otherwise the vein may be torn partially away from the auricle, resulting in serious hemorrhage and great difficulty in restoration of the vascular continuity.

3. Hemorrhage about the finger, or about the finger and instrument, and after removal of the finger from the heart, may be difficult to control. It may be managed by the use of a purse-string suture in the auricular wall or by a curved Potts-type clamp applied to the side of the auricle, or by both. The former puckers the auricle so much that it makes reparative suturing difficult. The clamp may lacerate the usually somewhat friable auricular wall. At times bleeding may be controlled by manually pinching the vein.

4. Thrombosis of the pulmonary vein beginning at the suture line may lead to hepatization of the upper lobe of the left lung and to death from what clinically appears to be pulmonary edema. This occurred only once in our series, when the pulmonary vein was incised transversely. The lumen of the vein was markedly diminished by the constricting effect of the sutures. Since using a longitudinal incision, we have had no more difficulty of this type.

5. Temporary obstruction of the blood return from the upper lobe (or from the entire left lung when only a single left pulmonary vein exists) may precipitate cardiac standstill in a borderline case. The difficulties of performing cardiac massage and resuscitation are greatly increased when there is a clamp on the side of the auricle.

In spite of these objections, we have continued to use this means of approach to the left auricular lumen and expect to continue to do so in certain cases. An example would be the case in which thrombosis and organization have taken place in the distant past, resulting in extreme contracture and near obliteration of the appendage. It is impossible to insert the index finger through such a diminutive structure. It is possible, at least theoretically, to avoid embolism of thrombotic origin to all locations, both cerebral and peripheral, by entrance through a pulmonary vein.

When it is deemed necessary to utilize the pulmonary vein to enter the left auricle, the left pulmonary drainage is inspected. Sometimes a single large vein drains the entire left lung. Technically such a single large vein affords an ideal entrance into the heart, but it also requires cutting off the entire blood return from the left lung during the period of clamping. Usually the pulmonary veins enter the auricle separately, and its accessibility commends the superior one for our use.

TECHNIQUE OF COMMISSUROTOMY VIA LEFT SUPERIOR PULMONARY VEIN

The chosen vein is carefully mobilized within the pericardium. A purse-string suture of no. 2 braided silk on an atraumatic curved needle is placed in the auricular wall about the base of the vein. It is wise to have the extremities of the purse-string emerge from the auricular wall at its most accessible aspect (laterally) and at about 1 cm. from the vein insertion. This permits one to extend the vein incision for a distance into the true auricular wall if the vein lumen is insufficient to permit complete insertion of the operator's index finger. The purse-string is now passed through a Rumel-Belmont tourniquet to permit flexible, reliable hemostatic control.

A Potts' ductus clamp, preferably of the angulated variety, is now applied across the distal extremity of the vein. A half-curved Potts' clamp is placed on the auricular wall proximal to the vein. A 2 cm. incision is made longitudinally in the most accessible wall of the vessel, extending, if necessary, for a distance into the auricular wall. A bistoury type of knife blade and a pair of angulated iris scissors are ideal for opening the vein.

The two lips of the vein incision are picked up with fine thumb forceps, and the tip of the operator's double-gloved right index finger is engaged in the opening. As the proximal clamp is released, the finger is quickly inserted into the left auricle and the purse-string is tightened sufficiently to control bleeding.

The auricle and especially the internal opening of the appendage are explored for thrombi. If any are felt, they are carefully avoided. In three instances we have felt eel-like streamers of evidently very friable thrombi protruding from the appendage into the auricular lumen. In four cases there was extensive intra-auricular thrombosis, with a large portion of the chamber filled by incompletely adherent soft clots. In one case an attempt to remove the thrombi proved fatal (from hemorrhage). In the other three, they were ignored; they produced no subsequent symptoms.

The valve is explored and commissurotomy is performed by digital pressure alone whenever possible. The digital method was used alone in 10 cases of our series. The remaining 11 valves required instrumental division. When an instrument is employed, great technical difficulties may be encountered and bleeding is likely to be serious, because the necessary mechanical maneuvers often distort the vein orifice, owing to the disadvantageous site of entrance and the increased distance from the valve.

When the definitive work is completed, the finger is withdrawn from the heart and vein and the auricle is reclamped for hemostasis. The vein is flushed with dilute heparin solution, and the lips of the incision are approximated by a continuous suture of 5-0 arterial silk on a curved needle. We usually run it as an everting mattress suture from one extremity of the incision to the other and then reverse the suture, using a more superficial running stitch to approximate the adventitia. The terminal end of the suture can then be conveniently tied to the original end. If the vascular incision has invaded the auricular wall, it is well to place a single mattress suture of heavier material at this point.

After release of the clamps, pulmonary vein flow is resumed. There should be minimal bleeding. The pursestring suture may be tightened at any time if the clamp fails to provide adequate hemostasis. This suture should be removed or at least divided after vascular continuity has been restored.

After a sizable experience with the technical and potential difficulties and dangers associated with the approach to the valve via the superior pulmonary vein, we became satisfied that it did not provide a completely satisfactory answer to the problem of cerebral embolization during commissurotomy. Part of this dissatisfaction stemmed from a realization that emboli might originate from calcium deposition on the valve itself. Another unsatisfactory feature was the difficulty noted in recognizing minor degrees of thrombosis in the auricular appendage until after it had been actually clamped and opened. We felt that there was appreciable danger of the damage having already occurred by the time this stage of the operation had been reached. Slight to extensive thrombosis of the appendage had occurred in 35.5% of 200 cases of commissurotomy reported elsewhere.[9] In only about half of these was the condition recognized prior to opening of the appendageal lumen.

It appeared that it would be impossible, at least on many occasions, to prevent the entrance of free particulate matter into the systemic circulation during the course of a commissurotomy. There would thus always be danger that these particules might enter the carotid arteries and produce cerebral embolization. The only satisfactory answer seemed to lie in the direction of "straining" the blood entering these vessels or of obstructing them during the strategic periods when the particles might enter the aorta. "Straining" the blood seemed impractical, so that attempts were made to occlude the vessels.

It occurred to us that, by simply squeezing the patient's neck very tightly for periods up to 60 seconds at the *strategic moments* (such as during clamping of the appendage, inserting the finger into the heart, and dividing the calcified valve), we might greatly slow the flow of blood through the carotid arteries. Then the possible emboli would undoubtedly pass by the mouths of these vessels and produce their effects, if any, in the upper extremities or in the lower portions of the body. This hope proved to be vain. There were two cerebral emboli in nine patients subjected to such external cervical pressure. In addition, another patient seemed in danger of cardiac standstill, presumably as a result of the attendant carotid sinus reflex simultaneously produced.

Finally, it was deemed necessary to actually mobilize the root of the innominate and left common carotid arteries in the mediastinum to permit transient occlusion of their circulation. On the counsel of one of us (A. K. O.) and in view of the experiences of Hamby,[10] it was deemed reasonable to completely prevent blood flow through the innominate artery for a period of up to 20 minutes, during which time the definitive valvular operation should certainly have been accomplished. Such innominate occlusion would seem surely to prevent any possibility of embolization to the right side of the brain (the site of embolism in five, or 41.6%, of our series).

Courville[11] has shown that the brain can tolerate oxygen want, and Stokes and Gibbon[12] have shown that it can tolerate complete cessation of circulation for periods of up to three minutes. It was, therefore, deemed safe simultaneously to occlude temporarily the left common carotid artery at its origin for periods of up to 60 seconds. This could be done at each strategic period of manipulation, with reliance on the cardiac contractions to evacuate all free particulate matter from the heart and the aortic arch within 20 to 60 seconds of dislodgment. This would afford a considerable degree of protection to the left side of the brain.

We found we could easily accomplish this intermittent obstruction to the left common carotid artery by kinking it with an umbilical tape applied around it near its origin. It takes little traction to kink the left common carotid in the average person. It was felt best to leave the left subclavian artery unoccluded. It provides a certain amount of the cerebral circulation by way of the left vertebral artery and, therefore, would tend to prevent the more serious features of cerebral anoxemia from appearing. Should a small embolus enter the left vertebral artery, it would seem unlikely to appreciably interfere with the cerebral circulation, since there is extensive collateral circulation at the termination of this vessel and of the basilar artery (*fig. 3*).

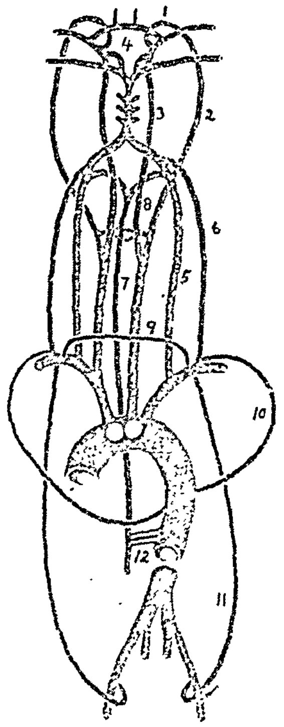

Fig. 3.—Possible collateral arterial pathways to the brain: (1) ophthalmic and supraorbital, (2) external carotid, (3) internal carotid, (4) circle of Willis, (5) vertebral, (6) ascending cervical, (7) anterior spinal, (8) superior thyroid, (9) inferior thyroid, (10) highest intercostal, (11) internal mammary, and (12) segmental.

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[9] Bailey, C. P., and Redondo, H.: To be published.

[10] Hamby, W. B: A Method for Control of Carotid Cerebral Circulation During Operation, J. Neurosurg. 2: 241-244 (May) 1945.

[11] Courville, C. B.: Untoward Effects of Nitrous Oxide Anesthesia, with Particular Reference to Residual Neurologic and Psychiatric Manifestations, Mountain View, Calif., Pacific Press Publishing Association, 1939.

[12] Stokes, T. L., and Gibbon, J. H., Jr.: Experimental Maintenance of Life by a Mechanical Heart and Lung During Occlusion of the Venae Cavae Followed by Survival, Surg., Gynec. & Obst. 91: 138-156 (Aug.) 1950.

TECHNIQUE OF PROTECTIVE CAROTID OBSTRUCTION

The technique we have worked out to apply this principle is as follows: A left lateral approach is used as usual. The fourth rather than the fifth intercostal space is chosen for entrance into the thorax, because it affords greater accessibility to the upper mediastinum. The lung is compressed downward, and the mediastinal pleura is opened longitudinally above the aortic arch between the left vagus and the left phrenic nerves. By blunt dissection, the left common carotid artery is mobilized at its origin. An umbilical tape is passed underneath it, and tentative traction is made so that the operator can determine the ease of kinking off its lumen (fig. 4).

In most instances the innominate artery is found to be more deeply located within the mediastinal tissues and to lie just anterior to the left common carotid. Occasionally it lies far enough anteriorly to require mediastinal dissection anterior to the left phrenic nerve. A silk suture is placed under it with a ligature carrier or gallbladder clamp but is not tied. Further mobilization and traction on this suture permits a noncrushing clamp to be placed across the origin of the vessel. We prefer to use a bulldog clamp of the Beck or Potts' type. Disappearance of the right radial and temporal pulse indicates complete occlusion (fig. 5). The innominate artery is kept obstructed during the entire period of definitive intracardiac surgery. This is usually less than 20 minutes but in one case was 34 minutes.

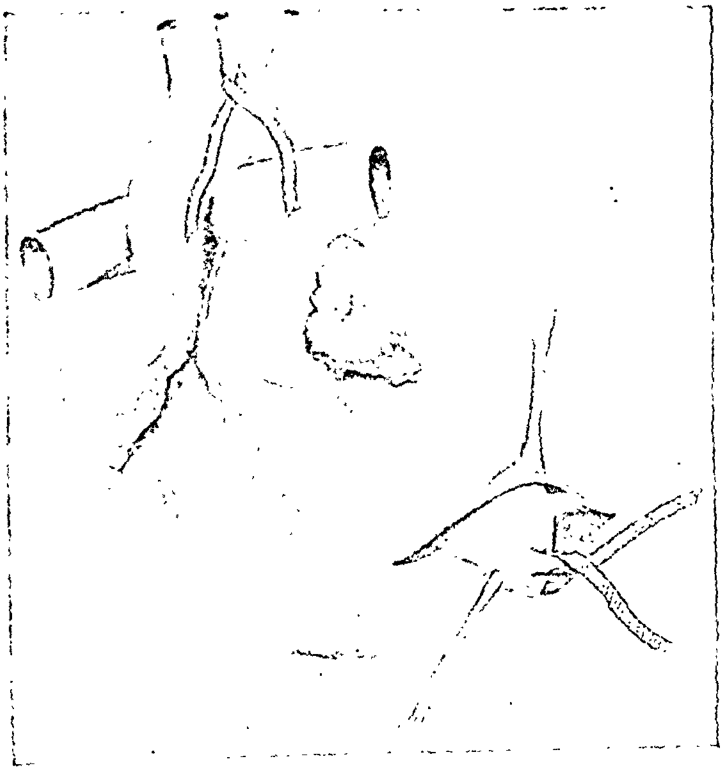

Fig. 4.—Umbilical tape about origin of the left common carotid artery for intermittent traction occlusion of blood flow at the strategic periods of the operation. Kinking of the vessel is carried out for periods of 30 to 60 seconds whenever it is felt likely that free particulate matter may be released into the blood stream.

The upper pericardium is then opened just posterior to and parallel with the left phrenic nerve. A purse-string suture of heavy braided silk is placed in the epicardium about the base of the appendage and is connected with a Rumel-Belmont tourniquet. If the appendage requires much mobilization or manipulation, the tape about the left common carotid may be drawn on for 30 to 60 seconds to permit safe evacuation of any inadvertently dislodged clots.

As a Satinsky clamp is applied across the base of the appendage, traction is again made on the carotid artery. The appendage is then opened and inspected for clots. They may be surgically evacuated if present. Then the operator's finger is inserted into the heart, and simultaneously traction is again made on the carotid for 30 to 60 seconds. Such carotid traction is repeated whenever the valve is examined or manipulated, if it is felt that any loosely attached calcium, vegetations, or clots are present. When a calcified valve is divided by either digital or instrumental means, traction is again made. Finally, as the finger leaves the heart and the appendage is tied the vessel is again occluded for a period estimated to be long enough to clear completely the heart and aortic arch of any loose particulate material. After the appendage is closed, the bulldog clamp is removed from the innominate artery and the suture and umbilical tape are cut and extracted. The operation is then terminated in the usual way, without any suturing of the mediastinum.

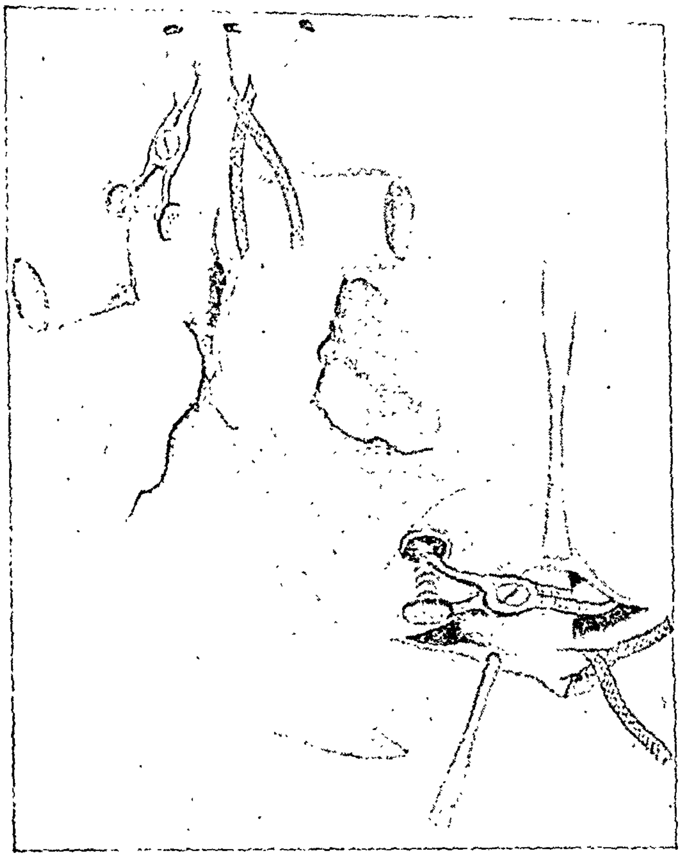

Fig. 5.—Bulldog clamp placed across the origin of the innominate artery to block its flow during the entire period of definitive intracardiac surgery. This should afford complete protection from embolization to the right side of the brain.

COMMENT

So far we have employed this type of partial obstruction to the cerebral blood flow in 80 patients undergoing mitral commissurotomy. In 26 of them, the appendage was grossly thrombosed. There have been no instances of cerebral embolization in this series. Neither have there been any symptoms suggestive of cerebral ischemia. There have been no recognized peripheral emboli. The longest period of obstruction of the left common carotid in this series was two minutes. The only recognizable untoward occurrence in this series of patients was development of a left recurrent laryngeal nerve paralysis in two patients.

SUMMARY AND CONCLUSIONS

Cerebral embolization as a result of the manipulations incident to mitral commissurotomy may be lethal or may permanently incapacitate the patient. This complication occurred in 5.1% of 235 cases in which methods other than carotid obstruction were employed to protect the brain.

Stellate ganglion block, in addition to other medical measures, was found to be of relatively small benefit in the treatment of 12 patients with cerebral embolization incident to commissurotomy. Prevention of the occurrence of cerebral (and peripheral) embolization of thrombotic origin during commissurotomy is theoretically and perhaps practically possible by utilization of the left superior pulmonary vein as a site of entrance into the left auricle. However, in 21 cases with obvious thrombosis of the appendage in which this technique was employed, there were two deaths, directly related to the vein approach. Because of the added technical difficulties and inherent potential dangers, this procedure is not recommended as a routine prophylactic method, although it will continue to be useful in certain cases of auricular thrombosis.

Prevention of complications by occlusion of the innominate and left common carotid arteries as described was attempted in 80 cases, with no evidence of cerebral or peripheral emboli and no evident untoward by-effects. Although the series is small, the results, to date, suggest that this technique may afford a reasonably easy, safe, and effective method for the prevention of cerebral embolization during commissurotomy for mitral stenosis and during other operations performed within the chambers of the left heart. Perhaps it should, in the future, be routinely used in all patients subjected to commissurotomy. Results suggest that there may be effective available collateral arterial supplies to the brain, which will support its life for periods somewhat longer than required by this technique.

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LOSS OF SENSE OF TASTE DUE TO METHYLTHIOURACIL THERAPY

Norman G. Schneeberg, M.D., Philadelphia

The thiouracil group of antithyroid drugs has received an extensive clinical trial in the past eight years, and their toxic effects have been amply documented. Loss of the sense of taste has been mentioned in only a single report.[1] One of the 16 patients treated with methylthiouracil by Leys complained of complete loss of taste while receiving a daily dose of 1.4 gm. Taste sensation gradually returned when the dose was reduced.

We have recently observed complete loss of the sense of taste in two patients receiving methylthiouracil. Because of the rarity of this toxic effect and its benign nature, a brief report is appended.

REPORT OF CASES

CASE 1.—A 48-year-old woman was referred by an ophthalmologist because of marked lid retraction without measurable proptosis of the eyes and persistent periorbital edema. Her history was long and complex and only those portions pertinent to the present discussion will be included. The thyroid gland was diffusely enlarged to about three times normal size. It was smooth though lobulated, firm, and easily outlined and exhibited no bruit. The pulse rate was 100 per minute and blood pressure 146/80 to 175/88. The thyroid uptake of radioactive iodine 24 hours after the oral administration of 50 μc of I^{131} was 43%. The serum protein-bound iodine was 7.4 μg per 100 cc. The basal metabolic rate was +6% and the serum cholesterol 178 mg. per 100 cc. Because of certain equivocal thyrotoxic stigmas and the high normal I^{131} uptake and protein-bound iodine level of the serum, a therapeutic trial of strong iodine (Lugol's) solution was instituted. Sufficient clinical improvement ensued to warrant extending the therapeutic trial with the administration of *methylthiouracil*, 600 mg. daily in divided doses. After 20 days of therapy, the patient reported the gradual loss of taste sensation. Within 10 more days gustatory sense was completely absent. She found food "flat" in taste and was unable to distinguish sweet, sour, salty, peppery, or bitter substances. Three weeks later she noted that the acuity of her sense of smell was somewhat dulled. She was unable to smell an onion, and customary lacrimation did not occur. Familiar odors could hardly be detected and could rarely be identified accurately. Otherwise she was clinically improved, exhibiting a weight gain of 5 lb. (2.3 kg.), reduction of pulse rate to 85 and blood pressure to 130/80, disappearance of lid lag, reduction of lid retraction, and absence of periorbital edema. There were no signs of hypothyroidism or myxedema. Cranial nerve motor and sensory function, aside from taste and smell, were intact. There was no evidence of facial palsy; the gag and corneal reflexes were normal; and phonation was unimpaired. The tongue appeared normal, showed normal motor activity, and was sensitive to touch, pain, and temperature changes. The buccal mucosa was normal. The parotid and submaxillary salivary glands and ducts revealed no abnormalities. Because the patient could no longer tolerate loss of gustatory and olfactory sensation, 5-iodo-2-thiouracil (itrumil,® Ciba Pharmaceutical Products, Inc.), 400 mg. per day, was substituted for methylthiouracil. She reported that three days after this change in medication severe pain in the thyroid region developed; the gland became more enlarged, hard, and painful; and her temperature rose to 104 F orally. Because of my absence, another physician was consulted, who made a diagnosis of acute thyroiditis. Aureomycin therapy effected a relatively rapid subsidence of the acute process. Two weeks later I examined the patient. The thyroid borders were prominent, and the gland was larger and firmer than on previous examinations. At this time, three weeks after cessation of methylthiouracil therapy, the patient was able to recognize only the taste of salt. During the ensuing two weeks, her sense of taste and smell gradually improved but was not completely normal. Food still tasted "flat," and sweet substances seemed intolerably saccharine. Six weeks after treatment with methylthiouracil was stopped, taste and smell were completely normal. The patient refused to retest the effect of methylthiouracil and continued taking iodothiouracil, with maintenance of clinical improvement and without toxic effects. The thyroid gland had returned to its original size and consistency prior to the episode when thyroiditis was diagnosed.

CASE 2.—A 39-year-old woman complained of weight loss of 22 lb. (10 kg.) in one year despite a good appetite and her usual diet, hyperhidrosis, heat intolerance, palpitation, and

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[1] Leys, D.: Hyperthyroidism Treated with Methyl Thiouracil, Lancet 1:461-464 (April 14) 1945.

The corticoid content of the urine was estimated according to the method of Heard, Sobel, and Venning,[8] which is based on the knowledge that most of the adrenal steroids have strong reducing sugar-like properties, which are imparted by the presence in the side-chain of the primary alpha ketol grouping, characteristic of fructose. All of the active hormones of the adrenal cortex have this reducing property.

Eosinophil counts were done by the method of Randolph[9] modified to stain only eosinophils. Counts were made in 0.2 mm. depth Levy counting chambers with improved Neubauer rulings. Pipettes were tested by the United States Bureau of Standards.

17-Ketosteroid, Reducing Corticoid, and Eosinophil Levels in Six Epileptic Patients *

Patient	E. B.	J. H.†	L. M.	J. R.	R. S.	M. S.	Mean
	17-Ketosteroids, Mg./24 Hr.						
Preseizure values	7.3	5.7	8.0	5.2	6.3	4.6	...
	5.7	6.2	6.4	9.0	9.6	6.3	...
	7.4	6.6	6.8	4.6	6.1	7.4	...
	...	7.7	...	2.5	9.4	6.8	...
	...	8.2	...	6.6	3.7	6.0	...
Mean	6.8	6.9	7.1	5.6	7.0	6.2	6.6
Postseizure values	9.8	...	6.7	8.9	4.4	8.6	...
	5.5	...	2.7	8.6	7.3	7.0	...
	2.5	...	5.8	4.4	5.8	5.9	...
	...	...	10.1	...	5.6	7.9	...
Mean	5.9	...	6.3	7.3	5.8	7.4	6.5
	Reducing Corticoids, Mg./24 Hr.						
Preseizure values	1.63	2.03	2.15	1.73	3.07	2.01	....
	1.47	1.68	1.52	1.19	1.18	1.33	....
	....	1.43	....	0.78	2.41	1.58	....
Mean	1.55	1.71	1.83	1.23	2.32	1.65	1.71
Postseizure values	1.71	....	2.28	1.51	0.31	1.41	....
	1.04	....	1.60	1.64	1.26	2.17	....
	....	....	....	....	1.05	....	....
Mean	1.38	....	1.94	1.57	0.87	1.79	1.51
	Eosinophils/Cu. Mm.						
Preseizure values	325	200	200	150	150	212	...
	257	211	175	118	166	306	...
	264	200	201	140	168	200	...
	297	229	231	147	159	303	...
Mean	286	210	202	116	161	255	210
Postseizure values	125	...	193	156	190	106	...
	88	...	206	244	150	86	...
	81	...	...	250	...	70	...
	75	...	...	...	...	68	...
Mean	92	...	199	216	170	82	127

* Analysis of variance:
17-ketosteroids: F = 1.07, not significant.
Corticoids: F = 1.22, not significant.
Eosinophils: F = 11.95, P = 0.01-0.001, significant.
† J. H. was not available for analysis of postseizure phase.

Data for the six patients are as follows:

R. S. was 52 years old. Her first grand mal attack occurred at the age of 16 and was set off by traumatic experience. The electroencephalogram showed a very abnormal right frontal area.

L. M. was 37 years old. Her first grand mal attack occurred at the age of 3, followed by convulsions for three years. Grand mal seizures started again at the age of 18. The electroencephalogram showed a very abnormal right frontal area.

J. H. was 56 years old. Her first grand mal attack occurred at the age of 20. The electroencephalographic tracings for both frontal areas were diffusely irregular.

M. S. was 30 years old. Her first grand mal seizure occurred at the age of 16. Electroencephalographic tracings were diffusely irregular.

E. B. was 34 years old. She had had grand mal attacks since the age of 12. Electroencephalographic tracings were diffusely irregular.

J. R. was 26 years old. Her first grand mal convulsion occurred at the age of 15. Electroencephalographic tracings for both frontal lobes were very irregular.

RESULTS

From the data in the accompanying table, it will be seen that the mean control excretion values for 17-ketosteroids are somewhat less than those reported in the literature for normal women in this age range.[10] It has been reported that 17-ketosteroids show a progressive decline with age.[11] Talbot and Butler[12] have estimated that a single determination of 17-ketosteroids on a 24-hour basis would give a value within 15% of the mean value obtained by repeated estimations. The variation from the mean values in this series is certainly greater than 15%. It should also be noted that there is no significant difference in the mean values of 17-ketosteroids excreted during the postseizure period in comparison with the control period (6.6 mg. per 24 hours before and 6.5 mg. per 24 hours after seizures).

A slight decrease in the excretion of reducing corticoids after seizures was observed. However, when subjected to an analysis of variance, this decrease was not statistically significant when compared with seizure-free values.

The effect of grand mal seizures on eosinophil levels is also shown in the table. It will be seen that there is a decrease in eosinophils following seizures in some cases. An analysis of variance indicated that this decrease was statistically significant. According to Rud,[3] the majority of authors agree that there is a general eosinopenia following convulsive attacks in epileptics.

COMMENT

The early work implicating the adrenal in convulsive states indicated that excessive secretion of the adrenals could, on occasion, precipitate seizures. Fischer and Fischer[13] in 1914 reported that inhalation of amyl nitrite produced convulsions in rabbits. They postulated that the capacity for convulsive reactivity in rabbits decreased with a decrease of adrenal substance. They also stated that one could increase convulsive susceptibility experimentally by causing a hyperfunction of the adrenal glands. Their work, although controversial, led to the examination of unilateral and subtotal adrenalectomy as a possible alleviating measure in cases of severe chronic grand mal epilepsy. The clinical results were equivocal.

The patients in this series exhibited an excretion pattern of 17-ketosteroids that was lower in quantity than comparable "normal" mean values for women in the same age group. The trend here would indicate that female patients with chronic epilepsy show a great deal more day to day variation in 17-ketosteroid excretion than do normal female subjects.

The failure to observe statistically significant changes in 17-ketosteroid and corticoid excretion patterns after

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8. Heard, R. D. H.; Sobel, H., and Venning, E. H.: Neutral Lipid-Soluble Reducing Substances of Urine as an Index of Adrenal Cortical Function, J. Biol. Chem. **165**: 699, 1946.

9. Randolph, T. G.: Blood Studies in Allergy: Direct Counting Chamber Determination of Eosinophils by Propylene Glycol Aqueous Stains, J. Allergy **15**: 89, 1944.

10. Mason, H. L., and Engstrom, W. W.: The 17-Ketosteroids: Their Origin, Determination and Significance, Physiol. Rev. **30**: 321, 1950.

11. Hamburger, C.: Normal Urinary Excretion of Neutral 17-ketosteroids with Specific Reference to Age and Sex Variation, Acta Endocrinol. **1**: 19, 1948.

12. Talbot, N. B., and Butler, A. M.: Urinary 17-Ketosteroid Assays in Clinical Medicine, J. Clin. Endocrinol. **2**: 724, 1942.

seizures, as compared with the seizure-free periods, indicates that adrenal cortical activity during the actual convulsive phase was not of sufficient intensity to produce changes great enough to be estimated by these two indexes of adrenal activity. However, the general eosinopenia following seizures does seem to indicate that the adrenal cortex is capable of responding to the stress of convulsive seizures with at least a "mild" activity. It would be expected that a stress such as a grand mal convulsion should be of sufficient intensity to induce changes in adrenal steroid excretion levels. Stresses of much less severity and intensity have been reported to induce changes in adrenal steroid excretion.[11]

The patients reported here had been experiencing seizures of the grand mal type for a number of years. A possible explanation for the failure to observe a urinary steroid response following seizures is that the adrenal cortex in these subjects, after years of being stimulated, may have become "conditioned" to the great number of attacks; therefore, it no longer responded to an "ordinary" stress, such as a convulsive seizure. The adrenal cortex in these patients with chronic epilepsy might simply be functioning at a very low ebb, probably only enough to maintain a slow excretion of corticoids, commensurate with the metabolic status of the subject in question.

It was noted during the course of this work that the incidence and duration of seizures increased during the menses and that patients who were maintained relatively seizure-free with diphenylhydantoin sodium and phenobarbital sodium therapy would occasionally "break through" with seizures during the menses in spite of continued medication. These and other observations suggest that, during periods of additional stress (such as the menses in female subjects), epileptics are more susceptible to convulsive seizures.

SUMMARY AND CONCLUSIONS

Women with chronic epilepsy exhibited a greater day to day variation in urinary steroid excretion than do normal female subjects of comparable age. There was no significant difference in the mean values of 17-ketosteroid and reducing corticoid values during the postseizure periods in comparison with control or seizure-free periods. There was a tendency toward eosinopenia after grand mal seizures. Patients with chronic epilepsy do not respond to convulsive stress in the same manner as nonepileptics. There is some indication that the adrenal cortex of patients with chronic epilepsy may become "conditioned" to convulsive stress.

333 Cedar St. (Dr. Bonnycastle).

13. Fischer, H., and Fischer, J.: Tierexperimentelle Studien über Amylnitritkrämpfe unter besonderer Berücksichtiggung der Frage nach ihrer Lokalisation und ihrer Beziehung sur inner Sekretion, Ztschr. f. d. ges. Neurol. u. Psychiat. 22: 241, 1914.

14. Mason and Engstrom.[10] Sayers, G.: Adrenal Cortex and Homeostasis, Physiol. Rev. 30: 241, 1950.

RESULTS OF SUBTOTAL GASTRECTOMY FOR GASTRIC AND DUODENAL ULCERS SINCE 1917

Alfred A. Strauss, M.D., Siegfried F. Strauss, M.D., Arthur H. Schwartz, M.D., David D. Kram, M.D.
and
Walter W. Masur, M.D., Chicago

In 1914, when most surgeons considered gastroenterostomy the proper surgical therapy for duodenal ulcers, we devised a method for pyloric closure in this operation, using submucous fascial transplants from the fascia lata. We found that this procedure was much more difficult in an inflamed stomach with a duodenal ulcer than it had been in dogs. In a number of cases the pyloric antrum was torn so badly that, as a means to an end, we did a subtotal gastrectomy. These were our first cases of subtotal gastrectomies, and we noted that the results with this operation were much more satisfactory than they had been with a simple gastroenterostomy.

Advocating subtotal gastrectomy as the operation of choice for duodenal ulcer, we presented six such cases at a meeting of the Chicago Surgical Society in 1915. Many of our colleagues at that time opposed this operation on the principle that radical surgical treatment was not indicated for duodenal ulcer, and we did not again have the courage to do subtotal gastrectomies until 1917. The excellent results, which we have noted over a period of years from our first cases of subtotal gastrectomy, had convinced us that this was the proper operation for duodenal ulcer.

For about two years we did some Billroth 1 and Billroth 2 gastric resections, but in 1919 we began routinely to do posterior mesocolic Polya resections. We are still using this procedure at the present time, since it affords such excellent functional results. During the succeeding years, we made slight changes in technique from time to time, but we returned to our original technique of 1919, which we are following today in minute detail.

There is probably no subject in medicine or surgery that has caused more controversy than therapy for duodenal ulcer. Not only has there been a great deal of dispute between surgeons and internists as to whether a duodenal ulcer should be treated medically or surgically, but there also have been variances among surgeons as to which type of operation should be performed for duodenal ulcer. Even after subtotal gastrectomy has been decided on in preference to gastroenterostomy or a pyloroplasty, there is often disagreement among surgeons as to whether it should be an anterior mesocolic or a posterior mesocolic operation.

This article has been shortened by omission of figures 7 through 18, which will appear in the authors' reprints.

From the Stomach Group and Surgical Services of Michael Reese Hospital, Mount Sinai Hospital, and Franklin Boulevard Hospital.

that has eroded into the pancreaticoduodenal artery, and (3) the gastrojejunal ulcer that has eroded into the mesenteric vessel of the jejunum. Operation should be limited to a simple ligation of the bleeding vessels, and subtotal gastrectomy deferred until the patient is in good condition. A hemorrhage from any of these locations is usually so severe and exsanguinating that immediate surgery is indicated to stop the bleeding and save the patient's life. Bleeding ulcers in other locations of the stomach do not require such emergency measures as the hemorrhage is much less severe.

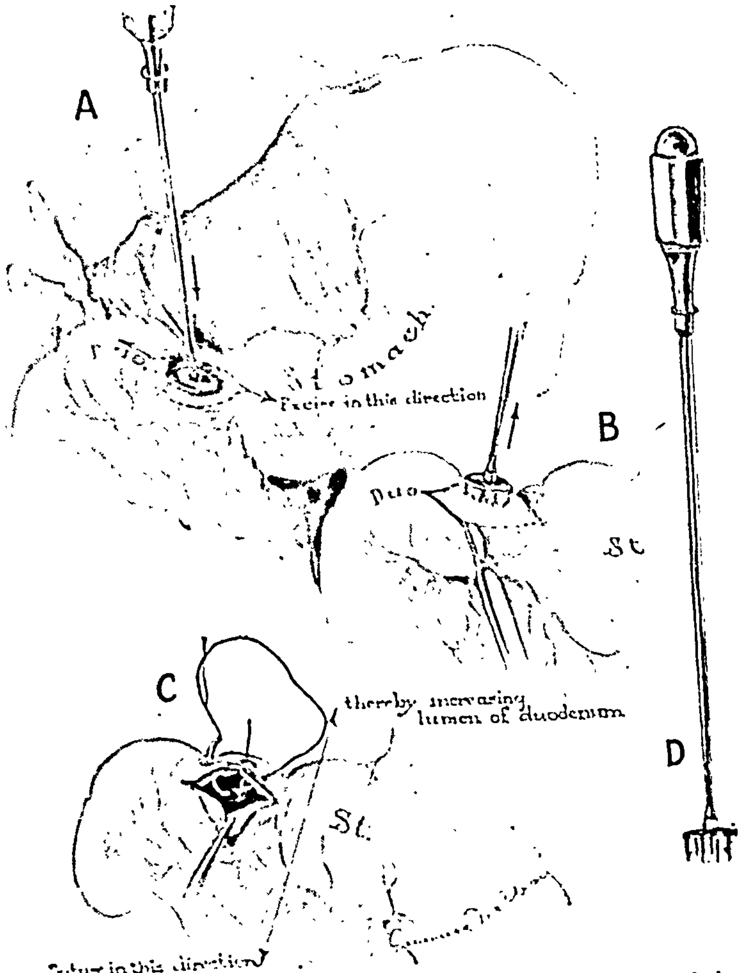

Fig. 1.—Operative procedure devised for treatment of perforated duodenal ulcer. *A* and *B*, excision through the pyloric ring into the duodenum with the perforation held in place by the fishhook spear *C*, transverse suturing of normal tissue with fine catgut and inversion wax silk suture after excision of the defect. *D*, fishhook spear devised to hold perforation in place.

PENETRATING ULCERS

Patients with acute symptoms of penetrating ulcers are kept on medical management from 10 to 14 days, when their symptoms usually subside and a subtotal gastrectomy can be done. We also treat patients with a retention from a pyloric or duodenal obstruction medically for 10 to 14 days, and institute continuous gastric suction through a Levine tube. This decreases the distention of the stomach, reestablishes its tonus, and relieves the inflammatory process in the duodenum and in the stomach wall. During this period the electrolyte balance is corrected by liberal infusions of glucose, saline, and amino acids. Several blood transfusions are also given.

TREATMENT FOR A RUPTURED DUODENAL ULCER

The ulcer area in a case of acute duodenal perforation should be widely excised, and the open area of the stomach anastomosed to the duodenum. If a gastroenterostomy is done in the presence of an acute perforation, we have found that in most of these cases a gastrojejunal ulcer forms shortly after the procedure. If a simple closure of the perforation is done and omentum is sutured over it, within six months to a year an obstructive duodenal ulcer usually occurs. For this reason, we resect a wide area around the perforated ulcer and close the defect (fig. 1). We have devised a simple instrument for this procedure (the metal fishhook type spear shown in fig. 1*D*) to hold the perforation in place. This is placed over the perforation, and forced through the muscular walls of the infiltrated areas (fig. 1*A* and *B*). A longitudinal elliptical incision is made through the pyloric ring into the duodenum. This is indicated by the dotted line in figure 1*A* and *B*. The entire area is widely excised, leaving only normal tissue, and then sutured transversely with fine catgut and a fine layer of inversion wax silk suture (fig. 1*C*). The area may be covered over with omentum. Our experience has shown us that there is a much lower percentage of recurring obstructive ulcers with this procedure than in cases in which a simple closure is done without excision of the perforated ulcer.

PATHOLOGICAL CONDITION

The chronic duodenal ulcer that is seen at surgery usually shows a marked contraction of the duodenal wall, with diverticula producing the clover-leaf deformity. The stomach is edematous and hypertrophied in appearance, and there is associated marked gastritis and duodenitis of various degrees. More than 75 to 80% of these patients have posterior wall ulcers penetrating into the head of the pancreas. The head of this organ is usually harder than normal, and slightly adherent to the pyloric ring and antrum and to the first portion of the duodenum. Th serosa of the duodenum and the hepatoduodenal lig ament are adherent and markedly thickened, and th stomach wall is also thickened in its lower third.

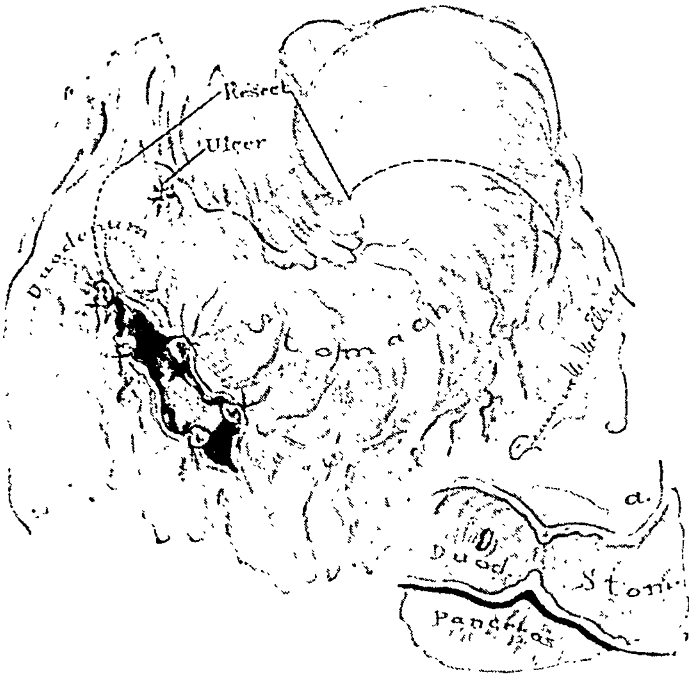

Fig. 2.—Ligation of blood supply to the greater curvature near th pylorus, and relationship of duodenal ulcer to the pyloric ring.

To achieve successful clinical end results with sul total gastrectomy, we have formulated two definite pri ciples: (1) all the pathological tissue should be remov if possible, and (2) from two-thirds to three-fourths of the stomach should be resected. We consider a rapid emptying time of 15 to 30 minutes ideal. This signifies that the stomach is only functioning from three to four times daily for 15 minutes, and is at rest the remainder of the time. We have also learned by studying the emptying time of stomachs following gastric surgery in which either a pylorectomy was done or too high a resection was performed that the stomach empties more rapidly and efficiently when three-fourths is resected. At this point it must be emphasized that the stomach varies in different persons as to size and configuration. Therefore *one has to visualize the structure in its entirety to determine* the proper level of resection.

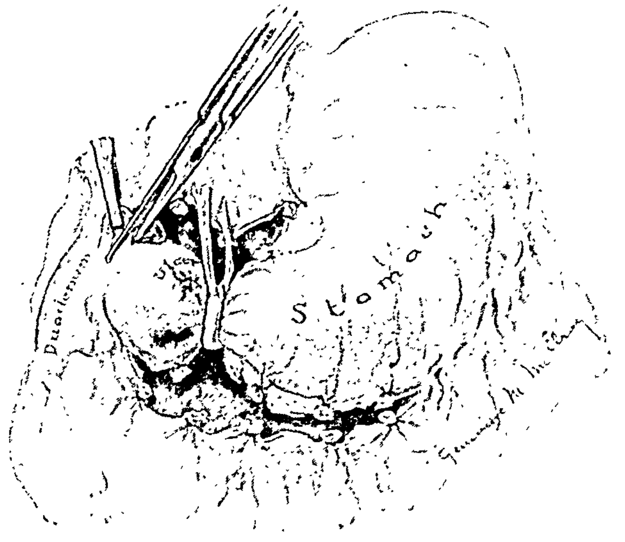

Fig. 3.—Division of anterior duodenal wall beyond the ulcer.

The various pyloroplasties, such as the Finney, Judd, Balfour, Horsley, and Heineke-Mikulicz types, have little value in therapy for duodenal ulcer, particularly for lesions that have penetrated to the pancreas. A well functioning gastroenterostomy cannot be considered as a satisfactory operation because of the high percentage of gastrojejunal ulcers occurring after this procedure. This has been reported in various clinics throughout the world, with incidences varying from 12 to 35%. In analyzing this evidence it is obvious that subtotal gastrectomy is the procedure of choice for duodenal ulcer. The mortality with this operation is very little higher than that of gastroenterostomy, and the incidence of gastrojejunal ulcers is only about 1%. Rather than subject a patient to a gastroenterostomy or another plastic operation, all of us in the gastrointestinal group would rather continue medical therapy.

SURGICAL TECHNIQUE

In doing a subtotal gastrectomy, we use a no-clamp method of the posterior Polya type. Through a median right rectus incision, the stomach is exposed. The prepyloric region is mobilized by forcing the index and middle finger through the lesser omentum along the posterior wall of the stomach. The blood vessels along the greater curvature of the stomach are clamped and ligated for about 2 in. (5 cm.) (fig. 2). The blood supply along the greater and lesser curvature of the duodenum is freed beyond the point where the duodenum is divided, which is usually just beyond the ulcer (fig. 3). A small piece of tape is tied around the pyloric end of the stomach to prevent the gastric contents from passing through the pylorus. This is tied off in place of using a clamp. The anterior wall of the duodenum is opened with a scissors below the point of the ulcer in the normal portion of the duodenum. After carefully determining its relationship to the common duct, the contents are then emptied by suction. The posterior wall of the duodenum is divided through the mucosa and the muscularis below the point of the ulcer and the contracted portion of the duodenum (fig. 4). *It is freed by sharp scalpel dissection* from the head of the pancreas where it is adherent due to penetration or perforation (fig. 5 and 6). It comes off very easily and the dissection is almost bloodless. The divided duodenum is now covered with a piece of gauze to prevent soiling (fig. 7). The inflammatory tissue resulting from penetration of the ulcer into the pancreas *is usually scraped out with a dull curette, and touched* with some tincture of iodine or thimerosal (merthiolate®) solution. This new method of dividing the duodenum below the point of the ulcer, below the point of the pathological condition, and freeing it from the head of the pancreas, is not only more rapidly accomplished but is much simpler. There is also much less danger of dividing or injuring the pancreaticoduodenal artery and the blood supply to the pancreas and duodenum. There is almost no danger of injury to the common duct, as it has been previously exposed. By dividing the duodenum below the ulcer, a free and loose duodenal wall results that can be closed as easily as a normal duodenum. We have used this technique in about 500 selected cases over a period of 10 years in preference to the older method described by us 25 years ago. *With the older technique, the* pancreas was freed away from the posterior wall of the duodenum, beginning at the pyloric end of the stomach, and dissected on down the posterior wall of the duodenum. This required more time, was more tedious, and made the closure of the duodenum much more difficult. The distal end of the duodenum is closed by inverting it with two rows of *wax silk sutures using the Connel stitch*

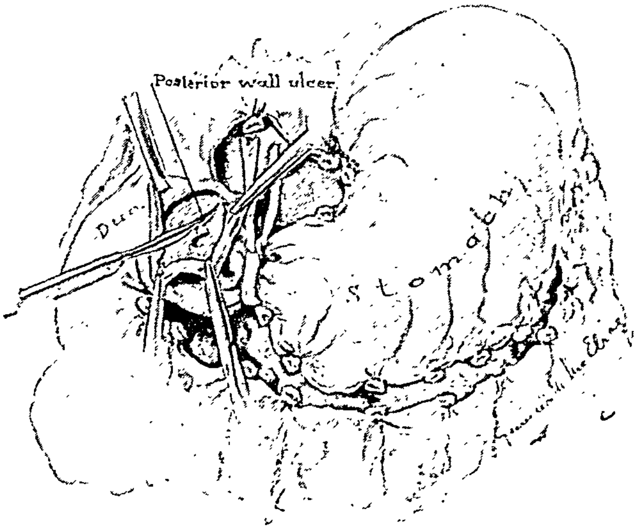

Fig. 4.—Incision of the posterior duodenal wall beyond the ulcer.

(fig. 8*A* and *B*). A third row is used to bring the lesser omentum and the capsule of the pancreas over the anterior portion of the duodenum (fig. 8*C* and *D*).

The blood vessels along the greater curvature are ligated between clamps until about 75% of the stomach is free. It is best to reach up to the cardia of the stomach to estimate and see the size of the stomach, as it varies greatly in different patients. The blood supply along the lesser curvature is divided at about the junction of the middle and upper third of the stomach (fig. 9). The mesocolon is lifted up and is split on the left side of the middle colic artery. The left half of the mesocolon is sutured with interrupted silk sutures to the posterior wall of the stomach as high up as possible (fig. 10).

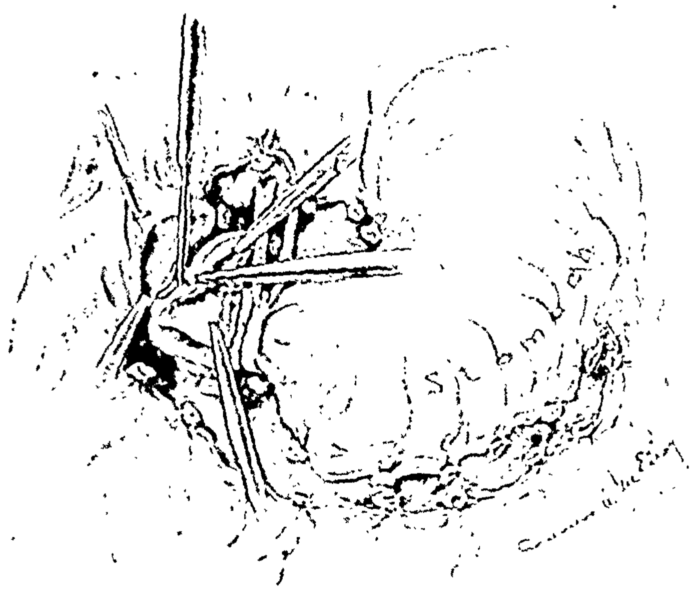

Fig. 5.—Dissection of the posterior duodenal wall from the head of the pancreas.

The first loop of jejunum as close to the ligament of Treitz as possible, usually about 1½ in. (3.8 cm.), is brought through the mesocolic slit, and sutured to the posterior wall of the stomach with continuous gastrointestinal catgut (fig. 11). The relationship of the jejunum to the remaining portion of the stomach is such that the proximal end of the jejunum is attached to the lesser curvature and the distal end to the greater curvature (fig. 11). The muscularis of the stomach and the jejunum is divided down to the submucosa about 4 mm. from the first suture line, and sutured with gastrointestinal catgut (fig. 12*A*). An opening is made through the mucosa of the posterior wall of the stomach, and the contents aspirated (fig. 12*B*). The jejunum and the posterior wall of the stomach are sutured with gastrointestinal catgut, starting at the middle of the posterior wall, using interlocking stitches (fig. 13) and continuing toward the greater and lesser curvatures.

The stomach is removed by cutting away the anterior wall, where vessels are clamped and ligated (fig. 14). The greater and lesser curvatures and the anterior wall are closed by continuous catgut, using the over and over oblique stitch (fig. 15*A*), which we devised in 1912. This suture only inverts a very small portion of the edge of the jejunum and the gastric wall (fig. 15*C*). A second layer of gastrointestinal catgut is used employing the Cushing stitch (fig. 15*B*). The anterior wall of the anastomosis is completed with a third row of continuous wax silk sutures. The right portion of the split mesocolon is sutured high on the anterior wall of the stomach so that the anastomosis is well below the mesocolon (fig. 16). Figure 17 illustrates the relationships. Figure 18 shows a high gastrectomy in which the mesocolon was short, and could not be brought over the anastomosis to the anterior surface of the stomach. If, in a high resection, the mesocolon is fat, we do not suture the mesocolon too high because of interference with the chest wall. We feel that it is much better to leave it in its normal position, and we have never seen any mechanical difficulty resulting from this. We never suture the split mesocolon to the jejunum, and have had no difficulties with this method. The mesocolon becomes agglutinated within a few hours after the operative procedure. We have never had a herniation through the small mesocolic slit.

CLINICAL END RESULTS

In a follow-up of 1,200 of these patients in both groups, we found that 95% were free of symptoms, had gained from 15 to 50 lb. (6.8 to 22.7 kg.), were on a full diet, and required no medication. A small number complained of fullness after eating, perspiration and weakness, and cardiac palpitation after each meal that lasts from 10 to 30 minutes. These symptoms disappeared after three to six months, and can be ascribed to a reflex of the vagus nerve and its branches. In a number of patients these symptoms disappeared after the stomach began to empty in from 15 to 30 minutes. In the remaining 5% of cases there were symptoms of distress that could not be explained on the basis of a faulty emptying time, or by roentgenological observations. Some of these were diagnosed as spastic colitis, others as visceral neurosis. Such conditions usually occur in highly emotional persons in whom it is difficult to ascertain whether their symptoms have an organic cause.

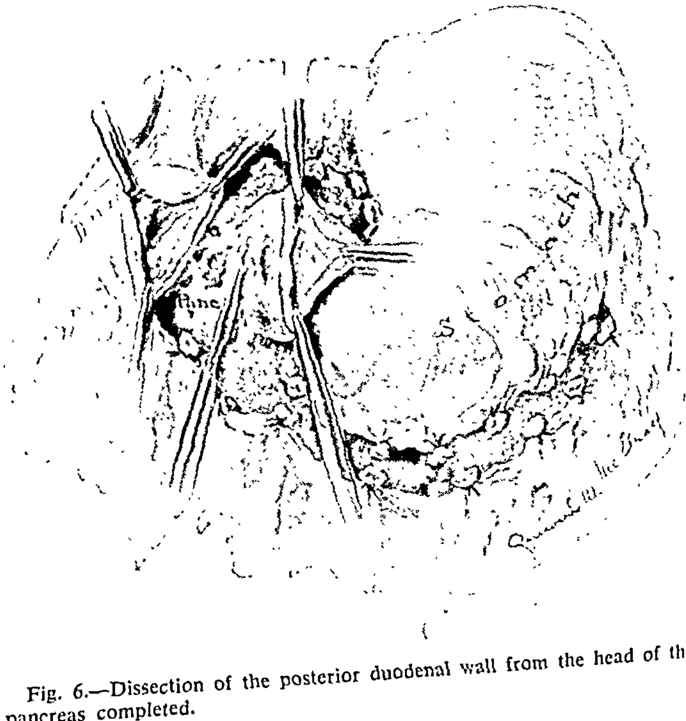

Fig. 6.—Dissection of the posterior duodenal wall from the head of the pancreas completed.

It was also observed that in patients whose stomachs at first emptied slowly, the emptying time after three to six months was between 15 and 30 minutes. This shows that the stomach and jejunum undergo muscular changes as the result of their new mechanical function.

The acidity following subtotal gastrectomy corresponds with that reported by Berg and Lewisohn. At the end of two weeks, 5% of these patients showed a free acid of from 10 to 15 units, with a total acidity of from 25 to 30 units. In the remaining group, there was no free acid, and the total acidity was from 5 to 10 units. Subsequent follow-up revealed no change from the immediate postoperative analyses. We believe that the low acidity among these patients can be attributed to the rapid emptying time of the stomach achieved by a high resection, preventing reflex irritation and the formation of hydrochloric acid. There has been almost no change in the blood picture postoperatively. Both the hemoglobin level and red blood cell count remained the same or increased slightly.

SUMMARY AND CONCLUSIONS

Experience with gastric resection over a period of 33 years in the stomach groups at Michael Reese Hospital, Mount Sinai Hospital, and the Franklin Boulevard Community Hospital have led us to the conclusion that the mortality in 1,796 nonselected cases of gastric, gastrojejunal, and duodenal ulcers justifies subtotal gastrectomy as the operation of choice for the surgical treatment of an ulcer. In group 1, in which there were 950 cases, the mortality was 3.5%, and in group 2, in which there were 846 cases, it was 1.4%.

The technique of a posterior Polya mesocolic resection, with the anastomosis across the entire width of the stomach, has been most satisfactory. That it presents very little mechanical difficulty is shown by the small number of cases in group 1 in which it was necessary to use a Levine tube with Wangensteen suction to aspirate the stomach.

While gastroenterostomy usually produces gastrojejunal ulcers in 20 to 30% of the cases, this complication occurred in less than 1% in our series following high subtotal gastrectomies. Other plastic operations are also unsatisfactory, since there is often a recurrence of the ulcer.

The removal of about 75% of the stomach is one of the important factors in the success of subtotal gastrectomy. The amount of stomach resected does not affect the mortality and morbidity. Subtotal gastrectomy is indicated for patients with a history of a second hemorrhage, a mechanical obstruction of the pylorus or duodenum, or those who have not responded to medical therapy after a sufficient trial. Medical treatment should be instituted in patients with slight symptoms, but those whose symptoms interfere with earning their livelihood should be treated surgically.

In 93 cases in which we did a cholecystectomy in addition to the subtotal gastrectomy, the morbidity or mortality did not rise. We believe in doing a cholecystectomy only if cholelithiasis is present.

The clinical end results in 1,200 cases in which a subsequent follow-up was done revealed that 95% of the patients were well, on a full diet without medication, had a normal blood picture, and showed an increase in weight from 15 to 50 lb. (6.8 to 22.7 kg.). All this should be ample proof that this operation, although a major surgical procedure, justifies itself.

104 S. Michigan Ave. (Dr. Strauss).

ANTIBIOTICS IN TREATMENT OF PANCREATIC FIBROSIS, WITH EMPHASIS ON TERRAMYCIN

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and
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The beneficial effects of aureomycin in the management of patients with pancreatic fibrosis (mucoviscidosis) have been demonstrated.[1] An extension of observations begun three years ago, and including over 100 patients, shows a favorable response in over 90%. Anticipation of the appearance of refractoriness to aureomycin and the poor response of the occasional patient to aureomycin suggested the use of terramycin.[2] The present report is an evaluation of terramycin in the treatment of 49 patients with pancreatic fibrosis. All patients, with two exceptions (the patients in cases 2 and 3), had pulmonary infections of varying severity. A brief note concerning the efficacy of terramycin has been made.[3] A full description of this disease is given elsewhere.[4] The main clinical features drawing attention to this condition in early infancy are hunger, with failure to gain weight on an adequate caloric intake; the passage of large, foul, and frequent stools; chronic cough; and excessive sweating. The index of suspicion should be high when examining siblings of a child with a proved case. Although a variety of laboratory examinations have been described to aid in establishing a diagnosis,[5] the most reliable one is an examination of the duodenal fluid obtained by intubation. The duodenal fluid in a typical patient is scanty and has increased viscosity and absent to markedly reduced pancreatic enzyme activity (trypsin, carboxypeptidase, lipase, and amylase). Observations from this laboratory indicate that all these criteria need not be present in every patient with this disease.

The most incapacitating feature of the disease, and the chief cause of death, is chronic pulmonary infection. The earliest respiratory symptom is usually cough, which often appears during the first few months of life. The cough may become increasingly severe and is associated

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The terramycin used in this study was supplied by Dr. Gladys L. Hobby and Dr. W. Alan Wright of the Chas. Pfizer & Co., Inc., Brooklyn.

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Dr. Robert Field Library
Medical College

with wheezing respirations or dyspnea; or it may become paroxysmal, suggesting pertussis. Repeated attacks of upper respiratory tract infections and bronchopneumonia are common. Progression of the pulmonary process may lead to bronchiectasis, empyema, and lung abscesses. Staphylococci are constantly found in association with the pulmonary infection.

Many patients show evidences of growth retardation and malnutrition. Some have a protruding abdomen and a prominent forehead. Those patients with advanced pulmonary infection are dyspneic and may have a typical cough, an emphysematous chest, clubbing of the fingers, cyanosis, and decreased vital capacity and exercise tolerance. On the other hand, patients with minimal involvement may present no abnormal physical findings. Roentgenograms of the chest in the early stages of the disease show hyperaeration with minimal evidence of infection. As the disease progresses, there is evidence of diffuse, patchy areas of emphysema and atelectasis, with dense hilar shadows.[6] Occasionally the chest roentgenogram may be sufficiently suggestive to give the clinician the first clue as to the presence of this disease.

In the management of these patients emphasis has shifted from the nutritional aspects to attempts at control of the associated pulmonary infection. The nutritional regimen consists of a high caloric, high protein diet with some restriction of fat and a liberal daily allowance of water-miscible multivitamin preparations. In small infants protein hydrolysates are freely prescribed. In addition, pancreatic substitution products [7] are administered to these patients at meal time.

In the control of the pulmonary infection numerous chemotherapeutic and antibiotic agents have been employed, either singly or in combination. Several sulfonamide compounds have been used, with temporary improvement in some patients. Penicillin and streptomycin have been employed both systemically and by aerosol. Administration of these antibiotics by aerosol has proved effective, especially when given for short periods; however, prolonged use is burdensome and often results in a diminished response. The introduction of aureomycin has resulted in a remarkable alleviation of the symptoms of most patients. A beneficial effect has been observed even in those patients with far-advanced bronchiectasis. Unfortunately, relapses of the pulmonary infection occur soon after the complete withdrawal of aureomycin, so that prolonged daily therapy is necessary. Some patients who responded well to aureomycin when it was first employed showed a poor response or no beneficial effect after a prolonged period of therapy. In addition to the control of the pulmonary infection, the treatment of the pulmonary involvement in this disease should be directed toward liquefying the thick, tenacious mucus that seems to be responsible, in part at least, for the associated obstructive phenomena.

The outlook for children afflicted with pancreatic fibrosis has been considered hopeless in the past. This pessimistic attitude is being modified as newer knowledge and greater experience are obtained. The increased clinical awareness of this condition in recent years has resulted in the recognition of many cases at an earlier age than was formerly the case. The prognosis in those patients with minimal pulmonary involvement, or in those in whom the disease is recognized early, should be much better than it was formerly.

PLAN OF STUDY

A total of 49 patients are included in this study, which was started in February, 1950, and continued through May, 1951. In each patient the diagnosis of pancreatic fibrosis was made by examination of the duodenal fluid. In many instances the diagnosis was corroborated by failure to find trypsin in one or more stool specimens,[5c] by failure of the patient to digest gelatin as determined by plasma glycine measurements,[5b] and by the patient's failure to absorb vitamin A when it was fed in an oily base.[5a] These patients were followed either in the hospital or at frequent intervals on an outpatient basis. The patients ranged in age from a few days to 16 10/12 years. They are divided into three groups as shown in table 1.

Group A consists of 13 patients who were treated with terramycin as the sole antibiotic from the time of diagnosis. Group B consists of 16 patients who had been treated with aureomycin as the only antibiotic prior to the administration of terramycin. Group C consists of 20 patients who had been treated with a variety of agents, such as sulfadiazine, aerosol penicillin and streptomycin, and intramuscular penicillin. In addition, aureomycin had been employed in each patient in group C for at least one year. Thus, the first group affords an opportunity for the observation of exclusive terramycin treatment, while the latter two groups permit comparison of the effectiveness of terramycin with other agents in the same patient.

At each clinic visit the history was carefully reviewed with respect to the following factors: respiratory infections, fever, cough, wheezing, exercise tolerance, physica activity, school attendance record, disposition, appetite, dietary habits, frequency and nature of bowel movements, sweating, and details of medication. The patient were weighed, measured, and examined. Laboratory examinations consisted of frequent nose and throat cul

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7. Many of the patients in this report received "viokase," a specially processed preparation of desiccated and defatted whole pancreas, which was provided by E. Levin of the VioBin Corporation, Monticello, Ill.

TABLE 1.—*Summary of Data in Patients Studied*

Case	Present Age		Age at Onset of Pulmonary Symptoms, Mo.	Age at Diagnosis, Mo.	General Condition at Start of Therapy	Weight at Start of Terramycin Therapy		Duration of Terramycin Therapy, Weeks	Weight After Therapy		Over-all Response to Terramycin Therapy	Comment
	Yr.	Mo.				Lb.	Oz.		Lb.	Oz.		
Group A: Patients Treated with Terramycin Daily from Time of Diagnosis												
1	..	11	8	9	Poor	10	0	9	13	10	Excellent	Also received intermittent aerosol penicillin and streptomycin
2	1	..	No symptoms	9	Good	14	8	13	18	3	Excellent	Sibling of patient in case 35; no respiratory symptoms
3	1	..	No symptoms	4	Fair	10	15	29	23	6	Excellent	No respiratory symptoms
4	1	6	1	3	Poor	11	6	56	31	0	Excellent	Cough-free; sibling died from pancreatic fibrosis
5	1	6	2	15	Poor	12	0	10	15	9	Excellent	See figures 4 and 5
6	1	8	3	17	Fair	19	0	12	21	0	Excellent	Minimal respiratory symptoms
7	2	..	5	19	Fair	23	3	21	27	8	Excellent	Rapid clearing of pulmonary symptoms
8	2	2	19	24	Poor	15	12	9	20	5	Excellent	Immediate response
9	2	2	9	22	Poor	16	0	16	18	0	Good	Advanced case; also received aerosol penicillin and streptomycin
10	3	2	24	30	Fair	19	13	31	26	0	Excellent	
11	3	4	1	36	Poor	20	0	17	25	13	Excellent	Playful and without cough for first time; see fig. 1
12	9	6	1	9 yr. 4 mo.	Fair	59	0	9	63	0	Excellent	Cough improved rapidly
13	16	10	18	15 yr. 10 mo.	Fair	128	0	54	133	8	Excellent	Clear of respiratory tract symptoms for first time
Group B: Patients Who Had Received Aureomycin as Only Antibiotic Prior to Terramycin Therapy												
14	..	9	2	1	Good	15	0	9	18	7	Excellent	Previous response to aureomycin also excellent; sibling of patient in case 23
15	1	1	1	2	Good	15	0	17	18	12	Excellent	Took elixir preparation well after refusing capsules
16	1	4	4	3	Fair	12	15	34	17	12	Excellent	Became cough-free; sibling of patient in case 38
17	1	6	2	Few days	Good	9	0	44	28	8	Excellent	Presented as meconium ileus; treated surgically
18	1	8	8	12	Poor	18	12	6	19	4	Fair	Had advanced lung involvement when first seen
19	1	8	8	16	Good	22	3	6	23	9	Excellent	Marked rapid improvement in respiratory symptoms
20	1	9	1	14	Fair	20	13	13	21	5	Fair	Vomited oral preparations; good response to terramycin aerosol
21	2	..	4	6	Fair	25	0	5	26	0	Fair	Terramycin tried after patient no longer responded to aureomycin
22	2	7	6	9	Poor	26	0	12	26	4	Fair	Advanced lung changes when first seen
23	2	8	3	18	Fair	28	8	15	30	0	Excellent	Sibling of patient in case 14
24	3	2	3	11	Good	34	0	3	35	0	Excellent	Rapid improvement in respiratory symptoms
25	3	3	1	3	Good	32	8	6	34	4	Good	Has done well with both drugs
26	3	10	1	25	Good	40	0	5	40	0	Excellent	Terramycin as effective as aureomycin
27	4*	..	1	39	Poor	26	8	4	24	12	Poor	Advanced lung changes when first seen; died Sept., 1950
28	4	8	6	4 yr. 3 mo.	Fair	37	12	6	38	0	Good	Pulmonary symptoms cleared quickly
29	7	10	5	5 yr. 6 mo.	Good	40	0	6	40	0	Fair	Coughing more than with aureomycin; far-advanced case
Group C: Patients Treated with a Variety of Antibiotics Prior to Terramycin Therapy												
30	2	9	1	Few days	Good	28	8	13	29	0	Good	Meconium ileus; treated surgically
31	2	9	2	3	Good	26	12	4	28	4	Good	Responded better to aureomycin than to terramycin
32	3	1	5	5	Fair	22	13	10	23	5	Fair	Has mild diarrhea with aureomycin and terramycin
33	3	1	1	3	Good	26	8	6	28	8	Good	Advanced pulmonary involvement
34	3	8	1	10	Good	29	0	6	27	8	Poor	Respiratory symptoms and appetite worse; sibling of patient in case 39
35	3	9	2	18	Poor	21	8	2	21	8	Poor	No longer responding to aureomycin when terramycin was tried
36	3	11	2	3	Fair	27	4	23	28	8	Excellent	Mild case; cough-free on aureomycin and terramycin
37	4	2	6	36	Good	34	0	8	35	0	Excellent	Did well with both drugs
38	4	6*	20	21	Poor	23	10	13	25	0	Fair	Appetite improved; far-advanced lung changes; sibling of patient in case 15; died Jan., 1951
39	4	8	3	23	Fair	30	0	3	30	14	Good	Advanced pulmonary changes; has never done as well as sibling, patient in case 34
40	4	11	3	7	Good	41	0	11	42	8	Excellent	Sibling died at seven weeks with pancreatic fibrosis
41	5	4	1	24	Poor	23	12	3	23	4	Poor	Advanced disease; markedly malnourished
42	6	3	15	22	Poor	32	4	10	32	8	Poor	Advanced involvement with bronchiectasis when first seen
43	6	9	2	21	Good	41	0	6	41	0	Good	Doing well but does better with aureomycin
44	7	10	3	4 yr. 6 mo.	Good	39	4	3½	40	4	Good	Status same when changed to terramycin
45	8	11	1	30	Fair	42	8	21	46	4	Excellent	Severe pulmonary involvement
46	9	2	4	Few days	Excellent	58	0	4	58	8	Good	Very active child with very mild cough
47	9	3	48	8 yr. 2 mo.	Good	51	0	18	55	0	Good	Bronchiectasis but remains in good control
48	10	11*	6	19	Poor	40	8	26	39	12	Fair	No longer responded to aureomycin; improved when changed to terramycin; died in Feb., 1951
49	11	4	6 yr. 6 mo.	8 yr.	Good	49	8	4	50	8	Good	Moderately severe lung involvement

* Age at time of death.

tures, roentgenograms of the chest, and occasional hemoglobin and leukocyte counts. Photographs were taken at frequent intervals. Close contact was maintained with patient's families between clinic visits, and the patients were seen whenever there was any question of the status. Some patients reported at weekly intervals, whereas others came once every two or three months, depending on their condition and other circumstances.

DOSAGE AND MODE OF ADMINISTRATION

The oral dose of terramycin ranged from 7 to 30 mg. per kilogram of body weight per day. Smaller or less severely ill infants received as little as 50 mg. a day in one dose, whereas older or sicker patients received as much as 750 mg. a day in two or three divided doses. Most patients received the total oral daily amount in a single dose. When acute respiratory infections developed with this schedule, the dose was increased to approximately 50 mg. per kilogram per day, given two to four times daily for the duration of the infection. Occasionally during an acute infection, supplementary aerosol penicillin (50,000 Oxford units) and streptomycin (50 mg.) were administered three or four times a day for about one week. Three patients in group A (cases 1, 5, and 9) received this supplementary treatment for brief periods at the time diagnosis was established.

TABLE 2.—*Clinical Response to Therapy*

		Excellent	Good	Fair	Poor
Group A (13 patients)........................	Respiratory symptoms	9	2 (5, 9)*	....	....
	Nutritional status	12	1 (9)	....	....
Group B (16 patients)........................	Respiratory symptoms	7	4 (15, 24, 25, 28)	3 (18, 21, 29)	2 (20, 27)
	Nutritional status	7	4 (21, 25, 26, 28)	4 (18, 20, 22, 29)	1 (27)
Group C (20 patients)........................	Respiratory symptoms	6 (30, 36, 37, 43, 45, 49)	8 (31, 32, 33, 39, 40, 44, 46, 47)	2 (38, 48)	4 (34, 35, 41, 42)
	Nutritional status	6 (33, 37, 40, 45, 46, 47)	6 (30, 31, 36, 39, 43, 49)	4 (32, 38, 44, 48)	4 (34, 35, 41, 42)

* Figures in parentheses are case numbers. See table 1.

Terramycin was used in four forms. The gelatin capsules contained 50, 100, or 250 mg. of terramycin and were given to the older children capable of swallowing capsules. Two cherry-flavored liquid preparations, one with a concentration of 250 mg. per 5 ml. and the other with 100 mg. per 0.5 ml. (as measured by calibrated dropper), were administered to the infants or to children who could not or would not swallow capsules. The concentrated liquid preparation was particularly valuable because of the ease with which it was administered. A preparation designed for aerosol therapy was used in five patients in a 50 mg. dose per treatment for periods up to 10 days. This preparation appeared to be irritating in this concentration.

BACTERIOLOGICAL STUDIES

Nasopharyngeal and oropharyngeal cultures were taken from all patients at the start of terramycin therapy and frequently on subsequent visits. Staphylococcus aureus was isolated from the nasopharynx in all 13 patients in group A at the start of therapy. After terramycin administration, staphylococci were still present in the nasopharynx of the nine patients on whom follow-up cultures were taken, even after 13 months of continuous therapy. No striking change in the pharyngeal flora was encountered in this group.

In the 36 patients of groups B and C, the following micro-organisms were isolated from the nasopharynx at the start of terramycin treatment: staphylococci, 27 cases; Pseudomonas pyocyanea, 6; Proteus, 5; Candida coli, 2; Hemophilus influenzae, 2; and pneumococci, 1. After varying periods of treatment with terramycin the pharyngeal flora was essentially unchanged. The micro-organisms recovered in 32 of these patients were as follows: staphylococci, 23 cases; Ps. pyocyanea, 6; Proteus, 3; C. coli, 0; pneumococci, 1; H. influenzae, 2; and Neisseria catarrhalis, 1.

In some instances the in vitro sensitivity of the micro-organisms isolated was tested against terramycin and other antibiotics. At the beginning of the study a test tube titration method, a modification of that devised by Randall and co-workers [9] for penicillin sensitivity, was used. The results of the observations made on the Staph. aureus obtained at varying periods during the study were as follows: 18 of 23 micro-organisms tested were found to be sensitive to aureomycin; 17 of 22 were found sensitive to terramycin; 9 of 11 were found sensitive to penicillin; and each of 11 was found sensitive to streptomycin. The level of sensitivity is taken as 2 μg in the case of aureomycin and terramycin, 2 Oxford units for penicillin, and 0.5 mg. for streptomycin. It is interesting to note that in some of the patients who showed a very striking clinical improvement the staphylococci isolated from the nasopharynx remained resistant in vitro to terramycin. Later in the study, sensitivity determinations were carried out by a modification of Bondi's method,[10] with paper discs and disc tablets. The results of these studies indicate no consistent correlation between the sensitivity of the staphylococci isolated from these patients and the clinical response.

CLINICAL RESULTS

The clinical effect of terramycin has been evaluated under the following two categories. 1. The effect on the respiratory features, including degree, severity, and frequency of cough, frequency of respiratory infections, fever, wheezing, dyspnea, exercise tolerance, and digital clubbing. The roentgenogram of the chest was also considered in the evaluation. 2. The effect on the nutritional status and general well-being, including changes in

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8. Deleted in proof.

[9] Randall, W. A.; Price, C. W., and Welch, H.: Estimation of Penicillin in Body Fluids, Science **101:** 365, 1945.

[10] Bondi, A., Jr.; Spaulding, E. H.; Smith, D. E., and Dietz, C. C.: A Routine Method for the Rapid Determination of Susceptibility to Penicillin and Other Antibiotics, Am. J. M. Sc. **213:** 221, 1947.

weight, alteration in nature and frequency of bowel movements, appetite, degree of physical activity, disposition, alertness and interest, and number of days absent from school. The over-all response of each patient to terramycin is presented in table 1. Table 2 shows the clinical response to terramycin evaluated in the manner referred to above.

Respiratory Symptoms.—All patients in group A showed pronounced improvement in respiratory symptoms. Improvement in cough was the most striking change and usually occurred within a week after the institution of therapy. This improvement occurred in spite of the fact that some of these patients had severe and prolonged symptoms with presumably irreversible changes. The parents of several of these patients made the comment that their child was free of cough for the first time since birth. Figure 1 demonstrates the improvement in the roentgenogram of the chest of the patient in case 11. This child had been troubled from the age of one month with a cough severe enough to keep him and his family awake many nights. The diagnosis was made when he was three years of age. Within a week after the institution of terramycin therapy he became cough-free and has remained so through the date of this report, a period of four months. Similar responses were noted in the other patients in group A. Changes also occurred in the objective physical findings, such as the disappearance of rales and the improvement in dyspnea and cyanosis when these were present. Digital clubbing became less pronounced in some patients. Acute infections of the respiratory tract occurred infrequently in this group, and the infections were not severe when they did occur. Usually the patient responded quickly when the amount of terramycin administered was increased temporarily.

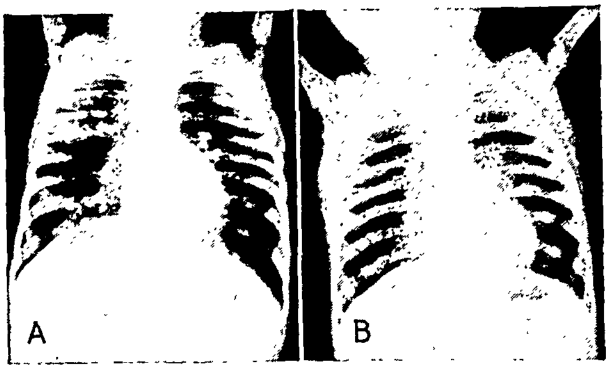

Fig. 1.—Roentgenogram of the chest of patient in case 11, *A*, before being given terramycin and *B*, seven weeks after start of terramycin therapy, 250 mg. daily.

The evaluation of the response of patients in groups B and C was more difficult, because these patients had already received antibiotics for varying periods of time. The patients in group B had previously shown the same dramatic response when first given aureomycin. The patients in these groups were generally older and had more advanced pulmonary disease. For the purpose of the study, terramycin was substituted for aureomycin at the same dosage level. From previous experience it was known that the discontinuance of aureomycin would result in a recurrence of respiratory symptoms; therefore, if a patient who had been relatively free of respiratory symptoms with aureomycin therapy continued to do well or did better with terramycin, the response was considered "good" or "excellent." If a patient was relatively free of respiratory symptoms with aureomycin therapy but showed evidences of relapse with terramycin, the response was considered "poor." Other patients, who were doing only fairly well or poorly with aureomycin therapy, improved somewhat with terramycin and were considered to have had a "fair" or "good" response. Still others in these groups were doing poorly with aureomycin therapy and continued to respond poorly to terramycin therapy.

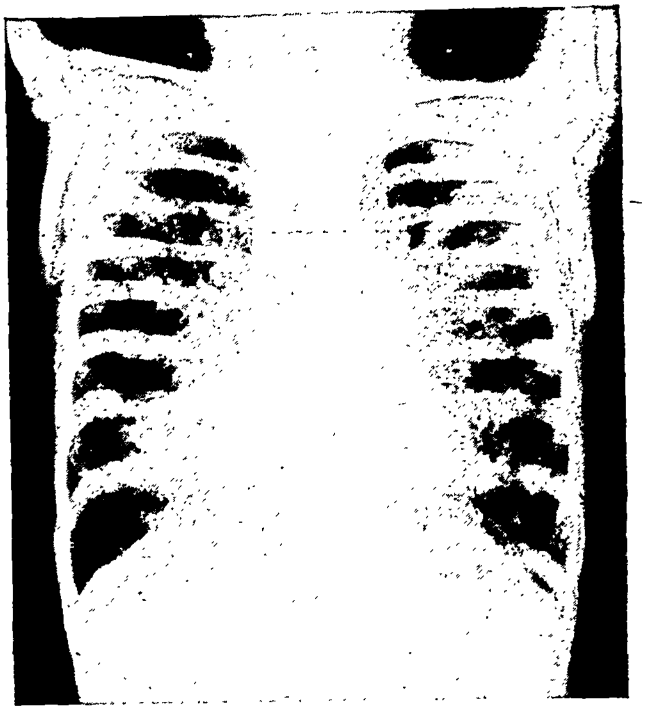

Fig. 2.—Roentgenogram of the chest of patient in case 27 taken one month before death.

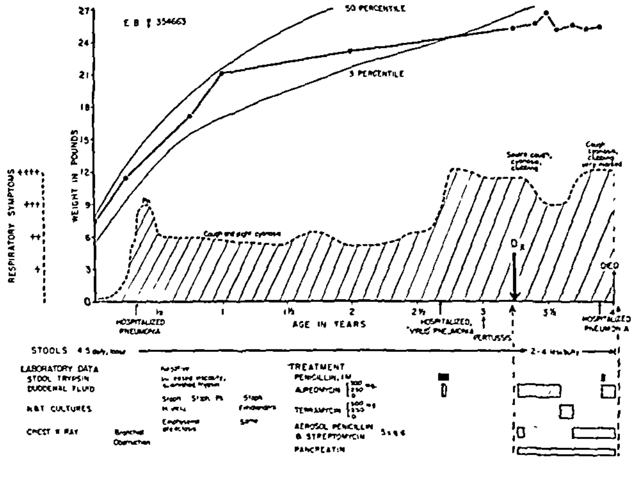

Fig. 3.—Course of patient in case 27. Note failure to respond to treatment. This patient had far-advanced pulmonary disease when a diagnosis was made at the age of 3 5/12 years.

Three patients (cases 27, 38, and 48) who had an initial poor or fair response to terramycin are now dead. The postmortem examination in each case showed far-advanced bronchiectasis, with patchy areas of emphysema and atelectasis. The roentgenogram of the chest of the patient in case 27, taken one month before death, is shown in figure 2. This patient's course is presented in figure 3.

General Well-Being and Nutritional Status.—Improvement in well-being and nutritional status was most marked in the patients in group A. Concomitant with improvement in respiratory symptoms, there was increased physical activity and rapid gain in weight, often accompanied by striking improvement in disposition. The few children who had poor appetite as a result of severe pulmonary infection regained their former excellent appetite. In general, physical development was accelerated in cases in which it had been retarded prior to therapy. The remarkable gains in weight after the institution of terramycin therapy in the patients of group A is shown in table 1. Figures 4 and 5 illustrate the changes observed in the patient in case 5. Patients in groups B and C showed a less dramatic improvement in weight and general well-being than those in group A; however, improvement in the nutritional status took place in the majority of patients (table 1).

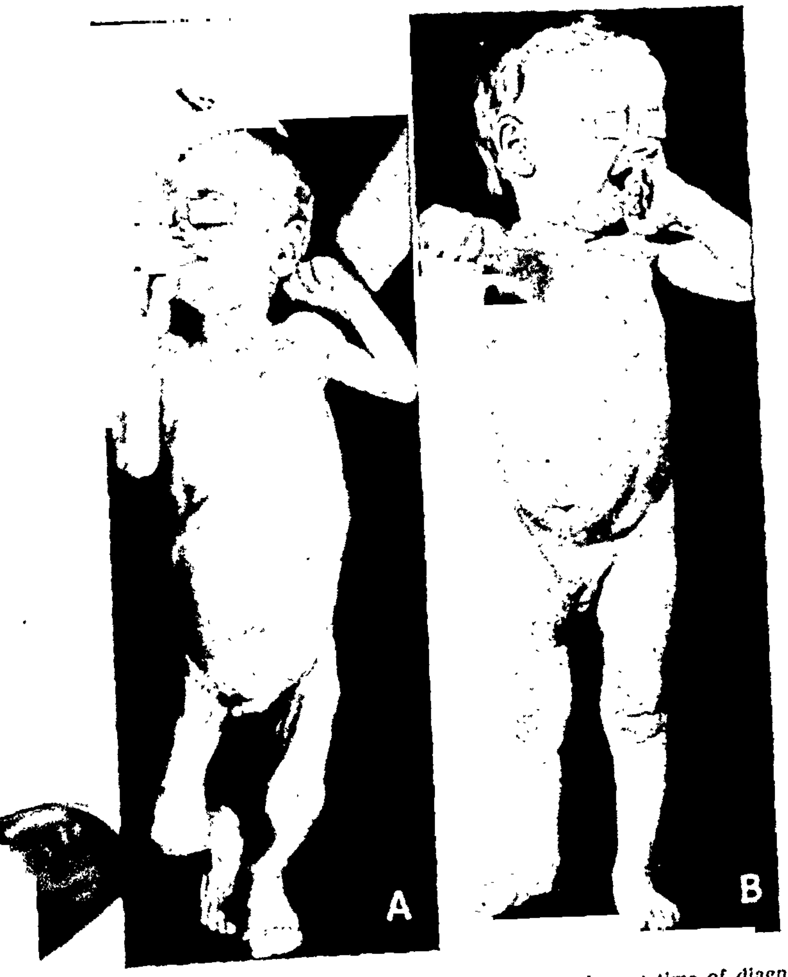

Fig. 4.—Photographs of patient in case 5, *A*, taken at time of diagnosis and *B*, 10 weeks later after treatment with terramycin.

SPECIAL PROBLEMS IN MANAGEMENT

Relapse.—In general, when terramycin therapy was discontinued for brief periods and no other antibiotic was substituted, the recurrence of pulmonary symptoms was noted. With the resumption of terramycin therapy, these symptoms disappeared. This has been our previous experience with aureomycin. It appears, therefore, that prolonged and continuous antibiotic therapy is necessary if respiratory symptoms are to be kept at a minimum.

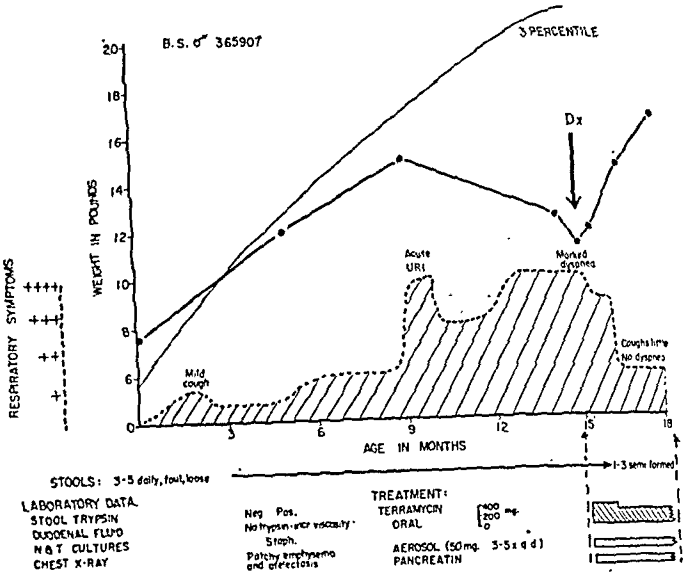

Fig. 5.—Course of patient in case 5. Note rapid gain in weight and improvement in respiratory symptoms after the beginning of terramycin treatment. URI = upper respiratory infection.

Undesirable Reactions.—In the dosage schedule employed, terramycin did not produce any undesirable reaction, except for one patient in whom mild diarrhea developed (case 32). One patient (case 30) who vomited when terramycin was given in capsules tolerated the dru well when it was given in concentrated liquid form. Another patient (case 20) vomited the oral preparations fo a short time and was later able to tolerate the drug an responded to terramycin aerosol.

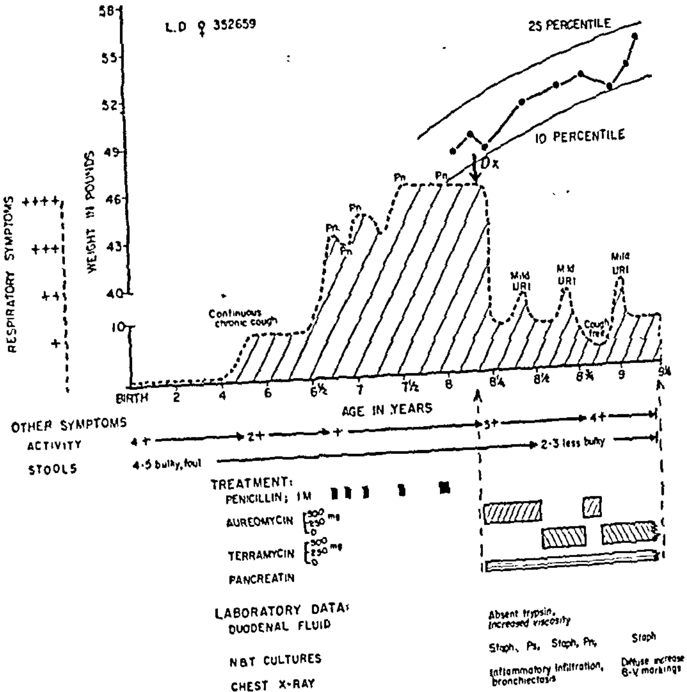

Fig. 6.—Course of patient in case 47. She had a favorable clinica response with both aureomycin and terramycin. Note the late age a which diagnosis was made. This patient had many attacks of pneumoni in the two years prior to referral to this clinic. There was no history o pulmonary symptoms during the first four years of life. Pn = pneumoni URI = upper respiratory injection, and BV = bronchovascular.

Refractoriness.—A diminishing clinical response to terramycin with prolonged therapy has not yet become a problem. Those persons whose initial response was good or excellent continue to do well. Judging by our experience with aureomycin, we anticipate some degree of refractoriness after prolonged therapy. Two patients (cases 38 and 48) had an initial favorable response to aureomycin but failed to respond after prolonged treatment. When the therapy was changed to terramycin, one patient (case 38) showed no improvement while the other patient (case 48) showed some temporary improvement. Both patients have died.

COMPARISON OF RESULTS WITH TERRAMYCIN AND AUREOMYCIN

Table 3 compares the clinical response of the 36 patients in groups B and C. Twenty-two responded equally well to both drugs; seven had a somewhat better response to terramycin and seven to aureomycin. Thus it would appear that the two drugs are equally efficacious in the management of pancreatic fibrosis. Figure 6 presents more detailed data on a patient who had a good response to both drugs.

COMMENT

The improvement that may follow antibiotic therapy can be so striking that the clinical appearance of the patient may cause one to question the validity of the original diagnosis. In spite of this clinical response, repeat duodenal fluid studies will reveal the characteristic findings that originally led to the diagnosis. Indeed, the withdrawal of therapy usually brings on a recurrence of former respiratory symptoms. We should like to emphasize the importance of establishing a diagnosis early so that therapy can be started before extensive pulmonary lesions become well established. The failure of therapy in a patient with extensive pulmonary involvement, as in the patient in case 27, is understandable and in contrast to the excellent response in the patient in case 4, whose treatment was begun at 3½ months of age. Another reason for starting therapy early is the observation that infants may have more trouble with thickened bronchial secretions and are more likely to die from the disease than older children. In the group of patients discussed, the diagnosis was established when they were under 3 months of age in 11 instances, under 9 months in 18 cases, and under 2 years in 36 cases (73.5%). The onset of pulmonary symptoms was noted in 26 patients before the age of 3 months, in 14 patients between 3 and 9 months, and in 7 patients over the age of 9 months. Two patients show no pulmonary symptoms at the time of this report.

Although great strides have been made in the early recognition of this disease, we must attempt to shorten further the span between the appearance of symptoms suggesting this condition and the date of establishing a diagnosis and instituting therapy. In this study approximately 54% of the patients had pulmonary involvement as demonstrated clinically and/or by roentgenogram by the age of 3 months; however, in only 22% was the diagnosis made by this age. It appears that the age at the time of diagnosis and establishment of a sound therapeutic regimen may be one of the deciding factors in the outcome. The severity of the disease is equally important, however, and this is oftener reflected by the intensity of the pulmonary symptoms than by the degree of gastrointestinal dysfunction. It is too early to draw conclusions as to the eventual outcome of these patients; nevertheless, comparison with the best results obtained prior to three years ago would be most favorable. The only major change in our therapeutic program has been the introduction of the newer antibiotics. The formerly used agents, such as sulfadiazine, penicillin, and streptomycin, were effective and often produced favorable results. The combined use of penicillin and streptomycin in aerosol form, 50,000 units of the former plus 50 mg. of streptomycin from two to five times daily, is still useful in the most refractory cases. In these same persons the use of a combination of agents may be most effective, such as combinations of penicillin and streptomycin in aerosol form, orally administered terramycin, or aureomycin plus sulfadiazine. Our experience with chloramphenicol (chloromycetin®) is limited, and it has been thought useful in the most advanced cases when combined with other drugs. The use of chloramphenicol alone in some early cases did not seem to be as satisfactory as either aureomycin or terramycin. Further experience is necessary for proper evaluation of this drug. We have been impressed with the effectiveness of sulfadiazine after prolonged periods of aureomycin or terramycin therapy. In the past we had been limited in the number of effective agents, but today, with so many, the rotation from one effective drug to another may become the ideal form of therapy.

TABLE 3.—*Comparison of Aureomycin and Terramycin Therapy in Groups B and C (Thirty-Six Patients)*

	Response to Aureomycin *			
Response to Terramycin	Excellent	Good	Fair	Poor
Better (7)	....	15, 16, 17, 24	39	45, 48
Equal (22)	14, 19, 23, 26, 37, 40	25, 28, 30, 33, 36, 44, 46, 47, 49	18, 20, 21, 22, 32	27, 38
Worse (7)	31, 43	29, 34	35, 41, 42	

* Figures in these columns are case numbers.

The emergence of resistant strains of staphylococci in the respiratory tract and the appearance of a new flora with resistant micro-organisms were problems of great concern when this study was started. Ps. pyocyanea was recovered with greater frequency in those patients treated with a variety of antibiotics over prolonged periods of time; however, in those patients treated with terramycin alone and for relatively shorter periods the bacterial flora of the nasopharynx showed little change. We were alert for the occurrence of moniliasis, but this complication did not occur except in a few patients terminally. The emergence of terramycin resistant staphylococci in vitro did not correlate with the therapeutic response. In spite of the presence of resistant staphylococci in the nasopharynx, patients continued to show a favorable clinical effect. This suggests that terramycin may have an action other than a pure antibacterial one. A similar action was suspected in the case of aureomycin. In studying the action of aureomycin in patients with pancreatic fibrosis, Stowens [1b] points out from his nitrogen balance studies an almost fourteenfold increase in the amount of nitrogen retained. He attributes this to three factors: (1) an increase in the amount of nitrogen voluntarily ingested, (2) an increase in the amount of nitrogen absorbed, and (3) an increase in utilization of absorbed nitrogen. An action similar to this in the case of terramycin has not been demonstrated but would appear likely. The weight gains recorded in table 1 of patients receiving terramycin as the sole antibiotic are very striking. This is especially true when one notes that in many instances there was insignificant or no weight gain for weeks before the institution of therapy. The gains recorded are in the same range of magnitude or slightly higher than those reported by Stowens, who used aureomycin. Whether this is due to differences in diet or to the use of pancreatin in most of our patients is difficult to say. We felt that the administration of pancreatin was advisable in view of its effect in improving nitrogen absorption. Pancreatin per se has no ameliorative effect on the respiratory tract symptoms. A few patients were treated without pancreatin for varying periods of time, and it was felt that this product did not interfere with our appraisal of antibiotic therapy. The changes in the intestinal bacterial flora have not been studied by us. Terramycin alone does not improve the defective proteolytic action in the small intestine, as determined by oral gelatin feedings and by comparison of the plasma glycine nitrogen of a two hour postprandial specimen with a fasting specimen. In this regard terramycin does not behave like pancreatin. It should also be noted that failure to absorb vitamin A in an oily base persists with this antibiotic therapy.

Although emphasis in this presentation is given to the antibiotic agent, other aspects of management must not be neglected. Our dietary regimen includes a high caloric iet with a high protein and slightly reduced fat intake, nd a high water-miscible, multivitamin intake plus the addition of pancreatin. The use of such agents as iodides, antihistamines, and bile salts may have a place in selected circumstances; however, since these measures alone do not produce striking improvement in most patients with pulmonary involvement, the greatest part of the favorable response must be attributed to the daily use of the antibiotic.

The inclusion of two patients (cases 17 and 30) who had meconium ileus at birth should be noted. In these infants, the clinical and laboratory features of pancreatic fibrosis have developed and have responded to therapy. Great credit is due the surgeons who have been able to relieve the meconium obstruction by surgical measures in the first few days of life in over 50% of the patients with this condition in the past three years.

SUMMARY AND CONCLUSIONS

1. An evaluation of terramycin in the management of 49 patients with pancreatic fibrosis is presented. Thirteen patients (group A) received no previous antibiotic therapy. The remaining 36 (groups B and C) had received aureomycin and other antibiotic and chemotherapeutic agents for varying lengths of time.

2. Each of the 13 patients in group A had an excellent response as judged by the improvement in pulmonary symptoms, well-being, and nutritional status. Of the other 36 patients in groups B and C, all but 5 responded favorably. No serious untoward reaction to terramycin was encountered. Clinical relapse usually occurred when the antibiotic was discontinued.

3. The effectiveness of terramycin is compared with that of aureomycin in the management of pancreatic fibrosis in 36 patients. It is concluded that the two drugs are equally efficacious. In seven patients aureomycin seemed to be more effective than terramycin, and in seven patients terramycin seemed more effective than aureomycin. In the remaining 22 patients both drugs were alike in their effect.

4. Terramycin is effective in the management of pancreatic fibrosis when administered in small doses of from 7 to 30 mg. per kilogram of body weight on a daily basis. Early diagnosis and prolonged therapy results in the most favorable clinical response. Suggestions for the management of refractory cases are made by the use of combinations of antibiotics and the rotation of these agents.

5. The importance of other measures that have been mentioned in the management of patients with this disease should not be neglected. Indiscriminate use of antibiotics without proper control and frequent observations should be discouraged. The employment of adjunct measures, such as dietary control, good nursing care, personal hygiene, and the use of expectorants, should be part of the provision of complete medical care for each patient.

300 Longwood Ave. (Dr. Shwachman).

Perforated Peptic Ulcer.—In evaluating the methods of therapy for a complex clinical problem, such as an acute perforated peptic ulcer, the important consideration is not whether one therapy should be employed to the exclusion of another but rather wherein each may conceivably be used with the greatest advantage. . . . At present, we are disposed to favor surgical exploration and the use of simple suture in all early acute perforations, whether the diagnosis is suspected or definitively proved, unless the condition of the patient contraindicates a laparotomy or the use of an anesthetic. Likewise, if a case is seen after a lapse of 12 to 24 hours without convincing clinical signs of localization, we still prefer this approach providing no operative contraindication exists. If the clinical evidence in such delayed cases indicates a definite spontaneous containment of the effects of perforation, we are prepared to entertain the use of the nonoperative method. Our decision, to a large degree, would be determined by the condition of the patient. Finally, we are prepared to modify our preference for the use of simple suture in acute perforations which occur within or adjacent to chronic calloused penetrating ulcer, particularly at the site of the reentrant angle of the stomach. In such cases, in which closure may prove difficult or uncertain, a partial gastrectomy may be justifiable providing the condition of the patient is favorable and the experience of the surgeon is adequate.—Sigmund Mage M.D., and Benjamin A. Payson, M.D., A Consideration of th Present Status of Simple Suture in the Treatment of Acut Perforated Gastroduodenal Ulceration, *Surgery, Gynecology an Obstetrics*, May, 1952.

ELECTROCARDIOGRAPHIC PATTERNS IN PATIENTS WITH SECOND MYOCARDIAL INFARCTIONS

STUDIES OF TWELVE PATIENTS WITH ANTERIOR MYOCARDIAL INFARCTION FOLLOWED BY POSTERIOR MYOCARDIAL INFARCTION

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and
Chauncey C. Maher, M.D., Chicago

The electrocardiographic patterns of acute myocardial infarctions are a matter of common knowledge, and the serial changes have been documented by a number of authors. Less attention, however, has been directed to the serial changes due to second infarctions, which are the subject of this study. This report is limited to cases in which an original infarction of the anterior type is followed at a later date by a second of the posterior type.

REVIEW OF THE LITERATURE

On this subject, the earliest pertinent reference we were able to find was that of Levine and Brown [1] in 1929. They reported a series of 46 cases of coronary thrombosis; 6 of these showed both "old and recent lesions." Of these six, one (case 19) showed an electrocardiographic pattern of the anterior type in 1922 and a posterior type pattern five months later.

Four years later, Wood, Bellet, McMillan, and Wolferth [2] said: "Electrocardiographic characteristics of an acute infarct may be altered by changes in the QRS complex caused by previous infarction. . . . Of 36 cases of acute coronary thromboses reported three had electrocardiographic evidences of previous infarction." A second report by Wolferth and Wood,[3] in 1935, stated that the patient "may have had a previous cardiac infarct. However, in our experience this has not prevented the localization of the recent lesion."

Barnes,[4] in 1935, wrote as follows:

> The presence of multiple infarction confuses the analysis of the resulting electrocardiographic changes, and in the final interpretation of such cases all possible infarcts must be sought for and their age sequence determined, if possible, by microscopic study. . . . The presence of a large healed infarct of the anterior portion of the heart may lead the examiner to overlook less obvious, but more recent infarct in the posterior base of the left ventricle; this happened in a case recently reported and led to the serious question of the hypothesis of localization. Moreover, it must be borne in mind that the more recent infarct dominates the RST changes in the electrocardiogram.

Sixteen cases in which an old and a recent infarction occurred as proved by autopsy were reported by Sprague and Orgain.[5] In this group with an old and a fresh coronary occlusion, the occurrence of low voltage and "intraventricular conduction defects" became more prominent in 11 cases, and the "T wave changes characteristic of coronary occlusion" were seen in 9 of the 16 cases.

In a discussion of the value of lead IVF in cardiac infarctions, Burton,[6] in 1945, stated:

> In each of eleven cases in this group electrocardiographic changes gave adequate evidence of two or more separate clinical episodes diagnosed cardiac infarction. It is interesting to note that in two of five cases with evidence of an old anterior infarct upon which had been superimposed a recent posterior infarct the well-marked initial negative deflection of QRS_1 that developed as the result of the anterior infarction, completely disappeared leaving a normal R_1 with the advent of the posterior infarct.

Levy and Hyman [7] recently wrote: "Serial studies of patients with multiple episodes of myocardial infarction will usually enable one to localize the lesion accurately, despite the fact that the final electrocardiogram may show QRS changes of the predominant area of infarction only."

From a textbook standpoint, Graybiel and White [8] (1946) report a case of acute posterior myocardial infarction on an old anterior myocardial infarction. Two electrocardiograms are presented. The authors stated:

> These electrocardiograms show very well, the effect on the record of the superimposition of a fresh posterior myocardial infarct on 2½ year old anterior myocardial infarct, rendering the electrocardiogram more normal in appearance because of the elevation of the T waves in Lead I and in the precordial leads even though the heart is actually more damaged than before, there being now two infarcts instead of one.

Katz [9] said:

> The occurrence of a second acute myocardial infarction is shown in the electrocardiogram by the appearance of the S-T stages. If the new infarct is in the area previously damaged, the record may resemble the type already established, otherwise an opposite or composite form will develop.

White [10] mentioned the subject in his book and specifically said:

> It is of especial interest that evidence of previous myocardial infarction in the limb leads may be more or less neutralized by a new infarct and that the electrocardiogram may actually look

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[1] Levine, S. A.: Coronary Thrombosis: Its Various Clinical Features, Medicine 8:245 (Sept.) 1929.

[2] Wood, F. C.; Bellet, S.; McMillan, T. M., and Wolferth, C. C.: Electrocardiographic Studies of Coronary Occlusion; Further Observations on the Use of Chest Leads, Arch. Int. Med. 52:752 (Nov.) 1933.

[3] Wolferth, C. C., and Wood, F. C.: Acute Cardiac Infarction Involving Anterior and Posterior Surfaces of the Left Ventricle; Electrocardiographic Characteristics, Arch. Int. Med 56:77 (July) 1935.

[4] Barnes, A. R.: Electrocardiogram in Myocardial Infarction; Review of 107 Clinical Cases and 108 Cases Proved at Necropsy, Arch. Int. Med. 55:457 (March) 1935.

[5] Sprague, H. B., and Orgain, E. S.: Electrocardiographic Study of Cases of Coronary Occlusion Proved at Autopsy at the Massachusetts General Hospital, 1914-1934, New England J. Med. 212:903 (May) 1935.

[6] Burton, C. R.: The Value of Lead IVF in Cardiac Infarction, Am. Heart J. 30:259 (Sept.) 1945.

[7] Levy, L., II, and Hyman, A. L.: Difficulties in the Electrocardiographic Diagnosis of Myocardial Infarction, Am. Heart J. 39:243 (Feb.) 1950.

[8] Graybiel, A.; White, P. D.; Wheeler, L., and Williams, C.: Electrocardiography in Practice, ed. 2, Philadelphia, W. B. Saunders Company, 1946, p. 202.

[9] Katz, L. N.: Electrocardiography Including an Atlas of Electrocardiograms, ed. 2, Philadelphia, Lea and Febiger, 1946, p. 284.

[10] White, P. D.: Heart Disease, ed. 3, New York, The Macmillan Company, 1948, p. 496.

better when a fresh posterior infarct changes an inverted T wave to an upright position in Lead 1, the result of an old infarct; the precordial leads are more discriminating.

With regard to this problem, Friedberg[11] stated: "The electrocardiographic changes produced by the fresh infarct may be neutralized by those of previous infarcts."

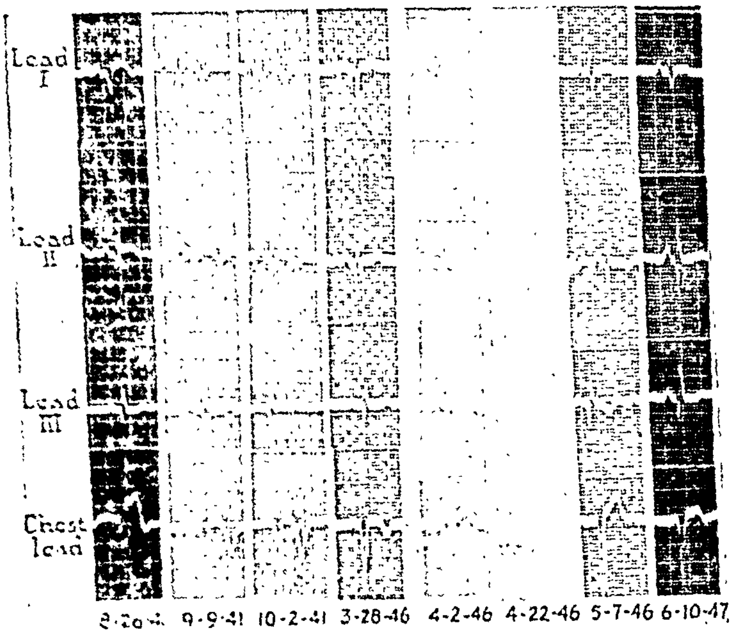

Fig. 1 (case 1).—This series of electrocardiograms was chosen to illustrate the major changes that occurred during the first (anterior) infarction (Aug. 26 to Oct. 2, 1941), the stabilized pattern following this infarction (March 28, 1946), the major changes of the second (posterior) infarction (April 2 to May 7, 1946), and the electrocardiogram revealing residues of both infarctions (June 10, 1947).

He further stated, in regard to "combined anterior and posterior infarction," that "When there is an old infarct on one surface and a fresh infarct on the other the changes due to the recent infarct may be added to or replace hose of previous infarctions."

Bellet and McMillan,[12] in discussing "variations of the usual patterns in posterior infarctions," stated: "Where there has been a previous old anterior infarction acute infarction of the posterior wall is characterized by the typical changes enumerated above." (They described patterns in Q and T waves in leads 2 and 3.) "In addition, T_1 may be inverted and there is frequently an absence of the initial upward deflection with some depression of the R-S-T segment, these additional findings being a result of an old anterior infarct."

MATERIAL AND METHOD

For this study the records of 12 patients with two myocardial infarctions were selected; these patients had an anterior infarction followed by a posterior infarction. The standards for diagnosis were those listed in the "Nomenclature and Criteria for Diagnosis of Diseases of the Heart."[13] The points of diagnosis were protracted precordial pain, variable degree of collapse, fall in blood pressure, mild elevation of temperature, leukocytosis, elevation of sedimentation rate, and the conventional serial electrocardiographic changes.

The charts selected were those in which there were a substantial number of electrocardiograms available for study, with autopsy records in some.

A characteristic single record reads as follows:

G. A. (case 1 in the accompanying table) was born in 1896. His parents lived beyond their 75th year. A brother died of pneumonia; another brother and two sisters outlived him. He had the usual diseases of childhood. At the age of 43, while working as an executive in a packing plant, he was found to have mild hypertension (150/90). At the age of 45 (Aug. 25, 1941), after his evening meal he experienced a bout of severe anterior chest pain, referred into the neck and left arm and lasting 15 minutes. He continued to have pain of similar character throughout the night, with episodes of increased severity. He perspired profusely and was apprehensive.

The following day, he was admitted to Passavant Memorial Hospital, where he remained for six weeks. His temperature for the first eight days ranged from 36.7 to 38.9 C (98 to 101 F). His pulse rate varied from 80 to 120 beats per minute, and his blood pressure varied between 98/64 and 138/80 mm. of mercury. The white blood cell count ranged from 14,000 to 6,500 per cubic millimeter. The Kahn reaction was negative, and the urine was normal. The electrocardiograms taken during this hospital stay showed the conventional changes of an acute myocardial infarction of the anterior type. He returned to work Dec. 1, after a disability of three and one-half months. Between the ages of 46 and 49 (1942 to 1945), his course was uneventful. His blood pressure varied from 120/86 to 140/90.

In his 50th year, on March 28, 1946, after a month's vacation in Cuba, he had recurrent attacks of chest pain and fatigue. An electrocardiogram on April 2 showed distinct changes, and he was again hospitalized at the Passavant Memorial Hospital, where he remained for six weeks. During this stay, his temperature ranged between 36.2 C and 37.4 C (97.2 and 99.3 F). His pulse rate ranged between 70 and 110 beats per minute, and his blood pressure varied between 118/70 and 170/100 mm. of mercury.

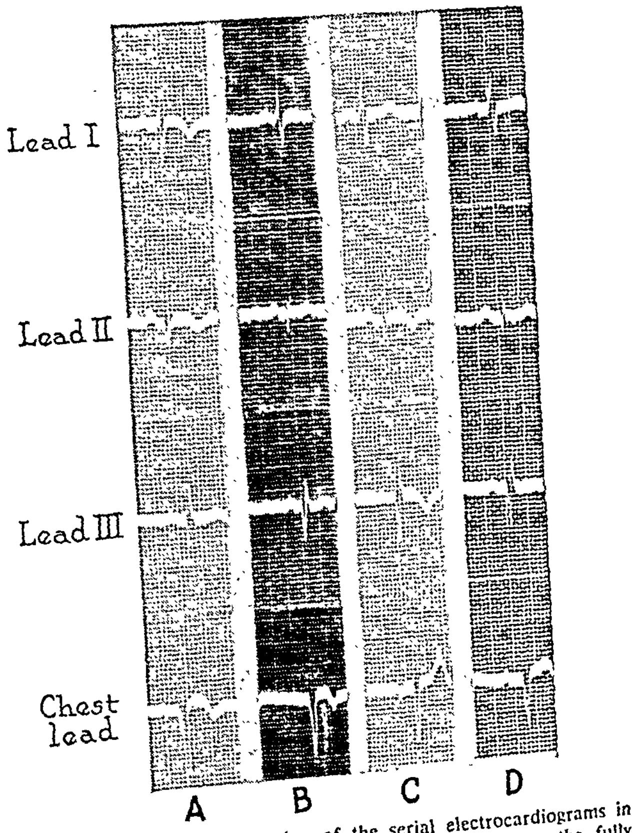

Fig. 2.—This shows a condensation of the serial electrocardiograms in case 9 (see the accompanying table). These tracings illustrate the fully developed anterior pattern (*A*), the residual anterior pattern (*B*), the fully developed posterior pattern that obliterates the residues of the original (anterior) infarction (*C*), and, finally, the residuals of both infarctions (*D*). This typifies the major sequential electrocardiographic changes that are found to occur in our study.

The white blood cell count ranged between 8,900 and 6,900 Again the Kahn reaction was negative and the urine normal The sedimentation of the red blood cells ranged between 30 an

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11. Friedberg, C. K.: Diseases of the Heart, Philadelphia, W. B. Saunders Company, 1949, pp. 242 and 450.

12. Bellet, S., and McMillan, T. M.: in Diagnosis and Treatment of Cardiovascular Disease, edited by W. B. Stroud, ed. 4, Philadelphia, F. A. Davis Company, 1950.

13. Nomenclature and Criteria for Diagnosis of Diseases of the Heart, ed. 4, New York, J. J. Little and Ives Company, 1943, p. 38.

15 mm. in one hour (Wintrobe). Electrocardiograms taken during this episode showed the T wave changes of a posterior wall myocardial infarction.

The patient returned to work in August, 1946. When he was 51 (1947), an allergic dermatitis of the hands developed. In July, 1949, at the age of 53, an unexplained temperature rise occurred and he was hospitalized at the Madison General Hospital. Gradually evidence of cardiac insufficiency with edema developed. Extensive roentgenologic and laboratory studies failed to clarify the cause of his febrile illness. Aureomycin and penicillin therapy had no effect, and sulfadiazine was given with seemingly favorable results. The edema was treated with digitalis and mercurial diuretics, with only fair success.

On Sept. 8, the patient died suddenly, and autopsy was performed by Dr. P. L. Gausewitz.[14] The heart, which weighed 500 gm. and had essentially normal valves, showed occlusion of the left descending coronary artery by an old thrombus. Distad to this point and mediad to the apex of the left ventricle, there was a cardiac aneurysm, 7 by 6 cm., with calcification of the pericardium over this area (the first infarction).

The clinical and laboratory records of the other 11 patients were as comprehensive as the case history presented above. Certain variations appeared, such as the time interval between the first and second infarctions. The minimal time was six weeks and the maximum six years. The clinical course of each patient was modified to some extent by coexisting diseases such as hypertension, peptic ulcer, cholelithiasis, trauma, and a variety of infections. Tabulation of pertinent details of the records in 12 cases of anterior infarction followed by posterior infarction is presented in the accompanying table.

The electrocardiographic records routinely consisted of the three standard limb leads. Tracings from 1937 to 1946 included also a single chest lead, usually CF_4. From 1946 to 1951, the chest leads were routinely CF_2, CF_4, and CF_5, or V_2, V_4, and V_5.

Data in Twelve Cases of Anterior Infarction Followed by Posterior Infarction

Case No.	Age at Time of Infarction, Yr.		Approximate Time Interval Between Infarctions	Other Diseases	Status of Patient at Time of Report	Postmortem Observations (Heart)
	Anterior	Posterior				
1	45	50	4½ yr.	Allergic dermatitis; fever of unknown origin	Dead (third infarction)	Old anterior infarction with cardiac aneurysm; old and recent posterior infarction
2	45	51	6 yr.	Fracture of elbow and femur	Dead (second infarction)	Old anterior infarction involving septum; recent posterior infarction with myocardial rupture; recanalized thrombus right coronary artery
3	54	57	3 yr.	Hypertension; arthritis	Living	....
4	37	38	1 yr.	Alcoholism	Dead (chronic cardiac insufficiency)	Autopsy not made
5	51	54	3 yr.	Hypertension; hemorrhoids; prostatism	Dead (third infarction)	Autopsy not made
6	56	56	6 wk.	Thrombophlebitis	Dead (second infarction)	Autopsy not made
7	57	61	4 yr.	Duodenal ulcer; diverticula of colon; Neisseria gonorrhoeae infection; hypertension	Dead (third infarction)	Autopsy not made
8	47	48	1 yr.	Alcoholism; gallstones; iritis	Dead (third infarction)	Old anterior infarction; old posterior infarction; recent anterior infarction
9	52	54	2 yr.	Psoriasis; obesity; arthritis; hypertension	Living	....
10	57	57	6 mo.	Hypertension	Living	....
11	47	52	5 yr.	Duodenal ulcer; pneumonitis	Dead (cardiac insufficiency)	Autopsy not made
12	57	58	1 yr.	Duodenal ulcer; gallstones; hypertension	Living	....

The left circumflex coronary artery was markedly arteriosclerotic, with hyalinization of the wall and containing a hyalinized calcified thrombus that had been recanalized. There was extensive connective tissue replacement of the myocardium in this area. The right circumflex artery was almost completely occluded at 7 cm. from its origin by what appeared to be old atheromatous lesions (the second infarction).

In the region of the recanalized calcified thrombus, there was a hemorrhage into the wall of the vessel, which had "apparently forced the thrombus out so that it almost occluded the lumen." In this area, there was an acute pericarditis with infiltration of polymorphonuclear leukocytes with acute degenerative changes of the myocardial fibers (the third infarction).

In summary, this case record (substantiated by autopsy) presents the clinical, laboratory, and serial electrocardiographic evidence (fig. 1) of consecutive myocardial infarctions—an anterior followed by a posterior infarction and then, as might be expected, the third infarction, which caused death.

Three of the 12 patients had had electrocardiograms taken prior to the first infarction. The majority of the patients had a few tracings during the first 10 days after the acute episode, and at 7 to 10 day intervals while in the hospital. Between the first and second infarctions, tracings were made at intervals, from one to four per year. During the posterior infarction, the schedule followed was similar to that used during the first infarction. Those who survived had tracings made at variable intervals.

RESULTS

Review of the electrocardiographic records concerned with the primary anterior infarction showed the conventional serial defects. The T waves of lead 1 were typically inverted. There were less definitive changes in

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[14] Personal communication to the authors.

lead 2. Inversion of the T waves occurred in one or more of the precordial leads. There were elevations of the RS-T segments recorded in 6 of the cases, and coving was evident in 11. In 10 of the group, a demonstrable Q wave was present in lead 1, lead 2, or a precordial lead.

A residual pattern of the first infarction was usually established in the interval of weeks, months, or years between the original, anterior, infarction and the second, posterior, infarction. The T wave in lead 1 remained inverted in five cases, blunt or isoelectric in three cases, and definitely upright in four cases. In the precordial leads, the T wave remained persistently inverted in one or more leads in seven cases. There were slight elevations in the RS-T segments in 7 of the 12 records in the limb and/or the precordial leads. The Q waves of the limb leads or the precordial leads noted during the acute stage did not change significantly in the residual patterns.

With the advent of the second infarction (posterior), conventional serial changes were evident. Most obvious and consistent was the inversion of the T wave in lead 3 in all cases. In the eight cases in which there were residual T wave defects in lead 1, the T wave became definitely upright within from one to four days. Coincident with the inversion of the T wave in lead 3 and the rise of the T wave in lead 1, the T waves of the precordial leads assumed an upright position. In five cases there was definite elevation of the RS-T segments in lead 3, and in three cases some elevation of these segments in lead 2. In two cases the RS-T segment in lead 1 showed some depression, and in no case was there a depression of this segment in any precordial lead. Contrary to expectation, in nine cases there was a slight elevation of the RS-T segments in a precordial lead. In no record was there any significant Q wave alteration that existed as a residual of the old anterior infarction.

Six patients survived for at least one year after the second infarction. Three of these had electrocardiographic patterns apparently the result of both infarctions, two had patterns of the posterior infarction only, and one had an essentially normal pattern.

COMMENT

The primary problem of this study is concerned with the diagnosis of a posterior myocardial infarction following an anterior myocardial infarction. There is a possibility that we have missed documenting an infarction before or during the time the patient was under observation. We believe, however, that our clinical records are accurate, and in three cases (see accompanying table) pathological verification was secured.

Electrocardiographically it is evident from our data that a posterior myocardial infarction superimposed on an old anterior myocardial infarction results in expected pattern changes. Old anterior residual defects, particularly T wave inversions in lead 1 and/or precordial leads, show substantial and prompt obliteration (fig. 2). Q waves in limb or precordial leads, however, were not significantly altered. In some patients the residual of the old anterior infarction reappeared after a few months, and in some the newer posterior pattern predominated. In one record, after 14 months, neither old nor recent infarction was evident.

In previous reports, Barnes,[4] in 1935, appears to have been the first to appreciate clearly, in any given patient presenting symptoms and electrocardiographic evidence of myocardial infarction, the importance of a previous infarct. He also recognized that the primary infarction "confuses the analysis" of the resulting electrocardiographic changes of the second infarct and that the more recent infarct "dominates the RS-T changes."

Other authors appear to have had a more casual interest in the second infarction. Of those who mention the subject, in discussions of the electrocardiographic diagnosis of myocardial infarction, Wood and co-workers,[2] Sprague and Orgain,[5] and Levy and Hyman [7] were interested in the QRS changes, and Katz,[9] in the S-T segments. Sprague and Orgain,[5] Graybiel and co-workers,[8] Friedberg,[11] and Bellet and McMillan [12] spoke of the T wave changes. We would be inclined to emphasize the entirety of the QRS-T pattern changes and the progression of the changes in serial tracings. As the second infarct is reflected in the electrocardiographic picture, the defects of the old infarct are substantially obliterated.

The statement of White [10] that the "electrocardiogram may actually look better when a fresh posterior infarct changes an inverted T wave to an upright position in Lead I" must be qualified. Serially, as the T wave in lead 1 becomes upright, the T waves in lead 3 and possibl in lead 2 are inverting (also Q waves in leads 2 and may appear), all of which are significantly abnormal.

The reappearance of the R wave in lead IVF in tw cases with the advent of the posterior occlusion, as reported by Burton,[6] was not evident in our chest leads.

Completion of this electrocardiographic study of the simple combination of an anterior infarction followed by a posterior infarction indicates that further work is necessary. It would be desirable to review a greater number of case records, substantiated by postmortem findings, to corroborate or modify our conclusions. We hope to present further information at our command, dealing with other combinations of two myocardial infarctions: the converse of the present one, a posterior followed by an anterior infarction; or two sequential anteriors; or two sequential posteriors; or others. The ultimate step, obviously, is the sequence of three or more myocardial infarctions.

CONCLUSIONS

1. The clinical and electrocardiographic records of 12 patients who had sustained two myocardial infarctions —first an anterior type, followed later by a posterior type —were studied with reference to the electrocardiographic changes resulting from the second infarction.

2. A second infarction (posterior) engrafted on a firs infarction (anterior) presents typical electrocardiographic changes in the serial tracings, similar to thos conventionally recorded.

3. The residual electrocardiographic pattern of th anterior infarction is substantially obliterated from t electrocardiographic records by the acute changes of t posterior infarction.

4. Months or years after the second infarction, the electrocardiographic pattern may be that of the posterior (second) infarction, a mixture of defects that are the residual of both infarctions, or a nearly normal pattern.

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MEASUREMENT OF BODY WATER

TECHNIQUES AND PRACTICAL IMPLICATIONS

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and
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A satisfactory method for the measurement of total body water in the living subject has long been sought. This is not surprising when one pauses to consider the overwhelming importance of water in the body economy. Water furnishes the preponderant single molecular element in the fabrication of tissue. Its presence is essential to the great mass of chemical reactions on which life depends. It furnishes the means of transport of virtually all enzymes, substrates, metabolites, gases, electrolytes, antibodies, and other substances that must be moved to and from the cell. Cellular function itself is affected by abnormal fluctuations in water content.

The methods that have been employed for the measurement of total body water may be considered under three general headings: desiccation procedures, body specific gravity measurements, and dilution techniques.

DESICCATION

The first estimates of body water content were accomplished with desiccation procedures. One of the early studies in man was reported by Bischoff[1] in 1863. He obtained a value equal to 58.5% of the body weight of the cadaver. More recently, Mitchell and his associates[2] have reported a value of 67.85% in a patient who died in congestive heart failure, and Iob and Swanson[3] have recorded a value of 75% in a stillborn infant. However, valuable as are the data obtained by this method, it is obviously not one that can be employed clinically.

BODY SPECIFIC GRAVITY

In 1941 Behnke[4] reported his studies dealing with specific gravity measurements in young men. He showed that in normal subjects the determination of body specific gravity enabled one to estimate the body fat content. Fat, with its low specific gravity of 0.92, was identified as the primary variable that determined body density. From these findings Behnke formulated his now widely accepted concept of the lean body mass. He conceived of the body as consisting of a fat-free portion of essentially constant gross composition and of a variable quantity of fat. He was not at that time primarily interested in determining values for total body water in his subjects, but he might easily have done so, for body water and body fat bear a reciprocal relationship to each other[5]; that is, the less the fat content, the greater is the percentage of water contained in the body. The specific gravity method of estimating body fat content (and body water) has since been amply confirmed.[6]

DILUTION METHODS

A number of substances have been employed in the search for an ideal material, which when introduced into the body would allow the safe and accurate measurement of total body water. Such a substance should not be toxic to the organism. It should equilibrate rapidly with body water. It should not be selectively stored or excreted, and it should not be metabolized. Its concentration in urine or plasma should lend itself to ready measurement. Among the substances that have been used for this purpose are potassium,[7] thiourea,[8] sulfanilamide,[9] tritium (the radioactive isotope of hydrogen, mass 3),[10] antipyrine,[11] and deuterium oxide (heavy water).[12] For various reasons, the first three of these substances are unsatisfactory. Values for body water obtained with tritium agree well with those obtained with specific gravity measurements,[10] but the long half-life of this radioisotope renders its use in human beings potentially hazardous. In addition, its triple mass raises the question of whether its kinetics of distribution are identical with those of ordinary hydrogen, especially since there is evidence that even deuterium (mass 2) behaves differently in high concentrations.[13] Antipyrine and deuterium oxide are currently regarded with a considerable degree of confidence, and they will be considered in some detail.

Antipyrine was first introduced for this purpose by Soberman and his associates in 1949.[14] These observers demonstrated that after injection it was distributed uniformly throughout the body water and that its concentration in body fluids could be determined accurately with the spectrophotometer. They also showed that the values for body water obtained with antipyrine agree closely with those obtained with deuterium oxide (heavy water). Messinger and Steele,[15] and Osserman and coworkers[6b] compared values for body water obtained with the use of antipyrine with those obtained from specific gravity measurements in the same subjects and found a satisfactory correlation between the two sets of

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values. Nevertheless, there are certain disadvantages inherent in the use of antipyrine: It equilibrates slowly with abnormal fluid deposits, such as ascites; it is selectively bound by protein; and it is metabolized at a rate which varies between subjects. Even so, by employment of suitable corrections an estimate of body water can be made.

Deuterium oxide (heavy water) was first used for the determination of body water by von Hevesy and Hofer, in 1934.[12a,b] It should prove an ideal tracer for water. There is no evidence that it is chemically identifiable by the body tissues, the heavy hydrogen equilibrates rapidly with body water, it is not selectively stored or excreted, and in the amounts used it is not toxic to the organism. Moore [12c] introduced its use in the United States in 1946. He and others [16] have shown in desiccation studies that the volume of dilution of deuterium oxide corresponds closely to the total body water.

The measurement of total body water with deuterium oxide is accomplished as follows. A measured quantity of the isotope (usually 25 to 50 gm. of 99.8% deuterium oxide diluted to 100 ml. with 0.85% sodium chloride solution) is injected intravenously, a minimum of one and a half hours is allowed for equilibration of the deuterium with body water, and a blood specimen is then drawn for analysis. The plasma water is first purified by appropriate distillation, and the distillate is then analyzed for deuterium concentration by an elaborate falling drop technique.[17] This method for the determination of deuterium concentration, accurate under optimal conditions to from one to two parts per million, consists of three essentially separate components. The first is an extremely sensitive thermoregulator that maintains the temperature of the water bath to within approximately 0.001 C. The second is an elaborate pipet mechanism that delivers a drop of constant size. The third is a distillation process that achieves a very high degree of purification of plasma water.

INFORMATION DERIVED FROM BODY WATER MEASUREMENTS

Body Water and Body Fat.—At the time his studies establishing the relationship between body specific gravity and body fat were reported, Behnke proposed, on a theoretical basis, that body water and body fat must bear an inverse relationship to each other. To test this concept, we [12d] selected a group of healthy adult male and female human subjects ranging in size from the very lean to the very obese. Heavy water was injected intravenously and, after equilibration with the body water, the concentration of deuterium oxide in the water distilled from plasma samples was measured by means of the accurate specific gravity method described above. The leanest male subject had a total body water value of 71% of body weight (44.5 liters), and the most obese male subject had a total body water value of 42% (48.3 liters). Similarly, the leanest female subject had a total body water value of 68% and the most obese female subject a body water value of 50%. However, the most obese female subject was not clinically as obese as the most obese male subject. In general, the values for percentage body water of female subjects appeared to be somewhat lower than those for male subjects of corresponding weight and habitus. The average value for body water for eight healthy men was 60% of body weight. The subjects who individually had values in the neighborhood of 60% were merely of good nutrition, being neither skinny nor fat.

Other workers have reported somewhat different values for body water. Using antipyrine, Soberman and his associates [14] obtained values for body water in normal subjects ranging from 43% to 58%, and these findings agreed well with those afforded by deuterium and specific gravity measurements. It is not clear whether an attempt was made to select subjects with a wide range of body weight and habitus. In a more recent study, Osserman and his co-workers compared the body specific gravity and antipyrine methods in a group of healthy young men of increasing degrees of obesity. They found that the body water ranged from 43% in the most obese to 72% in the leanest, and these values compare very favorably with those obtained by us (42% and 71%, respectively). These workers found, also, that the value for total body water was almost constantly 71% of the lean body mass. Schloerb and his associates [18] have reported an average value of 61.8% for body water in healthy young adult men and 51.9% in healthy young adult women. Again, these findings correspond very closely with the average of 60% in males and 55% in females found by us. It therefore becomes necessary to revise our concepts concerning the average percentage value for the body water content in healthy adults. The commonly used value of 70% is acceptable only in the unusually lean subject.

The concept of the lean body mass may prove useful clinically. Schloerb and his co-workers [18] found the total body water to be more closely correlated with surface area and oxygen consumption than with the other physical indexes examined. While it would be erroneous to assume that fat is metabolically inactive, it is probable that the lean body mass is metabolically more active. Thus, if the lean body mass can be used as a basis for a comparison of the metabolic activity of different subjects, it may be possible to adjust certain hitherto perplexing "discrepancies" in basal metabolic rates. Projecting this line of thought further into the clinical field, it would seem more reasonable to calculate dosages of drugs on the basis of the lean body mass than on body

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weight, as is now so frequently done. The markedly obese patient should not receive powerful drugs on a dosage schedule based purely on body weight. It would be preferable to employ some such convention as the "ideal" body weight.

BODY FLUID COMPARTMENTS

Hardy, Sen, and Drabkin [19] have examined the relationships of total body water, "extracellular" fluid, and plasma volume in eight healthy male subjects selected to cover a wide range of body fat content. Levitt and Gaudino,[20] Steele,[6] and Schloerb and his associates [18] have also examined these body fluid compartments. Although numerous materials have been employed to measure the "extracellular" fluid by the dilution method (among them thiocyanate, bromide, sodium, chloride, and inulin) only sodium thiocyanate and inulin have had wide use. Inulin is generally accepted as perhaps the most reliable substance thus far used for this purpose. It yields values for extracellular fluid in the neighborhood of 15% of body weight.[5] However, the analytical procedures involved in the use of this material render it impractical for general use. The use of sodium thiocyanate,[21] on the other hand, while subject to certain objections, has the solid merit of being readily available and its analysis in body fluids is a simple precedure. Thus, it is easily applicable clinically. The chief disadvantage of using sodium thiocyanate is that a fraction of the thiocyanate ion unquestionably enters the cells in normal subjects, and in certain pathological states this intracellular fraction may be very large.[22] Obviously, any appreciable loss of thiocyanate ion into the cells will result in a value for the extracellular fluid that is erroneously high. Nevertheless, for practical reasons we have employed sodium thiocyanate to estimate the extracellular fluid in normal subjects.[19] Thiocyanate space was found to be more closely correlated with body water than with body weight. Two very lean male subjects both had "extracellular" fluid values of 22% of actual body weight and 32% of total body water. These two subjects had total body water values of 69% and 71%, respectively, of body weight. The most obese male subject, on the other hand, had a thiocyanate space that was only 16% of the body weight but was 38% of the body water. The body water was only 42% of the body weight. Thus, the thiocyanate space represented a greater fraction of the total body water in the obese than in the very lean subjects. The explanation for this is not apparent. We know of no evidence that equilibration of sodium thiocyanate is less complete in the obese than in the lean subject. The possibility of a greater penetration of the fat cells by the thiocyanate ion was considered. Neutral fat within other cells of the body in the obese person might conceivably displace cellular water and thus augment the relative size of the extracellular volume. Finally, the space between the fat cells themselves would be available for extracellular fluid. More quantitative information concerning the "extracellular" fluid would be of substantial aid in estimating fluid replacement in states of dehydration, particularly since this fluid compartment is more rapidly depleted in disease states than is the intracellular fluid.

To preserve an open mind, it is important to think of the fluid compartments as physiological rather than anatomic entities. The use of radioisotopes has greatly altered previous concepts concerning the permeability of the cell membrane to certain electrolytes, particularly sodium, potassium, and chloride. It is now realized that these ions can, under appropriate physiological conditions, cross the cell membrane readily.

Plasma volume has been studied more intensively than has any other body fluid compartment. Yet, a dependable clinical guide for estimation of what the normal plasma volume of a given subject should be is still not available. Because of the unacceptability of values obtained for the plasma volume of normal persons when body weight was used as the basis for calculating this value, a study [18] was made of the correlation between plasma volume and total body water in a group of healthy adult men of widely different degrees of obesity. Plasma volume ranged from 2.75% (in the very fat) to 5.65% (in the very lean) of body weight, with a mean of 4.24% and an average deviation of 22%. Expressed as a percentage of total body water, however, plasma volume ranged from 6.2% in the most obese to 7.8% in the leanest, with a mean of 6.96% and an average deviation of 7%. Thus, plasma volume would appear to be more closely correlated with body water than with body weight.

DYNAMICS OF WATER FUNCTION

The measurement of body fluid compartments has been only one of the avenues of research opened by the availability of a satisfactory tracer element for water. Such studies may be characterized as "static" in nature. There are other types of investigation that are aimed at illuminating "dynamic" or "kinetic" aspects of water metabolism. As an example, one may cite determinations of the half-time or biologic half-life of a molecule of water in the body. By administering deuterium oxide orally and measuring rates of urinary excretion, von Hevesy and Hofer [12a] estimated that the length of time the average molecule of water remained in the body of their subject was approximately nine days. In a single subject, one of us (J. D. H.)[23] found this period to be 11 days, and in a number of subjects Schloerb and his co-workers [18] arrived at an average figure of 9.3 plus or minus 1.5 days. Clearly, however, this figure must necessarily be influenced by rates of water ingestion and excretion; that is, the more rapid the water turnover in the body, the shorter is the time the average molecule of water will remain in the body. Accordingly, one would expect the half-times to be different in warm and cold climates and to be abnormal in patients with febrile illnesses, uncontrolled diabetes mellitus, and thyrotoxicosis.

Body Weight Changes in Surgical Patients.—Even momentary reflection suggests that the rapid weight loss that follows many surgical procedures must often represent simple water and salt excretion. One of us (J. D. H.) has recently studied a patient who was admitted to the hospital in a state of extreme weakness and emaciation whose diagnosis was that of simple esophageal diverticulum with severe dysphagia.[24] Before treatment was begun her weight was 69 lb. (31.3 kg.) and her body water value was 22 liters. She was then tube fed for 17 days. During this time her general condition and appearance improved enormously, and her weight increased to 86 lb. (39 kg.). Immediately prior to successful surgery, her body water was again determined with deuterium oxide and was found to be 29 liters. Thus, despite the fact that clinical edema was not manifest, her weight gain consisted very largely of water. This single example is cited to indicate the relatively large variations in body water content that may escape clinical detection.

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Heart Failure.—Rates of water mobilization and distribution in cardiac decompensation offer a fertile field for investigation. In such patients the normal relationships between percentage body water and body weight do not hold. For example, we have determined the body water of frankly edematous patients and found their percentage of body water no greater than that in healthy lean subjects. The explanation of this lies, of course, in the fact that these patients were somewhat obese. If some simple means of determining their body fat content were available it would be found that their percentage of body water was appreciably greater than 71% of the lean body mass.

Other Factors That May Be Examined with Deuterium Oxide.—Rates of spinal fluid formation may be studied with the use of heavy water as a tracer. Fluid kinetics in thermal burns and shock represent one of the most important factors associated with the management of these injuries, and several surgical clinics are actively investigating these problems. The relationships between the blood supply of the bowel and the movement of water and electrolytes across its cellular membranes will surely be subjected to a more quantitative scrutiny than has been possible in the past.

SUMMARY

The several types of measurements that may be employed for the determination of total body water are outlined. The use of dilution methods has made available valuable information concerning the relation of body fluid compartments to body fat and to each other. Deuterium oxide (heavy water) appears to be an excellent tracer substance for water, and its availability has made possible a more critical examination of rates of water turnover in the body and rates of transfer of water across various cell membranes. It is reasonable to anticipate that our knowledge of body fluid compartments and of water kinetics in the human body will be enormously extended by the use of isotopes.

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[24] Hardy, J. D.: Unpublished data.

INCREASED INTRACRANIAL PRESSURE IN EMPHYSEMA CAUSED BY OXYGEN INHALATION

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Respiratory depression followed by delirium and coma has developed in several patients with chronic pulmonary emphysema while they were receiving oxygen therapy in this hospital. I have studied some of these patients as well as some normal subjects in an effort to determine the mechanism of production of this phenomenon.

Three normal subjects and four patients with chronic lung disease were studied. Their ages and diagnoses are listed in table 1. Brief summaries of the clinical states of the four diseased patients are given below.

PROCEDURE

The subject lay on his left side, and an indwelling needle was placed in the brachial artery. A lumbar puncture needle was inserted, connecting the subarachnoid space to a spinal fluid monometer. The subject breathed through a rubber mouth piece attached to a three-way valve by which connection could be made either to a 9L spirometer containing 100% oxygen or to room air. During an initial period of rest and room air breathing, basal spinal fluid pressure readings were obtained. The mouth piece valve was then turned to connect the subject to the spirometer that contained 100% oxygen, and a soda lime-carbon dioxide absorber. An attached kymograph recorded the respiratory excursions. Spinal fluid pressures were recorded every 30 seconds throughout all procedures. When the effect of oxygen inhalation had been observed, the valve was again turned to give the subject access to room air. The carbon dioxide absorber was then removed from the spirometer and the tank refilled with 100% oxygen. When the valve was again turned, the subject was made to rebreathe oxygen from the spirometer, which, lacking a carbon dioxide absorber, came gradually to contain increasing tensions of carbon dioxide. The spirometer gas was analyzed[1] at the end of rebreathing and found in all the carbon dioxide experiments to contain 6 to 8 vol.% of carbon dioxide. At appropriate intervals throughout the procedures, arterial blood samples were drawn with care being taken to exclude oxygen and stored in ice[2] until they were analyzed for oxygen and carbon dioxide in the Van Slyke manometric apparatus.[3]

From the Cardiac Laboratory, The Mary Imogene Bassett Hospital.

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[3] Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry Methods, Baltimore, Williams & Wilkins Company, 1932.

RESULTS

Ventilation (Table 2).—During oxygen inhalation the three normal subjects had an average increase in respiratory volume of 15% and a 16% increase in tidal volume. This slight increase in ventilation produced by oxygen in normal subjects is consistent with the findings of others.[4] Patients L. K. and J. W. showed slight increase in minute volume during oxygen inhalation, while the tidal volume of one decreased and that of the other increased slightly. There was no significant change in the carbon dioxide content of their arterial blood, which initially had been normal. Both subjects appeared to be consciously controlling their respirations during the procedure.

Patients J. H. and K. B. showed a prompt decrease in both minute volume and tidal volume during oxygen inhalation. The change in minute volume was less than the decrease in tidal volume, owing to a slight increase in respiratory rate. The arterial carbon dioxide content was abnormally high in both subjects initially (58 vol.%). It rose to 65 vol.% in K. B. during oxygen inhalation. A second blood sample was not drawn from J. H.

Table 1.—*Data in Cases Studied*

Subject (Sex)	Age, Yr.	Diagnosis
Normal 1 (F)	18	Convalescent from poliomyelitis; heart and lungs normal
Normal 2 (F)	64	Convalescent from cerebrovascular accident; heart and lungs normal
Normal 3 (M)	32	Convalescent from poliomyelitis; heart and lungs normal
Patient J. H. (F)	55	Cor pulmonale secondary to pulmonary fibrosis and emphysema
Patient K. B. (F)	65	Chronic obstructive emphysema secondary to bronchial asthma
Patient L. K. (M)	70	Chronic obstructive emphysema secondary to bronchial asthma
Patient J. W. (M)	71	Chronic obstructive emphysema secondary to bronchial asthma

Table 2.—*Effect of 100% Oxygen Inhalation on the Respiration of Normal Subjects and Patients with Chronic Lung Disease*

	Minute Vol. (Cc.) During First Minute	Minute Vol. (Cc.) at Time of Maximum Change	Change in Minute Vol., %	Tidal Vol. (Cc.) First Minute	Tidal Vol. (Cc.)	Change in Tidal Vol., %
Normal 1	4,500	5,000 (3 min.)	+11	300	340 (3 min.)	+13
Normal 2	5,000	5,700 (5 min.)	+14	240	265 (5 min.)	+10
Normal 3	6,080	7,260 (7 min.)	+19	380	480 (7 min.)	+26
Patient J. H.	5,000	2,750 (12 min.)	—45	340	170 (12 min.)	—50
Patient K. B.	3,920	3,160 (7 min.)	—19	216	110 (7 min.)	—49
Patient L. K.	10,500	11,000 (6 min.)	+10	585	502 (6 min.)	—14
Patient J. W.	7,500	8,500 (11 min.)	+13	418	439 (11 min.)	+ 5

Spinal Fluid Pressure.—The three normal subjects and patients L. K. and J. W. showed no significant change in spinal fluid pressure during oxygen inhalation (fig. 1).

The other two patients, J. H. and K. B., had, while breathing oxygen, a prompt, steady rise in spinal fluid pressure to over 600 mm. and 500 mm. of water, respectively. Both of these patients had elevated spinal fluid pressures initially and were the subjects in whom respiratory depression occurred, as described above. The spinal fluid pressure slowly began to fall toward its initial level when the oxygen inhalation was stopped in patient J. H. but was elevated again by the inhalation of 6% carbon dioxide. In patient K. B. the high spinal fluid pressure was rapidly reduced to a level below its initial pressure by hyperventilation with 100% oxygen. The patients had no symptoms or change in mental status during the acute elevations in spinal fluid pressure produced in these experiments.

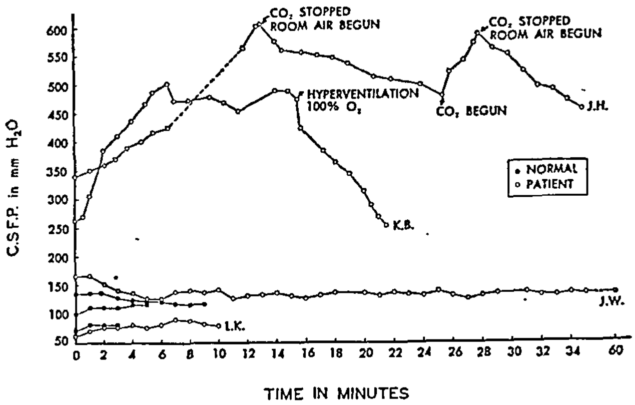

Fig. 1.—Effect of 100% oxygen inhalation on the cerebrospinal fluid pressure of patients with pulmonary emphysema and on normal subjects.

Figure 2 shows the effect of the inhalation of 6% to 8% carbon dioxide on the spinal fluid pressure of the three normal subjects. Carbon dioxide produced a rise in spinal fluid pressure to high levels. The rate of rise in pressure paralleled the accumulation of carbon dioxide in the spirometer during rebreathing. The pressure fell promptly to normal or below when carbon dioxide inhalation was discontinued. Except for an increase in respiratory minute volume, these subjects had no symptoms during these procedures.

REPORT OF CASES

Case 1.—Patient J. H., a 55-year-old housewife, was admitted to the hospital in 1949, with a history of frequent attacks of bronchial asthma for the previous 22 years. Two years before admission her asthma became more severe, and she began to notice cyanosis and a florid complexion. She had frequent headaches, dyspnea on exertion, unproductive cough, paroxysmal nocturnal dyspnea, and diminution of vision. Physical examination at that time revealed a moderately cyanotic woman in respiratory distress. There were bilateral papilledema, signs of

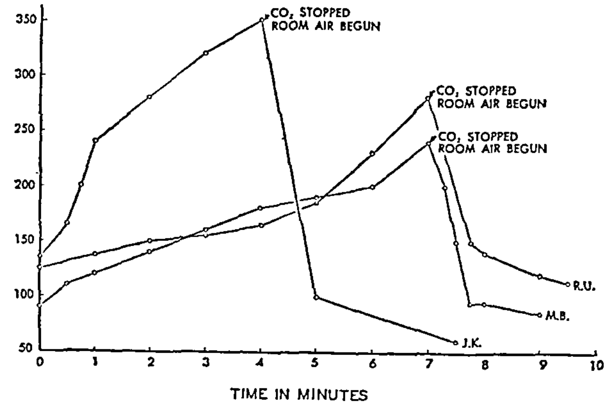

Fig. 2.—Effect of 6% to 8% carbon dioxide inhalation on the cerebrospinal fluid pressure of normal subjects.

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[4] Dripps, R. D., and Comroe, J. H., Jr.: The Effect of Inhalation of High and Low Oxygen Concentrations on Respiration, Pulse Rate, Ballistocardiogram and Arterial Oxygen Saturation (Oximeter) of Normal Individuals, Am. J. Physiol. 149: 277, 1947.

pulmonary emphysema, and congestive heart failure, with enlargement of the left ventricle and pulmonary conus seen on x-ray. Her blood pressure was 172/110, hemoglobin 20.6 gm. per 100 cc., red blood cell count 6,590,000, and venous pressure 16 cm. of water.

She was readmitted in October, 1949, at which time her clinical condition was essentially as before. A lumbar puncture showed an initial pressure of 350 mm.; manometric studies were normal. The spinal fluid protein, chloride, and sugar levels were also normal. There was electrocardiographic evidence of right ventricular hypertrophy. Her arterial oxygen saturation was 11.7 vol.% and carbon dioxide content 40 mEq. per liter. She was treated for bronchial asthma and had several phlebotomies.

She was again admitted in October, 1950, because of a severe asthmatic attack. Because of her intense cyanosis, she was given 100% oxygen by nasal catheter shortly after admission. The cyanosis disappeared after a few minutes, but she soon became lethargic and after seven hours of oxygen therapy lapsed into deep coma. Her respirations were shallow. The oxygen therapy was slowly discontinued, and she concomitantly regained consciousness. It was because of this episode that procedures described in this paper were performed.

During the past year this patient has gotten along well on restricted activity and treatment for asthma. The secondary polycythemia has required occasional phlebotomies.

Case 2.—K. B., a 65-year-old housewife, was admitted to the hospital in November, 1950, with the chief complaint of asthma of 36 years' duration. Her asthma began at the age of 29, after which she had intermittent attacks but was free of symptoms between them. Five months before admission, after an upper respiratory infection, an asthmatic attack started, which continued more or less uninterruptedly and brought about her admission to the hospital.

She presented physical signs of pulmonary emphysema. There were expiratory wheezes heard throughout both lung fields. There was slight cyanosis. Her vital capacity was 850 cc., venous pressure 11 cm. of water, circulation time 24 seconds, hemoglobin 16 gm., red blood cell count 5,890,000, and venous carbon dioxide content 29 mEq. per liter. Fluoroscopic examination of the heart showed generalized enlargement, with abnormally prominent pulmonary vessels. The patient's cerebrospinal fluid pressure response to oxygen inhalation was measured on her eighth hospital day, at which time her asthmatic attack had ended.

Case 3.—L. K., a 65-year-old retired farmer, was seen in October, 1950, because of a severe asthmatic attack of four days' duration. He had had bronchial asthma for many years. There were physical signs and roentgenographic evidence of obstructive emphysema. His heart was of normal size, and there were no signs of congestive failure. His carbon dioxide content and hemoglobin were normal. He was treated for asthma, after which he became symptom-free, except for exertional dyspnea. The studies reported were done five months later.

Case 4.—J. W., a 71-year-old retired farmer, was admitted to the hospital for the 11th time in October, 1950, because of a severe asthmatic attack. Bronchial asthma began at the age of 20. In recent years he had had frequent attacks, between which he was incapacitated by pulmonary insufficiency. In addition to physical signs of bronchial asthma and obstructive pulmonary emphysema, he had roentgenographic evidence of numerous large emphysematous blebs arising from the upper lobe of the left lung. There was no cardiac enlargement or signs of congestive failure. Laboratory findings were as follows: hemoglobin 14 gm. per 100 cc., red blood cell count 5,240,000, and carbon dioxide content 46 mEq. per liter. He was treated for asthma and discharged from the hospital. In December, 1950, the studies reported here were performed. He was apprehensive during the procedure and seemed to be voluntarily controlling his respirations.

COMMENT

Undesirable effects of oxygen inhalation in certain patients with pulmonary disease have been reported by others.[5] Barach [5a] described mental confusion and coma occurring in patients with chronic hypoxia when they were given high concentrations of oxygen to breathe. His patients all recovered consciousness, in spite of the continued oxygen therapy. Davies and Mackinnon [5c] described neurological signs, coma, and death with elevation in the spinal fluid pressure during oxygen therapy in certain patients with cor pulmonale. Comroe and coworkers [5e] studied the effects of oxygen on 65 chronically anoxemic patients; mental changes ranging from somnolence to coma occurred in nine. These patients showed diminished ventilatory volume, increased arterial pCO_2, and acidosis during oxygen therapy. Beale and co-workers [5f] reported respiratory acidosis and coma produced by oxygen administered to a patient with asthma but without pulmonary emphysema or fibrosis.

The inhalation of high concentrations of oxygen in normal subjects depresses respiration slightly during the first two minutes; there then follows an increase in minute volume probably owing to the irritant effects of oxygen in high concentration.[4] Table 2 shows the respiratory stimulation that occurred in our normal subjects. An initial depression was not demonstrable, since a preceding spirometric record of room air breathing was lacking.

In two of the subjects with pulmonary emphysema, J. H. and K. B., the 50% decrease in tidal volume during oxygen inhalation is abnormal. It indicates that in these patients anoxia was an important respiratory stimulant, which, acting presumably through the aortic and carotid chemoreceptors, had assumed much of the function of stimulation normally carried by carbon dioxide and the medullary centers. When the anoxic stimulus was removed by oxygen inhalation, respiratory depression ensued in spite of a rising arterial carbon dioxide tension. This is reminiscent of the suppression of carbon dioxide sensitivity of the respiratory centers by barbiturates and consequent respiratory depression produced by oxygen administration in patients poisoned by these drugs.[6]

Patients with chronic pulmonary emphysema are characteristically less sensitive than normal to the respiratory stimulus from carbon dioxide [7] and often have respiratory acidosis from carbon dioxide retention without an increased respiratory rate. This insensitivity of the respiratory centers may be reversible, at least in some cases. This is suggested by Beale's [5f] case of asthma without chronic lung disease and by the fact that Barach's [8] patients did recover from respiratory depression and coma caused by oxygen inhalation in spite of continued oxygen administration. These observations further suggest that carbon dioxide insensitivity may be promoted by anoxia.

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[5] (a) Barach, A. L.: Impairment in Emotional Control Produced Both by Lowering and Raising the Oxygen Pressure in the Atmosphere: Fundamental Relation Between Psychic and Somatic Processes, M. Clin. North America **28:** 704, 1944. (b) Godfrey, L.; Pond, H. S., and Wood, F. C.: The Millikan Oximeter in the Recognition and Treatment of Anoxemia in Clinical Medicine, Am. J. M. Sc. **216:** 605, 1948. (c) Davies, C. E., and Mackinnon, J.: Neurological Effects of Oxygen in Chronic Cor Pulmonale, Lancet **2:** 883, 1949. (d) Donald, K.: Neurological Effects of Oxygen: Letters to the Editor, ibid. **2:** 1056, 1949. *Mental Changes* (e) Comroe, J. H., Jr.; Bahnson, E. R., and Coates, E. O.: *Occurring in Chronically Anoxemic Patients During Oxygen Therapy,* J. A. M. A. **143:** 1044 (July 22) 1950. (f) Beale, H. D., and others: Delirium and Coma Precipitated by Oxygen in Bronchial Asthma Complicated by Respiratory Acidosis, New England J. Med. **244:** 710, 1951.

[6] Beecher, H. K., and Moyer, C. A.: Mechanisms of Respiratory Failure Under Barbiturate Anesthesia (Evipal, Pentothal), J. Clin. Investigation **20:** 549, 1941.

[7] Scott, R. W.: Observations on the Pathologic Physiology of Chronic Pulmonary Emphysema, Arch. Int. Med. **26:** 544 (Nov.) 1920.

[8] Barach, A. L.: Physiologic Therapy in Respiratory Diseases, ed. 2, Philadelphia, J. B. Lippincott Company, 1948, p. 188.

Carbon dioxide inhalation dilates cerebral blood vessels,[9] increases cerebral blood flow,[10] spinal fluid pressure,[11] and increases the rate of spinal fluid secretion.[12]

We have demonstrated, as did Davies and Mackinnon,[5c] that the carbon dioxide retention in these patients increases the intracranial pressure as does carbon dioxide inhalation in normal subjects. We interpret this as the result of increased cerebral blood flow from vasodilatation. The assumption of increased cerebral flow on the basis of measurements of increased intracranial pressure is justified under the acute circumstances of these experiments.[13]

The demonstration of increased intracranial pressure in these patients does not explain the production of their mental changes. There are, however, reports of experiments that suggest the explanation of these changes and illustrate the complexity of the problem.

In patients with increased intracranial pressure from brain tumor, there is a critical level of cerebrospinal fluid pressure beyond which cerebral blood flow and cerebral oxygen consumption decrease and coma ensues.[14] This level is 450 mm. of water. Below it the increased cerebral vascular resistance that results from the raised intracranial pressure is compensated for by reflex increase in mean arterial blood pressure; cerebral metabolism and consciousness are thereby maintained. Oxygen inhalation in J. H. and K. B. resulted in cerebrospinal fluid pressures above this critical level. We therefore cannot dismiss the possibility that coma may have resulted from diminished oxygen utilization as a result of the high intracranial pressure per se. If so, the diminished cerebral oxygen consumption and mental changes are in part a function of the duration of intracranial pressure increase, because Patterson and co-workers [10c] found that patients with pulmonary emphysema have a high cerebral blood flow and low cerebral oxygen consumption. When the patients were given high concentrations of oxygen to breathe for 30 minutes, their cerebral blood flow increased without change in cerebral oxygen consumption or mental status. Since the coma that occurs in patients with emphysema who have been given oxygen takes hours to develop, the short duration of their experiments makes them inconclusive.

Kety and Schmidt [10b] have studied the effects of 5% to 7% carbon dioxide and 10% oxygen inhalation for 15 to 30 minutes on the cerebral metabolism of normal subjects. Both hypercapnia and anoxia produce a fall in cerebral vascular resistance and consequent rise in cerebral blood flow. These changes occurred without any fall in cerebral oxygen consumption in the face of the development of mental changes. This surprising result illustrates the complexity of the problem of cerebral function, and the authors state:

> The conclusion is apparent that the higher psychic functions are associated with biochemical changes so subtle and complex as to render any attempt to describe them in terms of mere oxygen utilization no more adequate than to predict the fidelity of a radio by its power requirements.

Studies on cerebral metabolism in diabetic acidosis and coma [15] showed certain relationships between cerebral metabolism and function that are applicable here. Patients in diabetic acidosis without coma have a slightly increased cerebral vascular resistance and decreased cerebral blood flow. In those patients in coma the cerebral vascular resistance is diminished and the cerebral blood flow increased. The greatest difference between these two groups is in cerebral oxygen consumption, which, although depressed in both groups, is much lower in comatose patients. There is a critical level of cerebral oxygen consumption of 2.1 cc. per 100 gm. of brain tissue per minute (normal, 3.3 cc. per 100 gm. per minute), at or below which unconsciousness occurs. The cerebral oxygen consumption is almost completely unrelated to the cerebral blood flow but is well correlated with arterial pH. This suggests that in patients with emphysema and carbon dioxide retention the fall in pH that accompanies hypercapnia may after sufficient time diminish cerebral oxygen consumption by interference with its utilization and effect coma in spite of a greater than normal oxygen supply delivered to the brain.

Other possible mechanisms that have been discussed [5e] are carbon dioxide narcosis, cerebral vasospasm, reflex cerebral depression from high oxygen tension, or direct cerebral depression from oxygen.

We have demonstrated that the intracranial pressure increase is not the result of high oxygen tension itself. K. B. (fig. 1), in whom the spinal fluid pressure was lowered by hyperventilation with 100% oxygen, illustrates this and suggests a method of prevention, namely, that patients with ventilatory insufficiency who need oxygen in high concentrations be encouraged to breathe deeply. If they are conscious and cooperative, this can be accomplished by frequent reminder from an attendant at the bedside for a period long enough to tide the patient over an emergency. If they are not able to follow the directions for breathing given by an attendant, then artificial respiration should be given. In case they do not need high concentrations of oxygen from the start of therapy, following Barach's plan [5e] of gradually increasing the concentration of inhaled oxygen over a period of days would suffice. Unfortunately, one cannot predict with certainty which patients with pulmonary emphysema will suffer the adverse effects of oxygen; therefore all such patients should be under close supervision and their mental status determined at frequent intervals.

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SUMMARY

1. In some patients with pulmonary emphysema the administration of high concentrations of oxygen results in mental changes ranging from somnolence to coma and sometimes death.

2. The effect of oxygen and carbon dioxide inhalation on the pulmonary ventilation and spinal fluid pressure of four patients with chronic pulmonary emphysema and three normal subjects has been studied in an effort to elucidate the mechanism of production of this phenomenon.

3. The inhalation of carbon dioxide by normal subjects increases the spinal fluid pressure through cerebral vasodilatation. In two of the patients with pulmonary emphysema, oxygen inhalation resulted in respiratory depression, carbon dioxide retention, and increased intracranial pressure.

4. The possible mechanisms for the production of this sequence of events and the mental changes in these patients are discussed.

5. Occasional periods of hyperventilation during oxygen therapy will prevent the development of these undesirable effects.

LATE RESULTS OF ARTIFICIAL PNEUMOPERITONEUM IN PULMONARY TUBERCULOSIS

TEN YEAR FOLLOW-UP STUDY OF TWO HUNDRED PATIENTS

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and
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Early results of the use of artificial pneumoperitoneum in the treatment of 200 patients during recent years were reported by Dr. Edwin S. Bennett in April, 1938.[1] From his experience, he concluded that this procedure should be used in the treatment of several groups of patients, which he described. The present paper reviews the status of the same 200 patients 10 years after pneumoperitoneum in order to determine the usefulness of this treatment. Twenty-seven physicians participated in this series of treatments, in which there were no fatalities directly attributable to the treatment. Most of the patients had a poor prognosis before pneumoperitoneum. In almost 90%, the disease was far-advanced, and abundant tubercle bacilli were usually found in the sputum. Other forms of collapse had been tried unsuccessfully in all but nine of the patients. Artificial pneumothorax had been attempted in 90% of the patients. This procedure was often repeated, and it was usually found that there was no free space or only a small, ineffectual collapse. Phrenic paralysis, usually temporary, had been performed in 172 patients, and 3 of these patients had a thoracoplasty operation before the pneumoperitoneum.

The average period of sanatorium care before the pneumoperitoneum was nearly a year. The average duration of the pneumoperitoneum until discharge from the sanatorium was 7.3 months, but many patients continued the treatment with refills after discharge, for from 1 to 40 months. The refills done at the sanatorium averaged 800 cc. of air, and the average interval between refills was one week. In several instances in which, for various reasons, pneumoperitoneum had been abandoned, it was reestablished without difficulty. Pneumoperitoneum had been established in several patients on whom, some years previously, abdominal operations had been performed, after which there had been drainage for a period of months.

Complications that caused marked discomfort and led to discontinuance of treatment arose in 20 patients, or 10% of the cases. In three instances, pneumoperitoneum was discontinued because of the appearance of hernia, although in several other patients, the hernia did not necessitate cessation of treatment. Four patients complained of immediate extreme, but transitory, epigastric and shoulder pain. Today, the latter symptom is considered a favorable prognostic sign. Two patients had extensive subcutaneous emphysema. In seven patients, pronounced distention of the abdomen developed the day after refills were administered; this can be ascribed either to meteorism or to peritoneal effusion. Two patients had pronounced abdominal adhesions that were visible on the roentgenogram, and, in three patients, fluid developed to a degree that required aspiration. In another patient the procedure had to be discontinued because of obliterative peritonitis. A total of 31 patients suffered from hydropneumothorax prior to the initial pneumoperitoneum, and, it developed in 8 more after treatment was begun.

Free fluid, usually clear but occasionally turbid, was found in the abdomen in over half of the 20 patients on whom autopsies were performed, varying from 150 to 1,500 cc. in volume. In addition, a plastic exudate covering the viscera and matting together the coils of the intestines was found in two patients. In other patients, more organized reactions appeared in the form of fibrotic plaques or thickening, which were usually discrete and scattered but were occasionally confluent and extensive, covering the exposed viscera entirely. Although autopsy often revealed considerable peritoneal fluid or thickening, no such observation was made in the five patients on

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From the Olive View Sanatorium.

[1] Bennett, E. S.: Induced Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis: Review of 200 Cases at Olive View Sanatorium, Journal-Lancet 58: 187 (April) 1938.

whom laparotomies were performed during the course of the artificial pneumoperitoneum, and, postoperatively, all the patients had the pneumoperitoneum reestablished.

At the time Dr. Bennett made his report, in 1938, 27 of the 200 patients had died in the sanatorium, and 5 others had died after discharge. Since then, an additional 17 patients have died in the sanatorium, and 61 more died after their discharge, making a total of 110 deaths (55% of the group).The data on all the patients have been reviewed in order to determine whether any prognostic indexes could be found in this material.

The patient's race appears to have little influence on the course of the disease, the fatality rates being, 58% of the 106 white patients, 51% of the 71 Latin-Americans and Mexicans, and 52% of the 23 colored patients, who were chiefly Negroes. There were 48 deaths among the 93 males, about 52% of the group, as compared with 62 deaths among the 107 females, or 58%. There was little difference in the course of the disease in the various age groups, the average age being 28 years in both the survivors and those who died.

Associated phrenic paralysis appeared to have a beneficial influence in this series, the fatality rate being only 44% of the 15 patients who had such operations both before and after the induction of pneumoperitoneum and 49% of the 60 patients who had phrenic paralysis before pneumoperitoneum was instituted. This compares favorably with a fatality rate of 64% among those in whom the phrenic was paralyzed some time after pneumoperitoneum, and 84% of the 26 patients who did not have phrenic paralysis. Although there were numerous exceptions, it appeared, in general, that the patients in whom the paralyzed diaphragm rose more had, in this series, a somewhat lower, later fatality rate. The average rise of the diaphragm in the patients with phrenic paralysis was about 4 cm.

Cavities were found in the lungs in all but seven patients, and the prognosis was, in general, worse when there was larger, multiple, or bilateral cavitation, but the position of the cavities, whether they were in the upper or lower part of the lungs or whether they appeared to be central or peripheral on the roentgenogram, did not seem to affect the later fatality rates. The erythrocyte sedimentation rate was greatly accelerated in almost all patients, but, in general, the sedimentation rate was somewhat slower in those who survived and slowed more after institution of the pneumoperitoneum.

The severity of the disease may be indicated by the abundance of tuberculous complications clinically recorded prior to the institution of artificial pneumoperitoneum, including 39 cases of pleural effusion, with nine empyemas and four chest wall abscesses with one broncho pleural fistula, 36 cases of tuberculous enteritis with 10 tuberculous appendixes, 9 cases of tuberculous fistulas, 34 cases of tuberculous laryngitis, and 13 cases of tuberculous tracheobronchitis. Many more complications existed, but these were not recorded, as was indicated by the autopsies of the 20 who were examined.

In only 18 patients was the disease apparently arrested or arrested at the time of discharge from the sanatorium, and in 30 other patients it was said to be quiescent. Only 11 (23%) of these patients died subsequently. On the other hand, of the 72 patients discharged as "improved" and the 36 discharged as "worse," 55 (50%) have died.

Of the 200 patients in this series, pneumoperitoneum was induced in 35 for intestinal or peritoneal disease, of whom 23 (66%) died and 34 for tuberculous infiltration or pneumonias, of whom 19 (56%) died. A total of 74 patients were treated for cavities, of whom 36 (48%) died, and 56 were given pneumoperitoneum for symptomatic relief only, of whom 31 (55%) died. In comparing the early clinical results with the indications, it was concluded that the pneumoperitoneum had some desirable effect in only 65 (33%) of the patients, among whom only 20 (31%) died later, whereas, among the 135 in whom it had been entirely ineffective, 90 (68%) had died.

Pneumoperitoneum was terminated by death in 24 patients and because of discomfort or abdominal fluid in 28 patients; it was discontinued to allow more definite therapeutic measures in 20 patients. In 33 patients, it was abandoned as obviously ineffective, and in 87 it was still being given at the time of discharge from the sanatorium. In only one patient was pneumoperitoneum discontinued, while he was in the sanatorium, as an unqualified success, no longer needed; however, to this success should be added the 20 patients in whom, after pneumoperitoneum, thoracoplasty or other surgical measures that could not have been previously used were found feasible. A study of this group of 20 patients shows that 5 patients were unimproved or grew worse; 6 were improved; 1 case was quiescent; 1 patient worked for two years and 7 have been working for seven to nine years.

Ten years after most of the patients received their initial pneumoperitoneum, only 38 patients (40%) of the 90 living, reported that they were able to work. Many of these were enthusiastic over the benefit that they believed they had received from the treatment, and the physicians were often pleasantly surprised by the unexpected favorable results.

Of the 90 now living, 7 had arrested cases on discharge; 8 had apparently arrested cases; 22 had quiescent cases; 24 had improved cases; 24 had unimproved cases; and 6 deserted or left against medical advice. The reports made by our follow-up workers show that they were engaged in varied economic activities, ranging from the duties of a housewife, in some cases limited, to a full-time truck driver's job.

The incidence of survival, improvement, and recovery after induced pneumoperitoneum treatment in this series of patients with far-advanced cases is low. While its use in this series of patients would seem to have been justified, the results suggest that pneumoperitoneum should not be allowed to substitute or delay the institution of more direct and effective forms of treatment. It is still felt, however, that this procedure has therapeutic value, which may be more clearly brought out in later series, when it is applied to more favorable forms of the disease, and especially when it is supplemented by chemotherapy and other therapeutic measures.

5539 Murietta Ave. (Dr. Morris).

J.A.M.A., July 19, 1952

POSTOPERATIVE BACTERIAL SYNERGISTIC CELLULITIS OF ABDOMINAL WALL

FATALITY FOLLOWING HERNIORRHAPHY

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and
Theodore B. Krouse, M.D., Philadelphia

In this era of antibiotics and chemotherapeutic agents the incidence of postoperative wound infections has been reduce to a negligible percentage. This is due not only to these new agents but also to improvement in the efficiency of personnel, sterilization of instruments and fields, and improved surgical techniques that follow the basic concepts of good surgery and good tissue care. Against this background, there occur, rarely, certain infectious complications of normal surgical treatment. These complications, at first slow-moving, are insidious, relentless, and almost totally resistant to the usual modes of treatment. All concerned, the surgeon, the physician, the bacteriologist, and the pathologist, may well conclude that much is yet to be done in the field of bacteriology in assessing the specific organism or organisms causing such an infection and in determining the best specific therapy to combat them.

All types of chronic gangrene, although characterized by certain differences, are caused by special combinations of organisms. They may be conveniently classified as follows:[1] (*a*) postoperative progressive bacterial synergistic gangrene of the abdominal or chest wall, (*b*) gangrenous impetigo or ecthyma, (*c*) fusospirochetal infection of the skin, and (*d*) amebic infection of the skin. We are concerned with the first type as it affects the abdominal wall. This type of infection, due to an unusual synergism, has been described by Meleney,[2] Mester,[3] Brewer and Meleney,[4] Carroll,[5] Lichtenstein,[6] Dodd,[7] and Bassow.[8] Vier [9] has pointed out that it may be seen but once or twice in the experience of the average surgeon; he states, "... but when it does occur, it is so devastating to patient and distressing to surgeon that one is impressed with the importance of its early diagnosis and treatment." Cultures taken from such a gangrenous area contain microaerophilic nonhemolytic streptococci associated with hemolytic micrococcus pyogenes aureus.

The purpose of this paper is to report a case of a postoperative progressive inflammation of the anterior abdominal wall, in many ways similar to and as distressing as the gangrenous form. The infection was manifested grossly as an intense edematous type of cellulitis, with no evidence of gangrene. An anaerobic Streptococcus and a gram-positive hemolytic aerobic Bacillus identified as B. subtilis were isolated from the tissue. Injection of these organisms in combination into animals produced a similar tissue lesion.

REPORT OF A CASE

A 37-year-old white man was admitted to the Jewish Hospital Nov. 6, 1945, with the chief complaint of a hernia of 20 years' duration. The patient had first noticed a lump in the right groin. This lump had enlarged progressively over the course of years, and for the last 10 years the patient had been wearing a supportive truss. He had no other complaints referable to this local area. There were no gastrointestinal, genitourinary, or musculoskeletal symptoms. None of the other systems were contributory to his condition. The patient had been a regular blood donor and had made donations on 20 occasions prior to this admission. It was believed that these donations were properly spaced (not more frequently than every four to six weeks). Physical examination revealed a well-developed, well-nourished man in no distress and appearing to be in good health, except for the hernia, which he had elected to have removed. The temperature was 97.3 F, pulse rate 80, respiratory rate 20, and blood pressure 105/70 mm. Hg. The head and neck regions were normal, and the heart and lungs were normal. The abdomen was normal, except for the right lower quadrant, in which the right inguinal ring was enlarged. Preoperative laboratory data were as follows: urine normal; hemoglobin, 15.6 gm.; red blood cell count, 5,100,000; white blood cell count, 6,900, with 71% polymorphonuclear neutrophils (96% filamented and 4% nonfilamented), 28% lymphocytes, and 1% monocytes; blood sugar, 94 mg. per 100 cc.; and blood urea nitrogen, 14 mg. per 100 cc.

The preoperative diagnosis was right incomplete inguinal hernia. Two days after admission a Gallie herniorrhaphy was performed, with the patient under spinal anesthesia. Fascia lata strips from the left thigh, obtained with a Matson stripper, were employed in the repair. One gram of sulfadiazine was sprinkled into the wound, and closure was effected with clips. The patient's condition was good when he was returned to his room. In the first 24 hours after the operation the temperature climbed to 102 F and the pulse rate from 80 to 100. The blood pressure was 120/95 mm. Hg. On the ninth postoperative day there was slight tenderness and pain around the incision and the tissue seemed to be firmly indurated and edematous. The temperature at this time was 99.2 F. On the following day, after he had received a total of 160,000 units of penicillin in 21 hours, he was noted to be perspiring profusely and to have a rapid pulse rate. His temperature was normal. On Nov. 19 the wound area was anesthetized with procaine hydrochloride (novocaine®), and the wound was opened and a rubber-dam drain inserted. A culture grown from material taken from the site yielded colonies of aerobic hemolytic, gram-positive bacilli. The broth contained gram-positive streptococci in chains and gram-negative bacilli. The blood culture showed no growth after three days. On the 12th postoperative day the patient stated that he felt weak and

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9. Vier, H. J.: Progressive Postoperative Gangrene of the Abdominal Wall, with a Case Report, Surgery **7:** 334, 1940.

exhausted. He had perspired profusely during the night and had required 10 changes of clothing. He began passing small amounts of urine at frequent intervals. The lower quadrants of the anterior abdominal wall were considerably indurated, had the consistency of leather, and were edematous. This condition extended to the contralateral side. In addition, the penis was remarkably swollen, rendering micturition difficult. The pulse was difficult to palpate, and the blood pressure was 110/100 mm. Hg.

The condition at this time was believed to be an incisional abscess with cellulitis. The incision was reopened and the rubber-dam drain reinserted. There was no evidence of pus, despite the fact that several skin incisions had been made. The attending surgeon was impressed with the tissue consistency and stated that it cut as though it were leather. The patient was put into the position for shock, and administration of plasma and sulfadiazine was begun. Every three hours 30,000 units of penicillin was given. (It was not customary to give massive doses of penicillin at that time.) On the 13th day the patient was unimproved. The areas of induration and edema had advanced cephalad, the pulse was not palpable, and there was no recordable blood pressure. The patient was very apprehensive. He became dyspneic, and within a period of five to eight minutes he became successively more dyspneic, restless, talkative, delirious, and cyanotic. Venesection did not function, the most apparent reason being that intravascular clotting had begun. He was given a mixed gas gangrene antitoxin, 10,000 units intravenously, and an equal dose intramuscularly. He died on the 13th postoperative day.

Autopsy.—At autopsy the tissues in the incision were found to be partly necrotic, grey, and very firm; no pus or odor was noted. There was no evidence of gas formation in the tissues. The entire abdominal wall of the right lower quadrant and the medial third of the left lower quadrant were indurated and swollen. The overlying skin was a mottled purple color. The scrotum and both legs, particularly the right one, were edematous. Opening of the abdominal wall revealed the following condition: About an inch below the umbilicus, there was a sharply demarcated change in the subcutaneous fat underlying the abdominal wall. The tissues were brawny, indurated, and mottled hemorrhages were scattered throughout. The fat was greyish. There was no odor, pus, or gas in the deeper tissues. The rectus muscle on the right was completely involved by this process; it was swollen and greyish, and there were petechiae scattered throughout. The left rectus muscle of the lower quadrant showed early involvement. The preperitoneal fat was also indurated and swollen. The omentum was fatty, and the portion lying in the right lower quadrant seemed firm and edematous; it was exceptionally solid when cut. The terminal ileum was anchored to the pelvis by an abnormal band of peritoneum.

The vessels were essentially normal. The heart weighed 360 gm. and was somewhat flabby. The lungs were firm and congested, without edema, and both weighed 510 gm. The spleen weighed 200 gm. and was flabby, mushy, and dull red on section, with loss of architecture. The lymph nodes were normal. The kidneys weighed 180 gm. The liver weighed 2,660 gm. and was flabby and pale, the cut surface being tan to orange. The pancreas was congested, and the adrenal glands showed decreased cortical lipid. In the skin and subcutaneous tissue there were multiple capillary hemorrhages, with a fibrinous exudate but with no cellular reaction (inflammatory cells). A section from the incision area showed fibrinous exudate with a greater cellular reaction. There was necrosis of the vein and artery in the area of the incision and degeneration of adipose tissue. A section from the rectus muscle showed edema, congestion, necrosis, hemorrhage, and an extensive fibrinous exudate with little or no inflammatory cell reaction. Microscopic examination revealed that an overwhelming toxemia had followed infection of the herniorrhaphy wound, with all vital organs showing severe toxic and degenerative changes.

Bacteriological Observations.—Bacteriological examination at autopsy corroborated the previous observations. An anaerobic nonhemolytic Streptococcus and a gram-positive hemolytic aerobic Bacillus identified as belonging to the species B. subtilis were isolated from material taken from the edematous skin wound. When injected intradermally into guinea pigs and a rabbit, they produced edema and induration of the subcutaneous tissue. Similar effects were observed when a mixture of the cultures was injected into the muscles of guinea pigs.[10]

COMMENT

Postoperative progressive bacterial synergistic gangrene is due, as are the other chronic gangrenes, to the symbiotic action of two or more organisms. This symbiosis seems to develop in the area involved in the operation and probably in tissue that has been altered to favor such complimentary growth. In the case we have described, the predominant feature, in contrast to other lesions that have been described in this anatomic area, was an intense board-like edematous cellulitis rather than a gangrene. In nearly half a century of surgical practice we have never encountered an infection of this type. Although the presence of an anerobic nonhemolytic Streptococcus seemed typical for such an infection, that of its symbiotic mate, a variety of B. subtilis, did not. The family of B. subtilis is recognized to be of low pathogenicity. It is used as a test organism for sterilization procedures.[1] The pathogenicity of the anaerobic Streptococcus depends upon the proper conditions. These two organisms, as the major symbiotic pair, in the presence of suitable damaged tissue, appear to have been the causative agents. It may be that the other organisms played a supporting role or were secondary invaders. Weiss [11] has demonstrated that in a variety of infections harboring the anaerobic nonhemolytic Streptococcus a multiplicity of organisms was present.

Interesting facet of this case was the fact that the patient had been a frequent blood donor prior to the operation. There was no reason to suspect that these multiple bleedings had a deleterious effect on the condition of the peripheral blood; on the contrary, the hemoglobin and red blood count on admission to the hospital were good. Snapper, Liu, Chung, and Yu,[12] in 1939, studied 101 blood donors who had donated from 1 to 25 liters of blood in periods ranging from 2 months to 10 years; the frequency and extent of anemia increased with an increase in the total amount of blood withdrawn or in the duration of the service. In our patient there was no reason to suspect damage to the hemopoietic system as reflected in the peripheral blood. Doan and Cunningham [13] used rabbits experimentally as donors on many occasions. The bone marrow of these animals showed extreme hyperplasia of bone marrow elements, but they also showed small noncellular areas from which the myeloblasts had disappeared. The authors felt that the evidence indicated a secondary anemia of long standing. There were also small acellular areas containing either a hyaline-like structureless, ground substance or fine globules of fat, and abnormal myeloblasts were evident. In our patient the peripheral blood response was adequate, and there was no reason to suspect a faulty bone marrow. It is possible that there was some fault in tissue resistance engendered by the multiple bleeding, which may have, in association with the local trauma resulting from surgical procedure, prepared the bed for the resistant infection that followed.

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[10] The bacteriological investigations for this case were done under the supervision of Dr. Charles Weiss.

[11] Weiss, C.: The Pathogenicity of Bacteroides Melaninogenicus and Its Importance in Surgical Infections, Surgery **13**: 683, 1943.

[12] Snapper, I.; Liu, S. H.; Chung, H. L., and Yu, T. F.: Anemia from Blood Donation: A Hematological and Clinical Study of 101 Professional Donors, Chinese M. J. **56**: 403, 1939.

[13] Doan, C. A.; Cunningham, R. S., and Sabin, F. R.: Contributions to Embryology, Washington, D. C., Carnegie Institute, 1925, vol. 16, p. 163.

The anaerobic organisms are generally resistant to the action of chemotherapeutic agents, including both the sulfonamides and penicillin, and this is also true when they are in synergism with other organisms. As with the gangrenous forms of infection,[14] phlegmonous or cellulitis infection should be treated intensively, early, and specifically according to the etiological agents.

SUMMARY

1. A fatal case of postoperative progressive abdominal wall cellulitis is reported.

2. The primary etiological factor appeared to be a synergism between an anaerobic nonhemolytic Streptococcus and a gram-positive hemolytic aerobe of the species Bacillus subtilis.

3. Postoperative progressive synergistic bacterial gangrene and cellulitis are similar in clinical course, etiology, predisposition, resistance to treatment, latent period, general anatomic location, and rarity. They differ greatly, however, in appearance.

4. Bacteriological study of postoperative wound infections, even in this era of advancing antibiotic therapy, is still of importance. The organisms causing such an infection should be determined definitively, and the infection should be eradicated early.

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[14] Meleney, F. L.: A Differential Diagnosis Between Certain Types of Infectious Gangrene of the Skin, with Particular Reference to Haemolytic Streptococcus Gangrene and Bacterial Synergistic Gangrene, Surg., Gynec. & Obst. **56**: 847, 1933.

ACCIDENTAL POISONING BY TOXAPHENE

REVIEW OF TOXICOLOGY AND CASE REPORTS

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A recently developed insecticide, which has demonstrated its effectiveness against a variety of agricultural and livestock insect pests, is chlorinated camphene, commonly known as toxaphene. The empirical formula is $C_{10}H_{10}Cl_8$. The technical material is an amber waxy solid with a mild terpene odor. It contains from 67 to 69% chlorine and melts at temperatures of 70 C to 95 C. It is soluble in the common fat solvents and insoluble in water.

Single dose administration of toxaphene to laboratory animals shows a variable toxicity, depending on the physical form of the poison, the concentration, and the vehicle and means of administration. In white rats the oral L. D. 50 for a 1% emulsion in peanut oil and acacia is 40 mg. of toxaphene per kilogram of body weight of the animal.[1] The L. D. 50 in the rat for a 20% kerosene solution is 100 to 150 mg. per kilogram, for a 5% solution in kerosene, 300 to 350 mg., and for a 5% solution in corn oil, 120 to 125 mg. The intravenous L. D. 50 in the rat with a 1% toxaphene emulsion in peanut oil and acacia is approximately 13 mg. per kilogram.[1] In guinea pigs the L. D. 50 by oral administration of 5% toxaphene in kerosene is 350 to 375 mg. per kilogram, and of 5% toxaphene in corn oil, 250 to 280 mg.[1] In the dog, Lackey[2] found that generalized convulsions resulted from doses as low as 10 mg. per kilogram, when a digestible oil was used as a vehicle. The average lethal dose from oral administration in dogs lay somewhere between 15 mg. and 40 mg. per kilogram.

Toxaphene, in a suitable vehicle, can be absorbed through the skin of rabbits. From peanut oil solution on the skin, a dose of 250 mg. per kilogram may kill the rabbit in 24 hours.[1] From kerosene solution, 100 mg. per kilogram applied to the skin daily for six days gives a high mortality rate in the rabbit and guinea pig. By contrast there is poor absorption by the skin of toxaphene mixed in an inert powder. Rabbits require 500 to 1,000 mg. per kilogram of toxaphene, applied in a 40% dust, to give symptoms of poisoning or death in 24 hours. This species is unaffected by daily application to the skin for six weeks of a 5% toxaphene dust at the rate of 125 mg. toxaphene per kilogram.[1]

It has been found that feeding toxaphene at rates of 100 ppm and 800 ppm in the diet of white rats and guinea pigs for a period of six months shows no significant effect on growth, blood cell count, urinalysis, reproduction, mortality, or histological findings.[1] A study of chronic toxicity in the dog has been reported by Lackey.[2]

From these and other unpublished observations on the effects of toxaphene on 10 species of animals, it has been found consistently that the manifestation of poisoning by this insecticide is diffuse stimulation of the cerebrospinal axis resulting in generalized convulsions. Sublethal doses cause shorter and fewer convulsions than do higher doses. If the dose is lethal, death is due to respiratory failure after a series of convulsions of increasing severity.

Barbiturates protect dogs against supralethal doses of toxaphene. After recovery from the barbiturate anesthesia the animals show no abnormal neurological signs suggestive of organic pathology in the central nervous system. Apparently, the effects of the insecticide on the nervous system are drug-like and reversible. The strikingly selective action of toxaphene on the central nervous system can best be explained by its high lipoid solubility. Lackey concluded that the barbiturates were the preferred depressants in protecting dogs against poisoning.

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[1] Shelanski, H. A., and Gellhorn, A.: Unpublished data.

[2] Lackey, R. W.: Observations on the Acute and Chronic Toxicity of Toxaphene in the Dog, J. Indust. Hyg. & Toxicol. **31**: 117 (March) 1949; Observations on the Percutaneous Absorption of Toxaphene in the Rabbit and the Dog, ibid. **31**: 155 (May) 1949.

"If convulsions begin before treatment is instituted, sodium pentobarbital seems to be the drug of choice."[3]

During the five years in which chemists have done research on this material and in which scores of workers have been potentially exposed during manufacture of toxaphene there has not been an instance of poisoning in man. Periodic examination of these workers has shown no alteration in reflexes, subjective manifestation of disorders of the nervous system, or other signs of health impairment. Urinalyses and blood cell counts have been normal; furthermore, we have learned of no instances of accidental poisoning among workers exposed in the manufacture of insecticides containing toxaphene. Such exposure to a worker in a well-controlled manufacturing operation would undoubtedly be minimal. In some instances, however, it is known that recommended precautions in manufacturing processes have been ignored. Since several hundred men have been involved in such work it is apparent that the hazard from inhalation and skin absorption by man is actually not great. Such agents as solvents, inert dusts, and chlorine have been more troublesome than toxaphene from the point of view of the worker during manufacturing and compounding.

As with other economic poisons used in agriculture, one may expect that increasing use of toxaphene will be accompanied by instances of human poisoning through accidents and careless misuse of the agent. Physicians will be called on to treat such patients, and it is for this reason that a discussion of the essential pharmacological properties of toxaphene has been presented. Such pharmacology is based on observations made largely in laboratory animals that were poisoned by known doses of technical toxaphene. When the material appears in insecticides, it is diluted or dispersed with dusts, solvents, and water, and may be found mixed with other insecticidal materials. The latter may modify the manifestations of poisoning. The label on finished insecticides should show the presence and quantity of active ingredients.

The following case reports describe instances of death or illness that, in our opinion, were probably due to toxaphene. The poison was taken by mouth in each case. The symptoms were those to be expected from toxic amounts of toxaphene.

REPORT OF CASES

Case 1.—A Negro boy, aged 2 years and 8 months, played in a yard where there were strips of metal from flattened drums from which technical chlorinated camphene (toxaphene) had been removed previously in a processing plant. The metal sheets had been brought from the plant and part of them used that day to cover the walls of a garden tool storage shed. An amber-colored, beeswax-like material, identified as toxaphene, was subsequently found on and readily scraped from the metal sheets. No one saw the child ingest this material, but it was available to him, and he was known to have played in the vicinity of the sheets. The lack of taste and softening of technical toxaphene at body temperature enables one to chew it as readily as paraffin wax. The child left the play yard before 5 p. m. on this particular afternoon, had a bath and ate his supper, and played in his home until bedtime, at 8 p. m. There was no sign of illness until he awoke just after midnight, screaming and in a convulsion. The child was taken to the emergency ward of a hospital at 1 a. m. Here he received an unidentified hypodermic or intramuscular injection but continued to show intermittent convulsions. An hour later an attempt was made to transfer the patient by car to another hospital. He died en route at about 2:30 a. m. during a convulsion. At autopsy it was found that a "pale froth" was exuding from the mouth, the heart was greatly dilated, and the lungs were markedly congested and edematous. The bronchial tree was filled with a faintly blood-tinged froth; there was no foreign material in the respiratory tract. The stomach "was dilated with air." The liver, gallbladder, and spleen were grossly normal. There was much hard fecal material in the rectum. The kidneys were "rather swollen and the hilus of the right kidney showed a purulent-like material, possibly a pyelitis." A few petechial hemorrhages were found in the white substance of the brain. "No old scars of birth injuries were noted." In stating the immediate cause of death the pathologist reported "symptoms and pathological findings are consistent with a death from a central nervous system poison of a convulsant-type. Actual death was due to suffocation during a convulsion."

Case 2.—A 4-year-old white boy is believed to have ingested an unknown quantity of toxaphene emulsion in water from a gallon jug. The child's stomach was probably empty at this time. The first convulsion occurred about two hours later and continued at two to five minute intervals until death. Death occurred about six hours after the time the poison is believed to have been ingested. Intramuscularly administered magnesium sulfate, phenobarbital sodium, and curare and cyclopropane anesthesia were used in treatment at a hospital shortly before death of the patient. Autopsy was not performed.

Case 3.—A 17-month-old white boy played where his father was mixing toxaphene for a tobacco spray. The youngster picked up a tin cup being used in this process and drank at 6 p. m. what his father called "about a half teaspoon of full-strength toxaphene" (an investigator designated this a solution of toxaphene, presumably a 60% concentration in special solvents). After an unspecified interval, convulsive seizures developed in the child, which recurred with increasing frequency until death at 5 a. m. the following morning. There was no autopsy, and no details are available regarding treatment other than that medical care was given within an hour of the ingestion of poison.

Case 4.—A 2-year-old Negro boy is believed to have ingested an unknown amount of toxaphene. The history stated "while his mother was working in the cotton field, the child ingested a large amount of a chemical containing 20% toxaphene." After an undetermined interval, convulsive episodes developed in the child. He was hospitalized within one-half hour of the onset of the convulsions, and the record of physical examination described the child as "a well developed, well nourished colored male infant who exhibits mild cerebral excitement with hypertonicity of all extremities; aimless jerking motions of all extremities, proceeding to generalized convulsions associated with marked pharyngeal and laryngeal spasm and respiratory embarrassment and cyanosis. E.E.N.T.: Pupils are fixed and dilated. Eyes have a wild, staring expression. Otherwise, negative except for enlarged tonsils. Neck stiff, back bowed: Heart and lungs clear to percussion and auscultation. Abdomen: negative except for spasm of the anterior abdominal wall." The child received "large doses of sodium luminal and curare intramuscularly." Artificial respiration with an air way in place was given during the convulsive episodes. He remained in a critical condition for several hours. Twelve hours after admission to the hospital, he was in relatively good condition and recovered with no untoward symptoms. He was discharged two days later.

Two instances have been found of group poisoning believed to be due to toxaphene, following its misuse as an agricultural pesticide. All these patients recovered.

Case 5.—A Negro farmhand sprayed a collard patch twice on Oct. 4, 1951, with an insecticide that contained approximately 9% toxaphene as an aqueous emulsion. On Oct. 7, a Negro woman, aged 49, picked collards from this patch, washed them three times in different water, and cooked the greens for one hour with a piece of salt pork. Her family of seven ate the greens at 5 p. m. (no other meal had been served since breakfast). At about 6:30 p. m. she "got sick at the stomach," vomited, and took 2 oz (60 cc.) of castor oil in vinegar. She thought little of the episode because she "has a lot of gas most of the time." She had no further symptoms.

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[3] Lackey, R. W., and Weed, O.: Treatment of Acute Toxaphene Poisoning in the Dog, South. M. J. 44:165 (Feb.) 1951.

At 7:30 p. m. a 20-year-old Negro woman fell to the floor in a convulsive seizure lasting 10 minutes. After the convulsion, she became lucid but had no recollection of having fallen or of the convulsion. At 8:15 p. m. she had a second convulsion coincident with her arrival at a hospital. A stomach tube was passed, and the stomach was lavaged with 2 liters of warm sodium bicarbonate solution. Vomiting of "a large amount of collard greens" followed this treatment. At 10 p. m. 2 grains (0.13 gm.) of phenobarbital sodium was given subcutaneously. No further symptoms were observed. At 8 p. m. a Negro girl, aged 12, fell suddenly in a convulsion. This was followed by a lucid period of about 20 minutes' duration and then another convulsion at 8:30 p. m. At the hospital she was given apomorphine hydrochloride, 0.05 grain (3 mg.), by hypodermic and gastric lavage. "An enormous amount of collard greens were evacuated within a short interval." She received 2 grains (0.13 gm.) of phenobarbital sodium subcutaneously that night, ate a good breakfast the following morning, and objected to being kept home from school, since she felt entirely well. A 16-year-old Negro girl had brought the two patients described above to the hospital. She had no evidence of illness until she fell unexpectedly in a convulsion in the hospital corridor. The fit lasted three to four minutes, after which she was quite rational and cooperated in drinking a quart of warm water containing sodium bicarbonate. She was given apomorphine hydrochloride, 0.10 grain (6 mg.), after which she vomited a "copious amount of collard greens." The onset of her convulsion was approximately 3 hours and 45 minutes after eating the greens. She received phenobarbital sodium and had no further convulsions. The following morning there were no complaints or physical findings other than "weakness and lassitude." In the woman who had picked and cooked the greens and in three other members of the family, 30, 10, and 7 years in age, symptoms of poisoning did not develop during medical observation. Four hours after ingesting the greens each of them was given 2 oz. of castor oil. Subsequent examination of members of this family has revealed no evidence of disease. On Oct. 8, an examination of the garden showed that it was badly grown up in weeds and that both weeds and collard greens were severely burned by the spray material. Samples of the collard leaves were forwarded to the experiment station of the Hercules Powder Company for analysis for organic chloride content. The results of the analysis are shown in table 1. An extremely heavy insecticide residue was present on all of the material. Since toxaphene, like other chlorinated toxicants, is not soluble in water, washing in water does not remove significant amounts of the material from vegetation. On the basis of the analyses, one may calculate the amount of toxaphene ingested by assuming quantities of the greens that may have been eaten (table 2).

Case 6.—In the second instance of group poisoning, a white man sprayed his garden chard with an unspecified amount of toxaphene one morning and harvested the same chard leaves for the family meal that evening. The chard was washed before it was cooked. The meal was eaten at 6 p. m. by the man, his wife, and their son. At 10 p. m. the son was awakened from sleep by a "jerking and twisting" in his muscles. He left his bed feeling ill and provoked vomiting by placing his fingers in his pharynx. He was given strong coffee and tea and forced to vomit repeatedly. At 11 p. m. the man found that his "mind went blank with no warning of nausea or any symptom." Within two or three minutes he fell in a "stiffened heap on the floor." The wife and son lifted him to a couch, pried his "locked jaws open," and forced him to vomit. He is reported to have been unconscious for an hour. No mention is made of clonic movements. At an unspecified time his wife noted lightheadedness and a feeling of mild nausea but could vomit only by deliberately provoking it after taking fluids. The patients were not seen by a physician that night. The man reported the episode by letter, which included these statements: "We are fifty miles from a doctor; no help there. The family was extremely weak for several days. Two days after the poisoning I went to a doctor, and he said if I were all right as far as emptying out the digestive tract, there would not be after effects. However, I did have amnesia for about a week. There were many things I did about the ranch that I could not remember having done two weeks later, such as moving machinery and tasks accomplished." He stressed the absence of warning symptoms, nausea, and cramps, and stated that vomiting had to be induced in himself and his family.

Table 1.—*Residual Toxaphene on Collard Greens*

Sample	Description	Organic Chlorine Calculated as Toxaphene, Ppm
1	As picked	3,312
1-A	As picked	3,318
2	Washed 3 times	3,126

Table 2.—*Dose of Toxaphene in Relation to Amount of Collard Greens Ingested*

Weight of Greens		Residual Toxaphene, Mg.	Dose of Toxaphene in Mg./Kg. of Body Weight*
Gm.	Lb.		
200	0.44	663	9.5
500	1.1	1,658	24.0
1,000	2.2	3,315	47.0

* Assuming a body weight of 70 kg. (154 lb.).

These case reports appear to confirm the laboratory findings that toxaphene acts chiefly as a convulsant drug. No clinical picture of chronic poisoning in man has been developed. This is consistent with Lackey's finding[2] that dogs receiving a convulsant dose of toxaphene daily appear alert and behave normally except for the convulsion. Such doses given over long periods can produce in dogs degenerative cellular changes in the liver and kidney. There were no manifestations of poisoning in these cases until the acute overstimulation of the nervous system occurred following absorption of the poison. In human poisoning, in contrast to that sometimes noted in the dog, vomiting apparently must be induced (one possible exception is mentioned above). There is no abdominal cramping or diarrhea to serve as evidence of poisoning.

SUMMARY AND CONCLUSIONS

1. Three deaths in children presumably resulted from accidental ingestion of lethal amounts of an insecticide, toxaphene. A fourth child so poisoned recovered.
2. Two instances of group poisoning, that followed ingestion of toxaphene-sprayed collards and chard, respectively, have come to our attention. Among 10 persons known to have eaten the contaminated greens, in 7 symptoms developed consistent with toxaphene poisoning as seen in laboratory animals.
3. The onset of poisoning characteristically was abrupt and not associated with abdominal pain, involuntary vomiting, or diarrhea. Illness is manifested by a skeletal muscle response to central nervous system stimulation and by loss of consciousness.
4. These instances of poisoning in humans have resulted from accidents or injudicious use of the pesticide.
5. Since similar poisoning may occur in the future, it is essential that physicians recognize the cardinal symptom, a convulsion, and the importance of countering the acute central nervous system stimulation with barbiturates.
6. The complete absence of poisoning in workers who manufacture toxaphene and in those who are exposed to it, both in the compounding of pesticides and its use in agriculture, suggests that it can be handled with safety.
7. This and other effective pesticides are known poisons and must have lethal qualities if there is to be progress in man's war against insects. Such chemicals should be used properly and with precautions against human consumption if tragedies are to be avoided.

Delaware Trust Building (Dr. McGee).

LOW HEMOGLOBIN LEVELS IN WOMEN AS REVEALED BY BLOOD DONOR RECORDS

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In American Red Cross blood procurement records women constitute a minority of the donors represented. The greater response by men can be attributed largely to more intensive recruitment among male employee groups, to less fear of venipuncture, and to more widespread knowledge of the therapeutic uses of blood and its derivatives, gained while serving in the armed forces. Furnishing the stimulus for the present report, still another element of the disparity is the fact that on presenting themselves many more women than men are found to be unacceptable because of low hemoglobin levels.

Previously unpublished information concerning the incidence of low hemoglobin levels can be derived from mass data pertaining to white persons who volunteered to contribute blood in the period January, 1948, to June, 1949, inclusive.[1] All ranged in age from 18 through 59. Shown in table 1 are figures on 239,191 persons (165,408 men and 73,783 women) [2] who volunteered to make their first donations at the regional blood collecting and processing centers or mobile unit operations.

Compared to less than 1% of the men, 12.6% of the women were unable to meet a requirement of 12.3 gm. of hemoglobin per 100 cc. of blood, which was the minimum for acceptance of a person as a blood donor.[3] A copper sulfate technique modified from the method of Phillips and co-workers [4] was employed in all determinations.

During the past two years occasional checks have disclosed a practically unchanged aggregate number of low hemoglobin level disqualifications per 100 women. Assuming that the women who present themselves at the Red Cross centers are above the general average in health, a less satisfactory situation must exist in the nation as a whole. By extending census figures we place the total number of women who range between 18 and 59 years old, inclusive, at approximately 47 million. Risking an insecure estimation, the number of women deficient in hemoglobin and therefore potentially disqualifiable may run from nearly 6 million (calculated from the Red Cross data) to well over 10 mllion. Anemia is bound to engender rising medical interest if the hospital demand for blood should continue to increase, since procurement would be broadened to encompass larger segments of the population, and women would be prevailed on to donate in greater numbers.

TABLE 1.—*Donors and Disqualified Persons in Selected Groups*

	Men	Women
Total no. of persons	165,408	73,783
Number disqualified		
All reasons	13,798	15,233
Low hemoglobin level	1,382	9,322
Percentage disqualified		
All reasons	8.3	20.7
Low hemoglobin level	0.8	12.6

Analytical discussion is now feasible with special reference to a selected group of 6,911 women, included in table 2. All successfully donated blood on first visits and came back within one year to do so again. Thereupon 12.3% were disqualified owing to a low hemoglobin level. On the average the group members were 37.3 years of age at the time of second visits and had waited 5.6 months before returning. From tables 2 and 3 we find that those rejected (1) were relatively more numerous among persons returning from 2 to 10 months than earlier or later, (2) averaged 5 months younger than the women accepted, (3) were relatively more numerous among persons between 21 and 50 years than younger or older, and (4) averaged six days sooner in returning than those accepted.

TABLE 2.—*Distribution of Selected Women According to Interval Between First and Second Visits and Average Ages*

No. of Months Between First and Second Visits	Number			Percentage Disqualified	Average Age (Years)	
	Total	Disqualified	Accepted		Disqualified	Accepted
Under 2	224	13	211	5.8	33.3	33.3
2- 3.9	2,084	278	1,806	13.3	37.1	37.7
4- 5.9	2,041	274	1,767	13.4	37.0	37.8
6- 7.9	1,297	164	1,133	12.6	37.5	37.5
8- 9.9	741	82	659	11.1	35.3	36.9
10-11.9	524	41	483	7.8	36.3	36.0
Total	6,911	852	6,059	12.3	36.9	37.3

Whether the group was considered compositely or divided into subgroups according to the period between the first and second visits to permit views of the net relationships, a characteristic pattern unfolded. Starting with approximately 9.1% of the women 18 years old, the gross low hemoglobin level disqualification curve proceeded upward to a plateau, was above 13.5% from 34.5 to 44.5 years, and then declined to 6.3% by the end of the 59th year. Though differing in magnitude from one subgroup to another, the rejection rates adhered to the general trend no matter how long the interval to the second visit. Incidentally, the variations strengthen the frequently expressed view that women in their fifties, usually poorly represented in blood program records, should be desirable for donor recruitment.

From National Headquarters of the American National Red Cross.

[1] In the period designated, the blood program was composed of nine constituent programs, located in the following regions: Rochester, N. Y.; Wichita, Kan.; Atlanta, Ga.; Washington, D. C.; Los Angeles; St. Louis; Detroit; Columbus, Ohio; and Massachusetts exclusive of Boston.

[2] Based on convenient blocks of machine record cards. The totals in the top line of table 1 do not portray those of a typical division according to sex. Experience shows the following averages: men, 73.6%; women, 26.4%. These proportions applied to the figures on the bottom line of the table yield 3.9%, which represents typical low hemoglobin occurrence among white blood donors in both sexes.

[3] Present minimum, 12.5 gm.

[4] Phillips, R. A., and others: Copper Sulfate Method of Measuring the Specific Gravities of Whole Blood and Plasma, Bull. U. S. Army Med. Dept. (no. 71) pp. 66-83 (Dec.) 1943.

Obviously little meaning could be attached to the observed tendencies if the physicians on duty had subjected persons in the terminal age classes more than others to unusually careful questioning before accepting or rejecting them. Inquiry uncovered no intimation that such selectivity could have affected the reported figures in more than a very minor degree. Consequently, under the conditions specified for the subgroups (number of months between visits constant), the statistical findings seem plausible. Our demonstration that the host of factors governing the disqualifications included factors peculiarly associated with the ages of the women is consistent with published hemoglobin weights. The preponderant evidence in reports by Pett and Ogilvie,[5] the Medical Council of Great Britain,[6] Bryce and Jakobowicz,[7] and Howell[8] would indicate that the values are normally greater in women belonging to the age groups under 20 years and above 50 years than to other groups.

Another age phenomenon came to light in the form of a preliminary period upon the termination of which the determinations of low hemoglobin levels began to be comparatively less numerous. In regard to the women 21 through 59 years, aggregating 80.5% of the entire group, this period averaged 5.8 to 6 months in duration from the dates of the initial blood donations; however, the women 50 years and older were not disqualified in lessening frequency until the stage of nondecline had lasted 7.4 months, whereas for those under 21 years it ended at only 5.2 months. The differences seem to reflect corresponding differences between the older and younger women with respect to the power of their bone marrow to regenerate erythrocytes. (Nothing said here is incompatible with a requirement that at least eight weeks must elapse between donations, and no person shall be accepted more than five times a year.[9])

TABLE 3.—*Distribution of Selected Women According to Age on Second Visit and Average Interval Between First and Second Visits*

Age (Years)	Number			Percentage Disqualified	Average No. of Months Between Visits	
	Total	Disqualified	Accepted		Disqualified	Accepted
Under 21...	265	25	240	9.4	5.2	5.7
21-29.......	1,857	226	1,631	12.2	5.3	5.6
30-39.......	1,932	261	1,671	13.5	5.6	5.7
40-49.......	1,774	242	1,532	13.6	5.3	5.6
50-59.......	1,080	98	982	9.1	5.1	5.4
All ages....	6,911	852	6,059	12.3	5.4	5.6

After the expiration of the preliminary period the disqualification rates diminished in accordance with a lengthening interval to the second visit. The downward trend was noticeable when women of the same age were considered. Hence, the inference is forthcoming that the total causes of low hemoglobin levels embraced factors independent of age but definitely related to the passage of time to second visit. Toward the end of one year from the first visit the action of time influences weakened and relatively more women were then able to meet the prescribed hemoglobin test.

Left for brief mention is a supplementary group of 458 women who presented themselves 12 to 15 months subsequent to first donations. The low hemoglobin level disqualifications suggest that some women, up to 15.9%, psychologically hostile or feeling unequal to second contributions, eventually accede as a matter of civic duty. We know that so far as practicable many chapter recruitment committees seek out previous donors at the end of a year's nonreturn to the regional centers. The limited additional data suffice merely to raise the possibility that self-screening by the less physically able persons complicates the task of persuading previous women donors to maintain their support of the blood program.

SUMMARY

There are now in the United States an estimated 47 million women in the customary age range for blood donors, 18 through 59 years. Of that total, the number subject to being disqualified because of low hemoglobin levels may greatly exceed 6 million. Anemia is bound to be a subject of increasing medical interest if expansion of blood program operations should be continued, broader segments of the population included, and donor recruitment directed more intensely to women than at present. These comments result from a review of mass data collected by the American Red Cross.

Sample data on 239,191 white persons (165,408 men, 73,783 women) show that on their initial visits to donate blood at Red Cross regional centers or mobile unit operations 12.6% of the women (as compared to less than 1% of the men) were unable to meet a minimum requirement of 12.3 gm. of hemoglobin per 100 cc. of blood. A study of the records pertaining to a selected group of 6,911 women who had been successful donors on first visits revealed 12.3% rejected for the same reason on second visits made within a year thereafter. Statistical considerations indicate that the second visit disqualifications were related to the ages of the women, length of interval between the first and second visits, and other, undetermined factors.

The disqualifications were relatively more numerous during the most prevalent ages for child bearing and care and less numerous among younger women and those in their late forties and fifties. They were also fewer toward the end of one year from first visits than sooner; however, only upon the termination of a preliminary period averaging 5.9 months in duration were diminishing low hemoglobin levels in evidence. The exact point in time marking the beginning of the decline differed according to the women's ages, apparently reflecting a corresponding variation in the power of bone marrow to regenerate red cells of the blood.

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[5] Pett, L. B., and Ogilvie, G. F.: Hemoglobin Levels at Different Ages, Canad. M. A. J. 58: 353-355 (April) 1948.

[6] Haemoglobin Levels in Great Britain in 1943, Medical Research Council, Special Report Series, no. 252, London, His Majesty's Stationery Office, 1945.

[7] Bryce, L. M., and Jakobowicz, R.: The Haemoglobin Level in Blood Donors, M. J. Australia 2: 329-334 (Oct.) 1943.

[8] Howell, T. H.: The Hemoglobin Level in Old Age, Geriatrics 3: 346-352 (Nov.-Dec.) 1948.

[9] The American National Red Cross Blood Program: Medical-Technical Manual, no. 1702, Washington, D. C., American National Red Cross, rev. Feb., 1952, p. 1.

CLINICAL NOTES

MULTIPLE ANEURYSMS OF THE RIGHT CORONARY ARTERY

DEATH FROM A RUPTURED ANEURYSM OF THE ABDOMINAL AORTA

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REPORT OF A CASE

B. F., a Jesuit priest 67 years of age, was brought by ambulance to the Loretto Hospital on Oct. 26, 1950. He complained of severe abdominal pain, which was most intense in the left lower quadrant. The pain was not relieved by an enema or by 1/6 grain (0.01 gm.) of morphine sulfate given hypodermically. A second similar injection of morphine given three hours later induced a brief fitful sleep. The patient awakened with pain in the abdomen and back and moaned and tossed in agony. He had been in the hospital only six hours in his terminal stage and was thought to have a mesenteric thrombosis.

The blood pressure was 190/110 on several readings and was in marked contrast to his usual pressure of 124/80. The lower pressure had been recorded on numerous occasions in the preceding six years and even while the patient had been hospitalized for coronary insufficiency and myocardial infarction.

The pain in the abdomen was difficult to evaluate, because of a history of so-called colitis. Various episodes of this condition had been self-medicated with cathartics and alkaline powders for many years. An especially severe abdominal pain five days before death had been so treated. This was at a time, in the last two weeks of October, 1950, when the patient had a variety of other symptoms. First hemiparesis occurred and was associated with dysarthria. The conditions developed over several days and resulted only in a minor disability in movement. They had improved to the point where the patient had resumed his duties just prior to his death.

A review of his personal history disclosed "rheumatic trouble" at 6 years of age, but details were lacking. There was hospitalization in 1939 for repair of a ruptured left quadriceps femoris muscle. This was incurred 14 months previously in a baseball game. At this time the heart had a normal rate and rhythm and all laboratory tests showed normal blood and urine.

Despite the good condition of the heart in 1939, the patient had a severe anginal attack in 1940. His condition improved after a 10 day stay in the hospital. The electrocardiogram showed some inversion of the T wave in lead 1. No repetition of the attack occurred for six years, and in the interval the patient resumed his duties as a religious instructor. His pulse was good, and his blood pressure was from 115 to 125 systolic and 70 to 80 diastolic.

In February, 1946, the patient had two attacks of angina pectoris. These were so severe that he recorded their occurrence distinctly at 10 a. m. and 1 p. m. The pain was in the chest at the level of the heart and radiated to each shoulder. Glyceryl trinitrate therapy relieved the pain. The electrocardiogram showed low-voltage QRS complexes, suggesting diffuse myocardial disease. The P-R interval at 0.20 suggested impending first degree auriculoventricular block. The diagnosis on discharge was arteriosclerotic heart disease and angina pectoris without coronary artery occlusion.

No similar episode occurred in the interval between February, 1946, and October, 1950, when the aforementioned abdominal symptoms occurred, and the patient was brought to the hospital for emergency treatment on the service of Dr. Francis Foran.

At autopsy, performed immediately after death, the following observations were made: The abdominal cavity contained 50 cc. of fresh blood from minute tears in the peritoneum near the left kidney. There was hemorrhage beneath the mesosigmoid close to its attachment to the posterior wall, and the blood extended laterally to the outermost edge of the left rectus muscle. The hemorrhage also extended retroperitoneally to the region of the left kidney and downward beneath the covering of the urinary bladder. In the region of the lower pole of the left kidney the hemorrhage extended to the right of the midline, across the duodenum, and into the mesentery for the small bowel for 8 cm.

The left adrenal gland was extensively dissected by hemorrhage, and some parts of the cortex resembled canary yellow islands of tissue in a sea of blood. The first impression was of an adrenal tumor in which a blasting type of hemorrhage had occurred. The origin of this huge extravasation was, however, in a saccular aneurysm of the aorta just proximal to the bifurcation. The aorta was dilated to a circumference of 12 cm. at this point, whereas above the dilation the greatest circumference was 7.2 cm. There was dissection under the lining of the aneurysm in a place 6 cm. in diameter and through an atheromatous patch 8 mm. thick. The splitting of the aortic layers proceeded circumferentially to a rupture through the adventitia and produced the hemorrhage already mentioned. The free blood in the retroperitoneal fat amounted to 1,500 cc. There was, in addition, at least 500 cc. of blood in the aortic wall, in the mesentery for the small bowel, and in the fat close to the left kidney.

The lungs were emphysematous anteriorly and subcrepitant dorsally. There were coal-pigmented nodules with gray fibrous centers, resembling arrested tubercles in the upper lobe and apex of the left lung. There was a similar nodule in the lower lobe of the right lung.

The root of the aorta had its lining altered about 50% by fatty and fibrous patches. This change increased gradually throughout the thoracic and abdominal portions of the aorta, so that at least 85% of the lining was involved at the bifurcation and in the common iliac arteries.

The heart weighed 465 gm. The outer wall of the left ventricle was 27 mm. thick, while that of the right ventricle was 5 mm. In many places 3 mm. of the latter measurement was due to fat. The circumferences of the heart valve rings were as follows: tricuspid 13.2 cm., pulmonic 7.4 cm., mitral 9.6 cm., and aortic 7.8 cm.

The coronary arteries were markedly tortuous, and the lining of the left was altered about 65% by fatty patches. The lumen was 11 mm. in circumference close to the mouth and was irregular but nowhere occluded. The right coronary artery was phenomenally altered. The mouth was 5 mm. in diameter. There were six aneurysms of this artery, all along the main descending branch. The first dilation had a calcified lining and was 8 mm. long and 11 mm. in diameter. It began 18 mm. from the artery mouth. The second aneurysm was centered 25 mm. from the first, was of the same size, and had about two-thirds of its lining covered with a partially organized blood clot. The third aneurysm was pouch-like, 6 mm. in diameter, and involved the outer aspect of the artery and almost all of the lining. With the exception of 10% of the left edge, the lining was covered with a stiff red clot, 1 mm. thick. The next aneurysm began 2 mm. more distally, was 18 mm. across, and had a clot obstructing both its entire lumen and that of the vessel on which it was situated. The clot was rubbery and dark red.

After a sclerotic course of 7 mm., the artery expanded into an aneurysm 2 cm. in diameter. This was filled with gray to pink grumous material, which resembled disintegrated clot with added fatty matter from an opened atheromatous region. After a straight interval of 5 mm., the last aneurysm was discovered. It was 7 mm. in diameter, and its lumen was filled with fresh red blood clots.

The myocardium of the apex of the left ventricle was scarred in one place 15 mm. in diameter. The lining of the left ventricle on its septal aspect contained eight white scars from 1 to 3 mm. broad and 7 to 12 mm. long. The outer wall of the left ventricle was extensively scarred by white patches from 1 to 5 mm. in diameter, especially in the central third.

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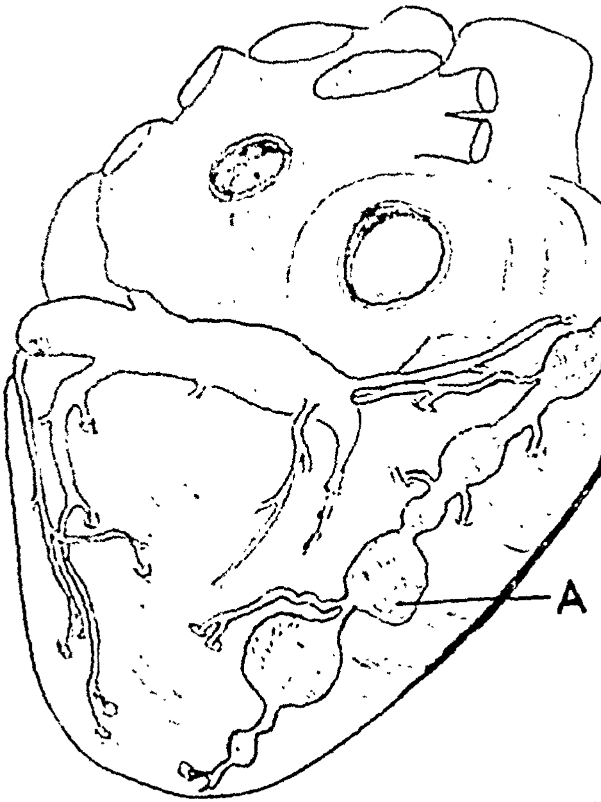
There was also an aneurysmal dilatation, 15 mm. in diameter, of the left common iliac artery 1 cm. distad to its mouth. The wall of the sac was sclerotic and as much as 7 mm. thick. The lining was partially dissected away by blood beneath it in a region 12 mm. in diameter. Here the detached lining was wrinkled. The other organs were remarkably free of changes other than sclerosis of the arteries. The spleen weighed only 75 gm., and its pulp was coarse and nearly bloodless.

Microscopic examination disclosed the following changes: The heart muscle cells showed hypertrophy and had moderate accumulations of polar liopochrome pigmentation. The scar tissue was, in general, old, but occasional sparse lymphocytic infiltrations were seen near the edges of the scars. There were also patches showing imperfect staining and haziness as is found in very early myocardial infarction.

Sections from the aortic aneurysm, whose dissection and rupture caused death, showed partial calcification in the atheromatous patch through which rupture occurred. The media showed the irregular track of dissection from the lining to the surrounding fat.



One (*A*) of six aneurysms of the right coronary artery. The heart is viewed from the back.

The aneurysm of the left common iliac artery showed a clot attached to the lining on its outer aspect. There was fresh hemorrhage into all layers of the artery wall. There was advanced sclerosis with partial calcification of the lining.

Sections of the aneurysms of the right coronary artery showed the changes typical of advanced senile sclerosis. There was extensive hyalinization of the media of the third aneurysm and acicular vacuolation indicative of cholesterol deposition. There was partial calcification of the lining.

The left adrenal gland had abundant hemorrhage into both cortex and medulla and into the surrounding fat. The cortical portions of the gland were dissected away from the capsule by hemorrhage in many places. In other regions there was indentation of the capsule from hemorrhage in the surrounding fat.

The prostate gland was involved in a benign hyperplasia of the left lateral lobe. This contained several well-circumscribed adenomas, in which there was papillary overgrowth of lining cells. There was also a slight interstitial lymphocytic infiltration close to the adenomas.

The anatomic diagnosis summarized the findings as follows: marked generalized senile arteriosclerosis; dissecting aneurysm of the abdominal part of the aorta; extensive hemorrhage into the retroperitoneal fat, urinary bladder wall, left adrenal gland, and mesentery for the small bowel; six aneurysms of the posterior coronary artery (several thrombosed); aneurysm with early dissection and clot formation of the left common iliac artery; benign prostatic hyperplasia; and scars of the knees (repair of quadriceps femoris tendons).

COMMENT

Rupture of an aneurysm of the aorta into the pericardial sac is occasionally recognized clinically. Tamponade of the heart by blood pumped into the pericardial sac causes rapid cardiac failure and death in most patients. The most frequent diagnosis is coronary thrombosis. The more gradual dissection through the wall of the aorta from the arch downward, either by rupture of a saccular aneurysm or by a true dissecting type, results in a perplexing series of symptoms. These are referable to the level of the aortic wall being split and are the result of a combination of factors. Chief among these are the disruption of the layers of the aortic wall by hemorrhage, the tamponing of aortic branches, the irritation of nerves in the vessel walls and involved organs, and ischemia of the organs. Patients so afflicted describe the onset of the sudden chest pain as gripping, pressing, squeezing, or "like a red hot poker" being pushed from the neck down through the chest.

In recent years the recognition of the true condition has led to a proper evaluation of the intense pleuritic pain, for example. This is due to dissection of the aorta by blood, which compresses the intercostal arteries especially in their course through the aortic wall. The intense pain in the various segments of the thorax is referable to the advancing thrust of blood within the layers of the aortic wall.

The speed of disruption is dependent on the blood pressure and on the resistance of the aortic layers primarily. The process may be temporarily halted if shock occurs; then the intramural blood may lose its driving force and clot. With the resumption of adequate pressure, the dissection may continue and encroach on any of the aortic branches. In this manner symptoms may occur that are ascribed to all the organ systems.

Death may not result from the first episode of dissection if it is slight. Again the patient's life may be spared if the column of intramural blood breaks through an atheromatous patch at one level in the aorta and, after a considerable distance, reestablishes contact with the aortic lumen. This condition may persist until a smoothly lined channel is formed in the aortic wall.

Dissection of this sort is not infrequent in saccular aneurysms of the abdominal aorta. Such was the condition in the case here reported. The bulging sac of the aneurysm was filled with a laminated thrombus in which there was extensive organization. Rupture of the aorta occurred in an atheromatous patch at the uppermost edge of the aneurysm. From this point the course was similar to that in a dissecting aneurysm, and death occurred in six hours.

The advanced sclerosis of the aortic branches was seen in the iliac arteries. On the left side this patient had a

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1. Long, M. C.: Aneurysms of Iliac Arteries, J. Gerontol. 3: 105, 1948. Jackman, R. J.; McQuarrie, H. B., and Edwards, J. E.: Fatal Rectal Hemorrhage Caused by Aneurysm of Internal Iliac Artery; Report of a Case, Proc. Staff Meet., Mayo Clin. 23: 305, 1948.

aneurysm in the common iliac artery 15 mm. in diameter. There was recent hemorrhage beneath the lining where dissection by blood had extended from the abdominal part of the aorta. Such aneurysms in themselves may lead to death.[1] In this patient it might have furnished the point of reentrance of blood into the vascular stream if death had not occurred so rapidly.

In addition to the aneurysms of the aorta and left common iliac artery, this patient had six aneurysms of the right coronary artery (see the accompanying figure). Their location on this artery and the condition of their lumens has already been detailed. Such aneurysms have been the subject of intensive study because of their rarity, their disputed causation, and their failure to produce typical symptoms.

Efforts by roentgenologists to demonstrate sclerotic cardiac coronary arteries in patients have been successful on numerous occasions. In 1927, Lenk[2] demonstrated calcified coronary arteries in the living by using the Potter-Bucky diaphragm. This was followed, in 1934, by a similar study by Wosika and Sosman.[3] They were able to show calcified coronary arteries in three patients. One of these subsequently was examined post mortem, so the clinical findings could be corroborated. More recently not only calcified coronary arteries but sclerotic changes in the heart valve rings have been reported by Koiransky and Ettman.[4] They also had corroboration of their findings at necropsy. Eventually coronary artery aneurysms may be found by such methods. In an article including a splendid review of the literature on coronary aneurysms, Scott[5] reported a globular shadow on the right heart border seen roentgenographically in his patient. At fluoroscopic examinations the shadow was seen to pulsate, and a diagnosis of probable aneurysm of the right auricle was made.

Scott made an exhaustive review and found 47 reported cases of coronary aneurysms since 1812. He classified these as diffuse and localized. The diffuse type apparently is on a congenital basis according to Harris,[6] Forbus,[7] and Rigdon and Vandergriff.[8] They develop in association with defects in the elastic lamella and muscular coats either at a branch or a bifurcation. The condition is similar to the changes noted in congenital aneurysms of the cerebral arteries.

The localized types have been divided as follows: congenital, 15; mycotic embolic, 12; syphilitic, 6; arteriosclerotic, 6; other types, 4; and unclassified, 4. In the 47 cases in which there was a localized aneurysm, the exact site was not specified in three. In the remaining 44, only the left coronary was involved in 27, and only the right in 11; there was involvement of both in six. Thirty-six bodies had single and eight had multiple aneurysms. In the group with congenital aneurysms, there were 14 male subjects and one female, ranging in age from 5 to 84 years. In the arteriosclerotic patients, four were men and one a woman. This group was from 58 to 77 years old. The mycotic-embolic group included six male and four female subjects, from 11 to 45 years of age. There were two additional cases in this group, but the sex was not stated. In the group with this type of aneurysm and the syphilitic variety there were 60% males, in marked contrast with the congenital variety, where males constituted 92.4% of the total.

The variety of alterations in this relatively small group of cases is fascinating. Prior to 1929, the most frequently reported cause of death from coronary aneurysm was rupture. Since that time coronary artery thrombosis and its sequelae have been most common. Not all patients died in a rapid or dramatic manner, however. Although three in the total series had chronic congestive heart failure, the general rule has been for these patients to present no symptoms referable to the heart.

The outstanding example of this fact was presented by Scott.[5] His patient, a man 84 years of age, died of carcinoma of the prostate gland and an ascending urinary infection. At autopsy his heart weighed 780 gm. There were 17 saccular aneurysms in an anomalous coronary artery circulation. The largest aneursysm was 10 by 8 by 8 cm. and was situated near the right auricle. This patient had an abnormal direct connection between the left coronary artery and an accessory right coronary artery. The vessel thus formed connected with the pulmonary artery. A person living to the age of 84 years with such a grotesque coronary circulation may serve as a natural experiment in altered blood flow to the heart. Venturesome surgeons are already furnishing new sources of blood supply to the heart by connecting a systemic artery to the coronary sinus.

Although the aneurysms of the coronary arteries usually produce no specific symptoms, many eventually cause death. Rarely does an aneurysm elsewhere in the body rupture and cause death in a patient who also has an aneurysm of a cardiac coronary artery. There is only one such case reported to date, by Manohar.[9] My case, as far as can be ascertained, is the second to be reported. The patient had six aneurysms of the right coronary artery, an aneurysm of the left common iliac artery, and a huge abdominal aortic aneurysm. The combination is unique for cases of the arteriosclerotic type of aneurysm.

SUMMARY

The literature on aneurysms of the cardiac coronary arteries is reviewed, and pertinent articles are cited. The clinical history and postmortem findings are reported in a case of multiple coronary aneurysms; the patient died of rupture, with dissection, of an arteriosclerotic aortic abdominal aneurysm.

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[2] Lenk, R.: Röntgendiagnose der Koronarsklerose in vivo: Gleichzeitig ein Beitrag zur Erkennbracket des Herzaneurysmas in Röntgenbilde, Fortschr. a. d. Geb. d. Röntgenstrahlen **35**: 1265, 1927.

[3] Wosika, P. H., *and* Sosman, M. C.: Roentgen Demonstration of Calcified Coronary Arteries in Living Subject, J. A. M. A. **102**: 591 (Feb. 24) 1934.

[4] Koiransky, H. G., and Ettman, I. K.: Roentgen Demonstration of Calcification of Cardiac Skeleton and Coronary Vessels in the Living, Am. J. Roentgenol. **60**: 642, 1948.

[5] Scott, D. H.: Aneurysm of the Coronary Arteries, Am. Heart J. **36**: 403, 1948.

[6] Harris, P. N.: Aneurysmal Dilatation of the Cardiac Coronary Arteries; Review of the Literature and Report of a Case, Am. J. Path. **13**: 89, 1937.

[7] Forbus, W. D.: On the Origin of Miliary Aneurysms of Superficial Cerebral Arteries, Bull. Johns Hopkins Hosp. **47**: 239, 1930.

[8] Rigdon, R. H., and Vandergriff, H.: Aneurysms of the Coronary Arteries: Review of the Literature and Report of a Case, Am. J. Surg. **61**: 407, 1943.

[9] Manohar, K. D.: An Aneurysm of a Coronary Artery, Arch. Path. **26**: 1131, 1938.

HEMOBILIA, CHOLECYSTITIS, AND GASTROINTESTINAL BLEEDING WITH RUPTURE OF LIVER

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Subcutaneous rupture of the liver is not a rarity. Our case is of particular interest on account of the complications of hemobilia, hemorrhagic cholecystitis, gastrointestinal bleeding, hemothorax, and hydropneumopericardium, with complete recovery. The over-all mortality from hepatic rupture in general remains about 60%, that from massive rupture being higher (60% to 80%).[1]

REPORT OF A CASE

R. G., 9 years of age, a white male child, sustained a severe injury to the abdomen and lower right portion of the chest at 5 p. m. on Feb. 9, 1951, when he fell from his sled into a jagged rock-filled creek. He was brought into the office 20 minutes later in a state of collapse. Shock therapy was immediately carried out with favorable response. X-rays were taken of the abdomen to determine the presence of air under the diaphragm and because of possible fracture of the ribs. The x-rays were all normal. His pulse rate was very slow (40 per minute). He moaned and complained of severe pain in the lower right part of the chest and right upper quadrant of the abdomen. Two central upper incisors were broken transversely in half. The diaphragm did not move with respiration, and there was a large ecchymotic area over the right upper abdominal quadrant. The abdomen was fixed, and peristalsis was not audible. A careful search of the head and spine did not reveal any evidence of trauma. Results of neurological examination disclosed no abnormalities; the eye grounds were normal. While the patient had reacted from the initial shock, the results of clinical survey and the severity of the trauma strongly suggested the possibility that a visceral injury and internal hemorrhage had occurred. The patient was thereupon transferred to the Mount Sinai Hospital.

Physical examination at the hospital revealed a rapid and feeble pulse, subnormal temperature, and cold clammy skin. Blood pressure was 80/30, and respirations were 30 per minute. The right side of the chest was fixed. The muscles of the right upper quadrant of the abdomen showed increased muscle resistance and absent reflexes.

The impression on admission was hemoperitoneum, probably due to a ruptured liver or spleen, with secondary shock and internal hemorrhage. He was given a transfusion of whole blood immediately, then glucose intravenously, a procaine penicillin G preparation (crysticillin®), vitamin K, and other vitamins. The blood cell count showed 62% hemoglobin (9 gm.), 20,850 white blood cells, and 33% hematocrit. The clinical evidence of hemorrhage clearly indicated the necessity of urgent surgery in an effort to locate the source of the bleeding.

Under general anesthesia, an upper right transverse abdominal incision transecting the right rectus sheath and muscle was used to open the abdomen. The peritoneal cavity was filled with bright red blood and clots, after the removal of which a thorough abdominal exploration disclosed a deep laceration of the posterior-inferior aspect of the right lobe of the liver, lateral and posterior to the gallbladder fossa and inferior vena cava. The hepatic area was sutured with surgical gut. The omentum was placed over and in the traumatized hepatic surface, being utilized as a cushion for the sutures. (Omentum acts as a valuable hemostatic and agglutinizing agent and must be sutured to the area of rupture.) Three pieces of gelfoam® (absorbable sponge) were also applied to the sutured site to further control hepatic bleeding. With the bleeding apparently controlled and the visceral injury repaired, the abdomen was closed in layers. The patient left the operating room in fair condition. He was given 750 cc. of whole blood and 250 cc. of 5% glucose by venoclysis during the operation.

On the first postoperative day, the abdomen was softer but peristalsis was still absent. A Levine tube was inserted to control vomiting and reduce gastric distention. Continuous Wangensteen suction was maintained. On the third postoperative day, fluid was found in the right pleural cavity. Hemoglobin was 73%. A transfusion of 250 cc. of whole blood was given. The temperature was sustained at 102 F. Tenderness at the site of his "cut down" in the ankle suggested the possibility of thrombophlebitis. He was treated with antibiotics, oxygen, and fluids given parenterally. X-ray studies showed that the right side of the diaphragm was fixed. It was difficult to determine at this time whether the fixed position of the diaphragm was the result of fluid in the pleural cavity or the beginning of a subphrenic collection. His fluid and electrolyte balance were maintained within normal limits.

On the sixth postoperative day, aspiration of the right pleural cavity yielded 450 cc. of brownish, bloody fluid. The aspirated pleural fluid showed xanthochromia. The right side of the diaphragm continued to be elevated. Persistent physical findings of fluid in the right pleural cavity required repeated aspirations.

Two weeks postoperatively, sharp pain developed in the right upper quadrant of the abdomen radiating to the right shoulder. Concomitant with the pain, vomiting of about 100 cc. of bright red blood and blood clots occurred. Blood pressure was 130/90 and pulse rate 96 per minute. Scleral jaundice and dark urine were evident. A gastrointestinal x-ray was normal.

The next day, the jaundice increased in intensity. The patient again vomited bright red blood and passed a tarry stool. Hemoglobin was 73%. The clinical picture suggested the presence of an acute duodenal ulcer resulting from the initial trauma. An ulcer regimen was instituted, with no subsequent clinical improvement. The pain in the right upper quadrant of the abdomen and hematemesis continued. The hemoglobin dropped to 60%. Frequent transfusions were given but to no avail. The development of a mass in the right upper abdominal quadrant and the patient's failure to respond to treatment prompted the decision to explore the abdomen again.

Accordingly, the patient was operated on 19 days after the first operation. Under general anesthesia, the abdomen was opened through the previous transverse incision. The abdominal cavity was dry, with no gross evidence of bleeding. The gallbladder was found to be acutely inflamed, distended to about six to eight times its normal size, and showed massive, diffuse mural edema. Insertion of a trochar into the gallbladder yielded thick blood clots and bile. A cholecystostomy was performed and a heavy rubber catheter inserted through the trochar opening in the gallbladder. Drainage continued satisfactorily from the cholecystostomy opening. Clinical improvement was immediately evident and continued to complete recovery. The drain from the gallbladder was removed after seven days. Several chest taps were performed, with the removal of slightly blood-tinged fluid. An x-ray of the chest six days after the second operation showed the presence of a small hydropneumopericardium on the right side at the level of the eighth dorsal segment. Since it did not cause any symptoms, it was allowed to absorb spontaneously.

The patient was discharged on March 10 (29 days after admission and 10 days after his second operation), in good condition. Follow-up x-rays two weeks after his discharge from the hospital revealed disappearance and absorption of the chest fluid.

The patient was last seen in January, 1952. He was healthy and active. Liver function tests showed normal hepatic function. A cholecystogram showed normal gallbladder function.

COMMENT

The interesting phases of this case were the occurrence of hemobilia, hemorrhagic cholecystitis, and gastrointestinal bleeding as complications of a ruptured liver. Hemobilia is a term suggested by Sandblom[2] to describe the symptom of bleeding into the biliary tree due to

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From the Mount Sinai Hospital.

Dr. T. H. Mendell assisted in the management of the patient described.

[1] Sanders, G. B.; Macguire, C. H., and Moore, R. H., Jr.: Massive Rupture of Liver, Am. J. Surg. 78: 699 (Nov.) 1949.

[2] Sandblom, P.: Hemorrhage into the Biliary Tract Following Trauma: "Traumatic Hemobilia," Surgery 24: 571 (Sept.) 1948.

trauma. His investigations reveal that this condition is rare. He described a 7-year-old boy who was hurt by the handlebar of a bicycle and who was operated on 10 days after the injury. A cholecystostomy with drainage was carried out for a distended gallbladder, the contents of the latter revealing bile mixed with blood. Opening of the gallbladder disclosed no definite bleeding site.

Siegel[3] (quoted by Sandblom) described a 32-year-old man who had sustained a blow from the handlebar of a bicycle. Several ribs on the right side were fractured, and bleeding into the right pleural cavity occurred. On the 10th day, the patient passed a blood clot via the bowel and felt better. On the 14th day he was operated on and a large distended gallbladder was found. There were several ruptures in the liver, varying up to 4 cm. in length. A spotty gangrenous gallbladder was removed and the gallbladder bed drained. Postoperatively there was considerable sanguineous drainage followed by the development of icterus. These were attributed to blood clots in the common duct. The patient gradually recovered without further complications.

Sandblom interpreted this case as one of hemobilia, the result of a central rupture of the liver, with the blood carried to the gallbladder through the biliary passages. He collected nine additional cases in which the source of the bleeding was attributed to probable rupture of the central portion of the liver.

Central rupture of the liver beginning in the center of the liver and extending to its periphery usually results from a compression type of trauma. Extension of a central hepatic rupture to or near the portal fissure may readily establish a communication between the vascular tree and one of the larger hepatic ducts. Thus, blood gains entrance into the duodenum and produces hematemesis and melena. The great danger of hepatic rupture is bleeding, particularly from the portal vein branches. The veins of the liver have little supporting tissue and do not tend to collapse. When mixed with bile, the blood shows little tendency to clot.

One cannot exclude the possibility that in our case the initial trauma of the liver was greater than suspected. The injury probably extended toward the portal fissure, where it communicated with the larger biliary ducts. It is conceivable that some of the blood could have drained through the hepatic duct into the cystic duct and remained in the gallbladder. The continued slow bleeding into the biliary tract was clinically masked by the numerous transfusions of whole blood. Two or more weeks elapsed before symptoms referable to this bleeding manifested themselves. By that time, the gallbladder was considerably distended, its walls thickened not unlike those observed in a cystic duct obstruction. The acute onset of jaundice is explained by the sudden blocking of the common duct by a blood clot or bile plug. The rapid disappearance of the jaundice suggests the clot was pushed into the duodenum, thereby opening the avenue for the normal escape of bile.

In further support of the concept that multiple hemorrhages through the biliary tract may stem from a central rupture of the liver is the case reported by Hawthorne and co-workers.[4] In this instance, the hemorrhage through the biliary tract was due to a cavity and a hematoma deep in the liver that continued to necrose and bleed through a large biliary duct until healing of the hepatic rupture occurred.

Additionally, our patient exhibited jaundice while his mother was recuperating from a severe form of infectious hepatitis. The child had last kissed his mother two months previously, when she was first admitted to the same hospital with a diagnosis of infectious hepatitis. The incubation period was well in line with the possible development of this disease in the child. However, subsequent liver function tests suggested an obstructive rather than hepatocellular jaundice.

SUMMARY

A case of ruptured liver complicated by hemobilia, traumatic hemorrhagic cholecystitis, gastrointestinal hemorrhage, hemothorax, and pneumohydropericardium with subsequent complete recovery is reported.

5217 Lebanon Ave. (31) (Dr. Epstein).

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[3] Siegel, E.: Ein Fall von traumatischer Gangrän der Gallenblase nebst einigen Bemerkungen über den Wert der Darmaufblähung zur Bestimmung der Lage von Unterleibstumoren, München med. Wchnschr. 56: 341, 1909.

[4] Hawthorne, H. R.; Oaks, W. W., and Neese, P. H.: Liver Injuries with a Case Report of Repeated Hemorrhages Through the Biliary Ducts, Surgery 9: 358 (March) 1941.

INTUSSUSCEPTION OF THE VERMIFORM APPENDIX

REPORT OF A CASE

David B. Allman, M.D.
Milton Ackerman, M.D.
and
Harry S. Hoffman, M.D., Atlantic City

The first report in the literature on intussusception of the appendix was made by McKidd,[1] in 1858. His young patient died one month after he was first seen, without benefit of surgical treatment. The tremendous strides in surgery since then have established successful therapeutic measures, but preoperative diagnosis has lagged far behind. Rarely has a correct preoperative diagnosis been arrived at, although certain characteristics of the disease are definite enough to warrant its inclusion in the differential diagnosis of surgical disease of the abdomen.

The case reported here is of interest because of the type of intussusception; an invagination of the appendix into itself is rare, and only three previous cases have been reported. The fourth case of this type is reported with a brief discussion of the possible causative factors and a review of the literature.

REPORT OF A CASE

The patient, a 65-year-old white man, was admitted to Atlantic City Hospital June 30, 1951. He was first seen that morning, complaining of severe, cramp-like, intermittent pains in the lower right quadrant of the abdomen, lasting one to three minutes and recurring at intervals of one to two minutes. These had been present for 24 hours prior to admission to the hospital. There was no vomiting associated with the pain, although self-induced vomiting relieved the pain. These attacks had been

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From the Department of Surgery (Dr. Allman, Surgical Director) and the Department of Laboratories (Dr. Ackerman, Director), Atlantic City Hospital.

similar and recurrent since 1949. At the insistence of his wife, he permitted a physician to see him, and immediate hospitalization was advised. Past history revealed that, in 1937, after sigmoidoscopy, an adenomatous polyp of the sigmoid colon was found and removed. There was no evidence of recurrence following this operation. In January, 1951, he consulted a gastroenterologist about abdominal, cramp-like pains of about two years' duration. There had been some slight weight loss. His general history, otherwise, was not remarkable. Physical examination failed to reveal any palpable portion of the colon. No masses could be felt. There was some slight increase in tympany throughout the abdomen, with occasional hyperperistaltic sounds. Results of rectal examination were negative. Sigmoidoscopic examination, for full distance of the 10-in. scope, disclosed only internal hemorrhoids. Examination with a barium enema showed only a spastic colon. Laboratory studies in January, 1951, showed a normal stool, blood cell count, blood sugar, and blood urea nitrogen and a negative Wassermann reaction.

The patient was pale and thin and did not appear ill. His pulse rate was 96, respirations 20, and temperature 99 F. His tongue and skin were dry. His abdominal wall showed no scars. There was slight distention. A sense of resistance was felt over the entire right side of the abdomen, most marked in the lateral area. No palpable masses were present, and the liver and spleen could not be felt. There was a definite area of tenderness pointing to the right costovertebral area. Results of rectal examination were negative.

Laboratory Data.—There were 16,200 white cells, with 60% segmented and 22% nonsegmented polymorphonuclear cells and 18% lymphocytes. The erythrocyte sedimentation rate was 30 mm. per hour. The blood urea nitrogen was 15 mg. per 100 cc. The urine contained acetone (2+) and a faint trace of albumin.

Course.—A preoperative diagnosis of acute suppurative appendicitis was made, and surgical procedures were instituted. With thiopental sodium anesthesia, laparotomy was performed, through a right rectus incision by one of us (D. B. A.). There was free fluid in the abdomen. The appendix was greatly thickened and shortened and appeared to be intussuscepted. The intussusception was not reduced, but the base was ligated and the appendix was removed by the retrograde method. Ten grams of a sulfonamide preparation was introduced into the abdominal cavity. Two Penrose drains were inserted and the abdomen closed.

Pathological Data.—The appendix measured 3.5 cm. in length and 2.5 cm. in its average diameter. The serosa was covered by a patchy, purulent exudate, and there were tabs of ragged, fibrous connective tissue adhering to it. The distal third was the intussusceptum and completely plugged the lumen of the middle third, or intussuscipiens. The wall of the proximal third was thickened; the mucosa of this portion was congested and its lumen narrowed. The intussuscipiens could be opened for only a short distance beyond the tip of the intussusceptum, beyond which point the two were fused. A cross section, distal to this point, showed a double-barreled structure with marked fibrous thickening of both walls.

Microscopic Examination.—A section of the appendix showed a double-barreled organ. The outer wall had an inner lining of flattened, congested mucosa. The submucosa and muscularis of this wall were broadened by a hyalinizing fibrosis and showed a light infiltrate of lymphocytes and a few polys. In the subserosa, there was capillary engorgement and a moderate polymorphonuclear infiltrate; a thin, fibrinopurulent exudate adhered to the serosa. The intussusceptum, occupying the lumen of this structure, showed a compressed mucosa and a hyalinizing fibrosis of the submucosa and muscularis with a sprinkling of lymphocytes and monocytes in the muscularis and subserosa.

Diagnosis.—The diagnosis was focal acute suppurative appendicitis due to intussusception of the vermiform appendix.

Postoperative Course.—The patient's course following operation was uneventful, and he was discharged from the hospital on the eighth postoperative day.

COMMENT

Less than 100 cases of intussusception of the appendix have been recorded in the literature since the first report of this condition was made by McKidd.[1] In 1941, McSwain [2] conducted a complete and thorough analysis of 77 cases and found the average age of the patients to be about 16 years; the youngest was 13 months, and the oldest was 75 years. He pointed out that the time lag between onset of symptoms and operation ranged from two hours to nine years.

Three types of intussusception of the appendix have been described by Szenes [3]: (1) invagination of the appendix into itself, (2) partial or complete invagination of the appendix into the cecum, and (3) compound intussusception wherein the intussuscepted appendix progresses distally into the colon, including the ileum. Of the total of 82 cases reviewed by Fraser,[4] the first type was present in 2 cases, the second in 44 cases, and the third in 32 cases. The remaining four cases were of intussusception of inverted appendiceal stumps.

The first case of invagination of the appendix into itself was published by Whitrow,[5] in 1930. The patient was a 12-year-old boy who was admitted to the hospital with a history typical of acute appendicitis of 18 hours' duration. At operation, the appendix was found to be the site of intussusception, the tip having invaginated itself until it lay at the base of the appendix. An appendectomy was performed, and the patient's recovery was uneventful.

The second case of intussusception of this type was reported by Fraser,[4] in 1943. His patient was a 75-year-old woman who had both intussusception of the appendix and carcinoma of the cecum. In this instance, the appendiceal intussusception was at the base of the appendix, where the walls were telescoped on themselves for ½ in. (1.3 cm.). The patient's symptoms were referable to the cancer of the large bowel, and the intussusception was observed incidentally.

The third case was reported in 1945 by Ingersoll and Meigs.[6] Their patient was a 45-year-old woman who had intussusception of the appendix with associated endometriosis in that organ.

The mortality rate found by McSwain [2] for intussusception of the compound type was 14.8%, while that of simple intussusception of the appendix was only 2.6%. In the compound type, the intussusception of the appendix will be masked and will not be apparent until the compound intussusception is reduced at operation or it will be discovered postoperatively when the resected specimen is examined. The extent to which the intussusception has progressed will depend on the length of time it has been allowed to persist.

Causation.—Many theories have been presented to explain the development of intussusception of the appendix. Wakley [7] suggested that hyperactive peristalsis

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1. McKidd, J.: Case of Invagination of Caecum and Appendix, Edinburgh M. J. **4**: 793, 1859.
2. McSwain, B.: Intussusception of Appendix: Review of Literature and Report of a Case, South. M. J. **34**: 263-271 (March) 1941.
3. Szenes, A.: Invagination of Appendix, Arch. f. klin. Chir. **119**: 88-115 (July) 1922.
4. Fraser, K.: Intussusception of Appendix, Brit. J. Surg. **31**: 23-33 (July) 1943.
5. Whitrow, F.: Intussusception of Appendix, Brit. M. J. **2**: 181 (Aug.) 1930.
6. Ingersoll, F. M., and Meigs, J. V.: Intussusception of Vermiform Appendix: Report of Case, Arch. Surg. **51**: 172-173 (Oct.) 1945.
7. Wakley, C. P. G.: Intussusception of Appendix: Report of 2 Cases, Lancet **2**: 691 (Sept. 23) 1939.

provoked by intestinal parasites or fecaliths may be responsible in some cases. He suggested, too, that the increased lymphoid tissue in the terminal ileum and appendix in young children acts as a foreign body and gives rise to increased and irregular peristalsis of bowel and appendix, which sometimes culminates in intussusception. MacDermott[8] conducted anatomic studies of the base of the appendix and found there a muscular sphincter. He thought the spasm of this sphincter would give rise to a hard mass at the base of the appendix, which would act as the apex of an intussusception. Fraser[4] found the cause of intussusception to be an inversion of the appendiceal stump in 4 patients in whom the appendix had been removed. McSwain,[2] likewise, noted this possibility and brought up for consideration the advisability of invaginating the stump of the appendix after an appendectomy. Ingersoll and Meigs[6] noted that the appendix is capable of rhythmic peristaltic contractions. These contractions, on encountering an area of inflammation within the wall of the appendix or an adherent concretion, result in a buckling inward of the mucosa, followed by the muscularis and serosa with the development of intussusception. If the base of the appendix is constricted and a foreign body or fecalith becomes impacted, the forceful peristaltic action of the appendix may carry that organ into the cecum. Previous inflammatory processes, such as acute appendicitis, may, therefore, set the stage for intussusception.

Symptoms.—McSwain[2] summarized the salient clinical manifestations of intussusception of the appendix as follows: The pain was generally intermittent, cramp-like, and paroxysmal, with complete relief at intervals. Nausea and vomiting almost universally accompanied the pain in 77 patients. Seventeen had blood in the stools. In the majority of patients, the bowels moved normally. There was only slight fever and slight increase in pulse rate or leukocyte count, except in patients in whom necrosis, gangrene, or peritonitis occurred. Distention occurred infrequently, and a mass was noted in 43 patients. There was little muscle spasm, and tenderness was not extreme.

The differentiation of acute appendicitis from intussusception of the appendix, unaccompanied by ileocolic intussusception, is difficult. McSwain[2] suggested the following points to aid in the differential diagnosis: (1) a history of more attacks than are usually seen in acute appendicitis; (2) more definitely cramp-like pains, similar to those of early intestinal obstruction; (3) a history of blood or mucus or both in stools, or their discovery on examination; (4) a history of palpation of a mass in the right lower abdominal quadrant; (5) less fever, increase in pulse rate, and leukocytosis than would be expected in a patient with an appendix inflamed enough to cause a clinical picture of equal severity.

SUMMARY

A case of invagination of the appendix into itself in a 65-year-old man is reported. Intussusception of the appendix into itself is rare, and only three cases have been reported previously. A review of the literature is included in the report.

26 S. Ohio Ave.

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8. MacDermott, E. N.: Appendiceo-Caecal Intussusception, Brit. M. J. 1: 793-794 (May 9) 1931.

COUNCIL ON PHARMACY AND CHEMISTRY

REPORT TO THE COUNCIL

The Council has authorized publication of the following report from the Committee on Pesticides, a standing committee of the Council on Pharmacy and Chemistry.

R. T. Stormont, M.D., *Secretary.*

The following article is the fourth in a series of reports concerning pesticides of immediate medical interest. The Committee wishes to acknowledge the assistance of Drs. Robert W. Lackey and Joseph F. Treon, who reviewed the manuscript.

Bernard E. Conley, *Secretary*
Committee on Pesticides

PHARMACOLOGIC PROPERTIES OF TOXAPHENE, A CHLORINATED HYDROCARBON INSECTICIDE

Toxaphene is the accepted common name for a polychlor bicyclic terpene with insecticidal properties. It may be more properly identified as a chlorinated camphene with the average empirical formula $C_{10}H_{10}Cl_8$. The structural formula has not been definitely established since the chlorination of camphene may involve the addition or substitution of chlorine, a rearrangement of the molecule, or a combination of these reactions.

The technical or insecticidal grade is an amber-colored, waxy solid with a mild pine-like odor. It contains 67 to 69% chlorine and melts in the range 70 to 95 C. Although insoluble in water, toxaphene is readily soluble in inexpensive commercial solvents, especially those of an aromatic hydrocarbon nature. It is practically nonvolatile and has residual properties resembling those of DDT (dichloro-diphenyl-trichloro-ethane). Like other chlorinated hydrocarbons of this type, toxaphene slowly evolves hydrochloric acid when heated, the rate depending upon temperature and the presence of catalytic impurities, such as iron compounds. Strong alkalies and ultraviolet light cause a similar breakdown.

The insecticidal effectiveness of toxaphene is caused by combined contact and stomach poison effects. It is a relatively slow acting residual insecticide which is formulated for use as a dust, emulsion, spray, or wettable powder, and incorporated into bait for grasshopper control. Because of its slow action, paralytic agents such as iso-bornyl thiocyanoacetate are used in contact sprays to assure a rapid knockdown of insects. Toxaphene has been used extensively against cotton insects and for the control of grasshopper infestations. It also has been used as a supplement to, and substitute for, DDT for the control of insects resistant to DDT. Toxaphene sprays are not considered to be safe for household application, nor have acceptable medicinal uses for toxaphene been discovered. At one time it was believed to be comparable to DDT as a louse powder because of its persistence and resistance to laundering. Uncertainty as to its cutaneous toxicity in powder form precludes its use for this purpose. In medical entomology, however, it is proving to be a valuable agent for the control of the ticks and mites which transmit disease.

PHARMACOLOGY

Toxaphene is a general convulsant which acts on the central nervous system. It causes diffuse stimulation of the brain and spinal cord resulting in generalized convulsions of a tetanoid or clonic character. In this respect, it acts somewhat the same as DDT and certain other chlorinated hydrocarbon insecticides. Since toxaphene is a chlorinated camphene, it is suspected to have the medullary excitant effects of camphor. Such effects have been observed in several species of laboratory animals.

The occurrence of salivation, vomiting, and reflex excitability to sound suggests a generalized stimulation of medullary centers.

Stimulation of the vasomotor center, evidenced by the abrupt rise in blood pressure in the presence of complete muscular paralysis, occurs in curarized animals following intravenous doses of the compound. There are, however, no significant changes in mean arterial blood pressure following oral administration of toxaphene. This difference in response is probably accounted for on the basis of incomplete absorption of the toxicant from the gastrointestinal tract. The generalized epileptiform spasms which terminate in respiratory failure are also characteristic of a camphor-like compound.

Toxaphene apparently is absorbed irregularly, as a consistent range of toxicity has not been noted in experimental animals. Absorption occurs from the alimentary tract, respiratory tract, or skin, and is partially influenced by the physical state of the technical product. In solution, toxaphene is readily taken up; in the solid form, it is absorbed in toxic amounts depending on the species of animal and the concentration of the powder (dust) used. For example, cutaneous exposure to the 40% technical dust proved to be lethal for rabbits, although higher concentrations applied to the skin of dogs were well tolerated.

Little is known about the distribution, storage, detoxification, and excretion of the compound. The lack of suitable microanalytical chemical methods for the positive identification of minute quantities of toxaphene in tissues or body fluids prevents an accurate determination of its metabolic fate. The high lipoid solubility possessed by this substance suggests that it is probably distributed throughout the various fat depots of the body. Determination of the chlorine content of brain tissue from chronically poisoned dogs indicated storage of the compound as a chlorinated derivative, although the organic chloride content of body fat was not significantly altered. With steers which had been similarly exposed, however, the amount stored in fat tissue was one to three times the amount ingested over a given period.

Toxaphene is believed to be slowly detoxified in the liver. This assumption is based on two observed characteristics; namely, its close chemical relationship to camphor, which is known to be detoxified in the liver, and the isolation of ethereal sulfate and glucuronic acid conjugates of toxaphene in the urine. Toxaphene is also excreted in milk.

TOXICOLOGY

Toxaphene has an irregular toxicity range which basically is due to the vagaries of the absorptive mechanisms in the various animal species. It is toxic by inhalation, ingestion, or skin contact in all forms, but the route of administration and the form in which it is available materially alter the degree of toxicity.

Wide variation in susceptibility has been noted among animals of the same species as well as among those of different species. Dogs are the least tolerant mammals to orally administered toxaphene, followed in an increasing order of resistance by cats, rabbits, rats, guinea pigs, and the larger domesticated animals. This pattern of susceptibility generally is similar to that of most chlorinated hydrocarbon insecticides with two exceptions. Birds, as a group, are not resistant to the compound, having a toxicity range comparable to that for the smaller laboratory animals (50 to 200 mg. per kg. of body weight). Conversely, toxaphene is much more toxic to fish than most other compounds in its class. In this respect it is as toxic as, or more toxic than, rotenone. For this reason, it has been suggested as a useful substitute for the plant insecticide in fish-management work.

The oral toxicity of toxaphene places it among the more toxic of the commercially available chlorinated hydrocarbon insecticides which are acutely poisonous by this route of administration. The oral $L.D._{50}$ ranges from 15 to 375 mg. per kg. of body weight depending on the species of animal and formulation employed, while the intravenous $L.D._{50}$ lies between 10 and 15 mg. per kg. of body weight. This wide variation is believed to be indicative of the irregularities in intestinal absorption which may be expected among most animal species. Since a similar variation among animals exists with orally administered DDT, estimations indicating that toxaphene is approximately four times as acutely toxic as DDT are valid. The fatal dose for man has been estimated to be 2 to 7 gm. Three human fatalities and eight cases of nonfatal poisoning from the accidental ingestion of toxaphene preparations and residual amounts of the technical material alone and on edible crops are reported elsewhere in this issue of THE JOURNAL (see page 1124).

The chronic toxicity of toxaphene for rats is less than that of DDT, being closer to that of lindane. Dogs are particularly susceptible to small amounts of toxaphene given repeatedly over long periods of time, while other animals similarly exposed appear to have a somewhat greater tolerance, especially in the lower dosage ranges. Gross injurious effects, such as retardation of growth, greater incidence of mortality, increased size of vital organs, loss of appetite, and occasionally other signs of chronic poisoning are evident in rats after repetitive feedings of toxaphene at a level of 100 ppm for a period of two years. Cumulative effects of poisoning in dogs, however, are noted at much lower levels of toxaphene in the diet. These variations in response among animal species coupled with the inadequacy of behavior and changes in appearance as criteria of chronic injury make it difficult to interpret laboratory data in terms of human hazard. For these reasons, histopathologic studies are being relied upon increasingly to measure the chronic toxic effects of chemicals. On the basis of such evaluations of chronic toxicity, toxaphene has been found to compare favorably with many of the chlorinated hydrocarbon and metallic insecticides, although less so in comparison with most of the botanical insecticides and the remaining synthetic insect control chemicals in common use.

In laboratory animals toxaphene in concentrated form is reported to be a mild to moderate skin irritant. A conventional patch test of human subjects yielded no evidence of irritation or sensitization. Irritation of the eyes, nasal mucosa, and throat has been reported in persons exposed to toxaphene dusts and sprays. These manifestations are believed to be caused primarily by the diluent or solvent used in the formulations.

Systemic effects may be induced by absorption of toxaphene through the skin in dry form and in solution. Absorption through abraded skin surfaces is the most rapid. Dry formulations of the insecticide are the most variable in this respect and skin hazards are dependent on several factors. The rate and extent of cutaneous absorption of such preparations depend on the concentration and extent of exposure, the presence of wetting agents (as used in wettable powders), and the susceptibility of animal species. High concentrations of dusts (50%) do not elicit symptoms in dogs, although lesser concentrations are lethal to rabbits. Wettable powder preparations have provoked symptoms in cattle, although to a lesser degree than oil solutions. Oil formulations and emulsive forms of the chemical are readily absorbed through the skin and are the most dangerous. A rapid vat-side test for assaying the amount of toxaphene in cattle dips has been developed which allows the use of dip solutions without running the risk of serious injury to the animal. It is not recommended for use on dairy cattle or in dairy barns because of the danger of milk contamination.

When it enters the respiratory tract toxaphene is potentially toxic, especially in solution. Erratic results were obtained in the available animal studies on this phase of its toxicity. Forty, 100, and 60% mortalities were obtained in groups of mice exposed to oil-based mists of toxaphene in a concentration of 0.2 mg. per 100 cc. of air per minute for 112, 116, and 158 minutes, respectively. On the basis of this test, the $L.C._{50}$ for mice is 0.2 mg. per 100 cc. of air per minute for a two-hour exposure period. Absence of significant consolidation in the lungs suggests that death was primarily caused by toxaphene. Field studies of livestock exposed to toxaphene solutions used as cattle dips indicate that the insecticide may be toxic both cutaneously and by inhalation, especially to the young of the species. Autopsy of calves dipped in toxaphene solutions showed lung involvement which was attributed to the dip solution.

Gross pathologic changes are uncommon in either acute or chronic toxaphene poisoning. Histologic studies of chronically poisoned animals revealed degenerative changes in the renal tubules and liver parenchyma. The latter are characterized by centrolobular hypertrophy of the hepatic cells which may progress to central necrosis and reparative hypertrophy. The brain, heart, lungs, skeletal muscles, adrenals, pancreas, stomach, small and large intestines, and spleen were uninjured.

Symptoms which may be anticipated in human poisoning from toxaphene are those of a convulsant drug affecting the brain and spinal cord. Generalized epileptiform convulsions are the most prominent features in acutely intoxicated animals and may be expected in human counterparts. Salivation is usually the first sign of toxicity in animals. This is followed by emesis, which is believed to be caused by direct stimulation of the emetic center. Shortly after the onset of vomiting, intermittent muscle spasms develop. These are followed by generalized convulsions at three- to five-minute intervals. Convulsions become progressively more frequent until finally a convulsive state exists which terminates in exhaustion and death from respiratory failure. The onset of symptoms usually occurs in one hour, although symptoms may be delayed as long as three or more hours after an oral toxic dose. Death occurs in four to eight hours or may be deferred up to 24 hours.

The treatment of toxaphene poisoning is directed toward the evacuation of the stomach and bowels and control of convulsions and other central nervous system manifestations. Gastric lavage and saline cathartics are the accepted measures for the removal of ingested insecticide. Toxaphene-induced convulsions in experimental animals seem to be controlled with greater ease when this practice is followed. This is probably because of the great affinity for lipoid substances and the negligible water solubility which is characteristic of toxaphene and other chlorinated hydrocarbon insecticides. Oily cathartics and fatty substances are usually contraindicated because of their capacity to increase absorption of the toxicant. Prompt removal of the oil-solubilized insecticide before passage into the intestines, however, reduces the chance for absorption of appreciable amounts of the toxicant.

A variety of drugs have been tried for the control of toxaphene convulsions. These include morphine, paraldehyde, methadone, dilantin,® tridione® with urethane, the sodium salts of amytal,® pentobarbital, and phenobarbital. All were given intravenously except paraldehyde, which was given intramuscularly, orally, and rectally. Morphine, methadone, and tridione® with urethane were found to be ineffective in experimental animals. Paraldehyde effectively controlled convulsions when given in large doses intramuscularly but produced sloughs. It was less effective by stomach and by rectum. Dilantin,® when given with small amounts of phenobarbital, was effective when given early but it was ineffective for recurrent convulsions after an initial dose of phenobarbital.

All of the barbiturates used were effective in controlling convulsions. When treatment is instituted before the onset of convulsions, long acting depressants, such as phenobarbital sodium, are the drugs of choice. If convulsions develop prior to treatment, short acting barbiturates, such as pentobarbital sodium, are considered to be more effective, since a full anesthetic dose of the drug can be given if necessary. Experience with animals indicates, however, that convulsions can be most readily controlled if treatment is begun before their appearance. Once convulsive seizures occur it is advantageous to build up the dosage of the drug as rapidly as possible to a level sufficient for control, since the longer convulsions persist the more difficult they are to control.

REPORT TO THE COUNCIL

The Council has authorized publication of the following report from the Committee on Research, a standing committee of the Council on Pharmacy and Chemistry.

R. T. Stormont, M.D., *Secretary.*

Physicians often are disconcerted by premature or sensational announcements of medical discoveries appearing in the lay press long before any authoritative information on the subject is published in a reputable medical journal. The professional resentment aroused is frequently directed against the investigators whose names are mentioned in the news release. As a rule, the investigators are not directly responsible for the appearance of the story in the national press.

The enormous interest of the public in any item concerning medical progress results in thorough coverage of the scientific meeting of any large medical society by newspaper reporters free to attend such sessions. What the reporter considers newsworthy and interesting he prints with names, pictures, and his own interpretation of the story. This is his inherent right as expressed and guaranteed in the term "freedom of the press." Nevertheless, it would seem advisable for science writers to check over the facts contained in their stories with the investigators. The latter invariably are willing to correct errors or misinterpretations of their reports in the interest of the reading public. Undue haste in publication may result in harm to both the laity and the writer's reputation for accuracy.

Another source of news stories apparently emanates from public relations counsels employed by some pharmaceutical firms to place items concerning new drugs in the newspapers. In this the public relations counsel is merely carrying out his client's wishes. The firm, however, has recognized the commercial advantage in creating a demand by the public for a new and untried drug or for a new and scantily documented use for an older drug. By such publicity the firm apparently hopes to avoid the long but necessary period of evaluation for a new method of treatment. Following publication of these planted items, the public is quick to press the profession for employment of newly mentioned drugs or uses and shows little sympathy or understanding for the reluctance and resistance of physicians to accept such publicity as evidence of therapeutic worth. There is no denying that the wisdom of releasing some of the information disseminated in newspapers and periodicals, and on radio and television, is open to question.

The concern expressed by research investigators and practicing physicians regarding the detrimental effect of many of these press releases on physician-patient relationships indicates a need to clarify the situation for the profession and to suggest means which might aid in avoiding unpleasant premature release of information.

The following article on this subject has been reviewed and approved by the Committee on Research.

Paul L. Wermer, M.D., *Secretary,*
Committee on Research.

DOCTOR, MEET THE PRESS!

John L. Bach, Chicago

Times have changed in the field of medical science reporting since Osler uttered this caution: "Believe nothing that you see in the newspapers—they have done more to create dissatisfaction than all other agencies. If you see anything in them that you know is true, begin to doubt it at once."

The medical profession's attitude toward newspaper reporters has changed from definite coolness to warm and friendly cooperation, especially since World War I.

More doctors are meeting more reporters today than ever before with the result that there is more and more news about medicine and health in newspapers and magazines and on the radio. This reflects a strong desire by the public for knowledge of the progress of medical science. Independent newspaper and press association surveys have shown that there is more reader interest in medical news than in any other branch of science.

Director of Press Relations, American Medical Association.

Therefore, the average doctor, whether general practitioner or specialist, can expect sooner or later to meet the press. It may happen while he is busy in his office or walking up the steps of the hospital or addressing a medical meeting or entertaining socially or speaking at a luncheon club. It may happen suddenly when a prominent patient dies or when the doctor plays a part in the development of a new drug.

Many physicians like to think that they meet reporters, or highly trained science writers, on a basis of equality, with the best of good will. How the doctor handles himself to meet these extraordinary situations can spell either success or failure. While medical press relations have improved tremendously during the last few years, it is still surprising how often specific encounters between physicians and science writers result in disappointment or annoyance on one side or both.

This can be blamed on the complicated aspects of both writing and reading news of scientific discoveries in medicine.

As Dr. Alan Gregg, director of the Rockefeller Foundation, said in one of his speeches not long ago: "The issue of what a discovery may eventually mean injects a tremendous emotional strain. The possible meaning of a discovery affects the investigator, the news writer, the medical profession, the anxious patient, and the news reader—each in a different way.

"The investigator's ambition to have accomplished something of undisputed and permanent value, as well as scientific soundness, is quickened—to put it rather mildly. The news writer suffers from the temptation to play up undeniable but unlikely possibilities of the discovery. The medical profession, with a long experience both of bitterly disappointing flashes-in-the-pan and almost miraculous conquests of disease, eagerly awaits confirmation and application of the new fact. The anxious patient and his family experience the anguish of uncertainty and the all but intolerable vacillation between incredulous despair and new renewed hope. Even the question of patents and proprietary control may be involved. Everyone is disturbed."

With such a tense and complicated background to the release of news of advances in medical science, both the writer and the doctor who becomes associated with an important announcement are faced with certain moral obligations.

William M. Pinkerton, director of the Harvard News Service and a newspaperman for more than a decade, once wrote an excellent piece in the Harvard Business Review on the attitudes of the public toward the press. He said that busy executives—and that includes doctors—often complain that:

"The papers never get things right." "You can't trust reporters." "They didn't put in half of what I told them." "They missed the whole point of the story." "I didn't say that at all."

The truth is, Mr. Pinkerton said, that most of the time newspapers do get things right; you can trust most reporters; they do have the space for good news; one man's point is another man's side issue; occasionally someone is misquoted, but often the real trouble is surprise at the naked look of the spoken word in print.

Why do such situations arise? The simple fact is that most of the misunderstanding, when there is misunderstanding, arises because few physicians know how reporters work or what makes them tick.

The doctor often believes that he knows all about newspapers and can determine for himself what they should say and exactly how they should say it. In reality, newspaper writing today, whether it is medical news or the reporting of a big fire, is an exacting business—as exacting as removing a gallbladder or prescribing a drug for a pain in the stomach. It is a highly competitive business. The people who run a newspaper are as well trained for their specific jobs as the doctor is in his field. Like some physicians, many consider themselves specialists in their own writing sphere. That is why newspapers have medical science writers, financial writers, sports writers, editorial writers, and outdoor editors. Each of these has special talents which he applies in his work and, over the years, becomes recognized as a specialist in a certain field or a branch of the newspaper business.

One important point to remember is that all of these writers work for an institution or business whose freedom is guaranteed under the Constitution of the United States and all of the state constitutions. Under such guaranties, the press stands by common consent first among the organs of public opinion.

Remember, too, that a reporter never stands alone. "A reporter's stake in a story is shared by his editors," said Mr. Pinkerton. "If he is pushed around and left cooling his heels in an outer office, his grievance may well become their grievance, too. No matter how hard an editor may strive to handle the news impartially or without bias, the 'bad' news source is sometimes in danger of suffering in print for his 'acts.' And the 'good' news source is likely to benefit in the long run."

A reporter's job is to report news accurately and impartially and if he is working on a medical story, for example, he naturally turns to a doctor for the facts. He should turn to the medical profession for his information because presumably it is the best informed and should always be considered the most authoritative source. A reporter is not expected to mold the results to some pre-conceived notion—yours or his.

The facts about American medicine and the health of the American people have to be told by those who know the facts. If the reporter cannot get the facts from you, the doctor, he will go elsewhere and your chances and the chances of the medical profession, generally, of getting an accurate story before the public may be lost.

"Physicians must realize that what the public knows and how the public feels toward the profession depends largely upon where it gets its information," said C. Lincoln Williston, Manager of the University of Illinois' Office of Public Information, adding: "If physicians and medical societies do not facilitate the publicizing of the authentic information which they possess, they then must accept the blame for public ignorance and public misunderstanding.

"The fact must be recognized that, no matter what physicians and scientists might accomplish, newspapers, magazines, radio and television are going to report on subjects currently within the realm of medicine. The choice then is not between publicity and no publicity. Rather, it is the choice between authentic news reports prepared with the active cooperation of physicians on the one hand, and 'black market' publicity of questionable accuracy on the other."

Someone once said that if doctors, especially research men whose business is the accumulation of knowledge, do not accept their educational responsibilities, they will have abdicated in favor of ignorance.

The framers of the "Principles of Medical Ethics of the American Medical Association" must have had this thought in mind when they drafted the following section:

"Many people, literate and well educated, do not possess a special knowledge of medicine. Medical books and journals are not easily accessible or readily understandable.

"The medical profession considers it ethical for a physician to meet the request of a component or constituent medical society to write, act or speak for general readers or audiences. The adaptability of medical material for presentation to the public may be perceived first by publishers, motion picture producers or radio officials. These may offer to the physician opportunity to release to the public some article, exhibit or drawing. Refusal to release the material may be considered a refusal to perform a public service, yet compliance may bring the charge of self-seeking or solicitation. In such circumstances the physician should be guided by the decision of official agencies established through component and constituent medical organizations.

"A physician who desires to know whether, ethically, he may engage in a project aimed at health education of the public should request the approval of the designated officer or committee of his county medical society." In order for a physician to comply with the last statement, it is imperative that every state and county medical society have an active press relations committee, empowered to speak, which can serve effectively as the liaison between physicians and the press, magazines, radio or television.

Recognizing the importance of full cooperation between physicians and the press, many of the state and county societies have adopted a Code of Cooperation, setting forth a workable policy on medical news which would satisfy both sides.

The Colorado State Medical Society's Code, which has served as a model for Codes adopted in many of the other states, sets up a system of official spokesmen for each county society—usually the president, secretary and publicity chairman. These men and women, as well as the spokesman for the hospitals, are to make themselves available to the press and may be quoted "in matters of public interest for purposes of authenticating information." The Code states specifically that this action by the spokesmen "shall not be considered by their colleagues as a breach of the time-honored practice of physicians to avoid personal publicity, since it is done in the best interests of the public and the profession."

The Colorado Code and the Codes in effect in many of the other states are a tremendously encouraging sign that the physicians and the press are alive to the importance of giving accurate medical information to the public. The Codes, however, should be drafted cooperatively between the press and medical societies; they should never be drafted by the medical society alone.

A doctor or medical researcher, in order to make his job of meeting the press easier and, at the same time, stay within the bounds of good ethics, should call upon such a committee of his state or county society for help in working out publicity problems. In most states, the publicity committee is guided in its work by experienced public relations people, most of whom are former newspapermen. The job which these committees are doing, and have done, is of great import to the medical profession as a whole.

But the Codes in themselves do not provide all the answers to publicity problems on a local or national level. Their effectiveness rests on whether the publicity committee is an active one or a dead one. An active committee, really interested in the day-to-day problems that come up, will do a good job, while an inactive committee, one in name only, will do a poor job which will show up continually in bad public relations for the profession.

National medical organizations, especially the specialty groups, and at least the larger state societies, should by all means set up the physical mechanics for doing a good press relations job at their annual meetings. Sometimes bad publicity emanates from national meetings and this bad publicity usually can be traced to poor press relations on the part of the medical organization. The blame cannot be laid on the press alone.

The job of handling press relations at a national meeting requires, above all things, experienced personnel fully familiar with both the doctor's viewpoint and that of the press. The experienced press representative can save the doctor many a headache. Physicians who specialize more or less in research are often invited to appear on a program of a national organization. But, at times, they are almost reluctant to give information to their fellow physicians because they fear that a garbled story might appear in the newspapers, resulting in ridicule by their colleagues because of the consequent publicity. This is a pertinent point in the whole realm of medical press relations. But there is more to it than that. Even if a news story is 100% accurate, physicians will frequently deride their colleagues who figure in a story with such a jibe as: "I saw your advertisement in the paper today." Such a statement, good natured or not, makes the conscientious physician, who is trying to help the profession, quite reluctant to be a part of any future stories. This hurts medicine!

It is much easier for everybody concerned if the doctor can consult in advance with the press representative who has been empowered by the national organization to handle its press job.

In cases where no such representative is available at a national meeting, it is best for the doctor to consult or work with the public relations director of the medical society located in the city where the meeting is being held. In all probability this man is well acquainted with the newspaper contacts in that city and can be of invaluable help to the visiting doctor.

Several national specialty organizations still adhere to the time-honored custom of mimeographing in advance the full papers which doctors are scheduled to deliver at a meeting. This is a good and bad practice—depending on the way a newspaperman and a doctor look at the service from their own individual standpoints.

Some writers who devote their full time to science and medicine, prefer the full texts; the more the better. Other writers, especially those on newspapers, prefer to work from good abstracts of the papers because they do not have the time to review the full texts of all papers.

The majority of writers, however, would prefer to work from abstracts carrying interpretive scientific facts, definitions and background. "If a doctor's contribution is an important and interesting one," a prominent writer told me, "nothing takes the place of a full text." He was of the opinion that many of the inaccuracies in a story resulted because a reporter, working from an abstract only, tried to fill in the gaps himself.

For any medical organization to adopt a policy of automatically mimeographing all papers in full is the laziest way of handling a press relations job. But it does not help the physician who prepared his paper with only a medical audience in mind. The doctor may be discussing a subject based on only a handful of cases; yet his experimental research may be important enough to present to the medical meeting even though he knows it is "premature" in every sense of the word. In such a case, the physician is presenting his facts to his colleagues only and not to the public. For any organization to follow a general practice of mimeographing such a paper and passing it on to writers, without first weighing its impact on lay readers, is to do a disservice not only to the doctor who was invited to deliver the paper, but to the medical profession and the public as well.

Whether to provide a full text or an abstract only is best determined by the common sense and good judgment of an experienced person, who carries the responsibility of handling the press relations for a medical organization.

Abstracting of medical material for publicity purposes only should be the responsibility of the national or state organization if they are interested in doing a good press relations job. By having an experienced medical press writer abstract the papers in advance in newspaper phraseology, the organization is, almost automatically, assured more accuracy when the final story appears in print. By the same token, the doctor who is involved in the story often avoids embarrassment by having his facts appear in public print correctly.

A physician who is a party to an important announcement, not necessarily given at a national or state convention, should in the best interest of ethics seek the advice of the publicity committee of his local medical society well in advance. In addition, he should seek the advice and help of a press writer experienced in the medical field who can aid him immeasurably in presenting his case to the publicity committee. Then, if the committee grants its approval, the writer also can help the physician to prepare a proper press release. The extra effort which the physician exerts in advance will pay off in a more accurate story—a story he can proudly and properly endorse.

Since he usually lacks experience in preparing or presenting facts for lay consumption, a doctor should rarely prepare his own news release. This is true especially with statements pertaining to cancer, poliomyelitis, heart disease, arthritis, and asthma. The public is vitally interested in these subjects and news stories dealing with them should be carefully handled so that they do not imply that a cure has been found unless, of course, the word "cure" with all its full meaning is actually involved.

In reporting drugs for the treatment of some of these diseases, pharmaceutical firms are often censured by members of the medical profession, who charge, among other things, that the announcement is premature. The highly ethical drug firms are seldom responsible. Most of the time they leave no stone unturned in trying to keep a premature story out of print. Many times they fail because the news leaks out from a wholly unsuspected source. Then, again, there are less reputable firms which rush into print with the most flimsy reports based on few, if any, scientific facts or on articles "in press" and therefore unavailable to the physician to enable him to make an independent appraisal of the worth of the drug. These bad reports offset the really good reports.

Some physicians seem to feel that nothing should be said in the public press about a new therapeutic agent until it has been "fully proved." But when is a drug "fully proved?"

Mr. Steven M. Spencer, popular medical editor of *The Saturday Evening Post*, discussed the matter of "premature" publicity in a fairly recent article in *New York Medicine*. He said:

"We know that very few contributions represent immortal and immutable medical truths. But that does not mean they are not worthwhile contributions, capable of giving valuable aid until something better comes along. Sulfanilamide was not without its untoward side effects, and ultimately it was largely replaced by other, better chemotherapeutics. Yet in the meantime it saved many lives and prevented much suffering. Would those who charge the press with being 'premature' in its reports have denied the public information about sulfanilamide simply because it had some drawbacks?

"No informed person expects science to move forward at a steady, unchanging pace. It is inevitable that medicine may occasionally take three steps upward and then find it must back down one step to secure a firmer foothold—but it has made a net gain of two steps. Surely the public is entitled to witness this progress, no matter how irregular and interrupted it may be. It is the science writers' contention that the public should learn of new developments touching on its health just as soon as a number of qualified physicians feel that the developments have promise and that they have been *tried out sufficiently to justify continued use.* Those who are ill need all the hope and encouragement they can find, and they find a great deal in the good news of medicine's accomplishments. The disease which a patient is suffering may be incurable today, but his faith in medical science may help sustain him until the arrival of tomorrow's successful treatment."

A few physicians like to represent themselves as knowing everything there is to know about medicine, and they are quick to criticize any newspaper announcement about a drug or technic unknown to them. A moment's reflection will show that such a position is indefensible because the diversity of specialties in medicine is so great today that no one can assume he knows everything about all of them.

Consequently, to the patient inquiring about a new drug reported in the press but not yet in a medical journal, the physician might say something like this: "It's new and I hope what I read in the paper, too, is true. I haven't seen anything about the drug in the medical literature, but I'll do some checking and try to appraise it in your particular case. Please give me a little time and I'll see if it is a safe treatment for you."

The patient will appreciate the doctor's position and will respect his attempt to exercise his own personal judgment in evaluating the drug for the patient's use. There is certainly nothing embarrassing in such an explanation.

The careful reporter often qualifies his story by warning of dangers and short supply of a new substance or insufficient evidence. Physicians should look over the published story rather carefully and use such qualifications to calm the patient who calls.

Another bugaboo to an accredited medical science writer is the doctor's reticence at seeing his name in the public print. He prefers to stick by the tradition of hiding his light under the proverbial bushel labeled "Don't quote me."

Again Mr. Spencer says: "Our argument for using names is simply that we believe the public has a right to know whose work is being described and whose opinions are reflected in the text of the article. To adopt a policy of not quoting authorities would open the way for unreliable reports on medicine by irresponsible publications—and there are a few. In addition, we feel the story of medicine's advance is the story of people as well as of facts."

Some doctors who protest against the use of their names in a news story claim that such publication amounts to personal glorification and advertising and that this type of publicity can become a device whereby charlatans promote their own trade.

Many doctors would prefer to remain anonymous, but Lester Grant, former science editor for the *New York Herald Tribune* and now a medical student at Harvard, believes this is really a dangerous line of thinking. "A doctor's name in a story," he said, "is some protection for the public; at least, it's a check against careless reporting. Without having to worry about where he got his information, the reporter might be led to the following fantasy: 'A cure for cancer has been discovered, it was learned last night. This cure involves the drinking of a quart of water a day, elimination of pepper from the diet, and strict bed rest for 19 years.' If a doctor protests against the spreading of such nonsense and demands to know the source of the information, the writer might stand on his professional ethics, so called, and refuse to give the name of his informant. Such hyperbole may seem out of reach, even of some of the less responsible newspapers, but one has only to track down the sources of some of those 'reliable' reports coming from 'well informed spokesmen' to appreciate the risk of doing this sort of thing in science stories."

From the standpoint of newspapers and magazines, good pictures are just as important as the text of a story. Both usually maintain elaborate photographic departments. The photographer is on much the same level of the working newspaper ladder as the reporter. You cannot work with one and ignore the other because their assignments probably come from the same source—the editor.

What are the photographers' common complaints against doctors? Well, here are two examples of how best to "burn" a photographer. When he calls and asks to take your picture, say "yes" in a friendly, cordial way. Then, after he spends an hour getting to your office or meeting, tell him you have changed your mind because "it may be unethical." Or, if you do consent and he spends considerable time setting up his camera and lights, tell him as he starts posing you that he can only take a picture of the back of your head; side or front view shots are out. Just visualize a photographer's frustration!

This all makes for bad public relations toward the medical profession, and such problems should no longer have a place in present-day medical reporting.

When the photographer makes his original request, the physician should refer him to the chairman of the publicity committee of his local society, assuming that the committee is an active one and not serving in name alone. He should provide the photographer with the chairman's name, address, and phone number and invite him to contact the chairman, explaining the reasons for the procedure. In turn, the chairman and/or his committee should inform the photographer of their decision in a comparatively short time, preferably immediately. The less time the better. There is nothing more disconcerting to a photographer than to make a request for a picture and not have the publicity committee act for two or three days. The photographer, like the writer, has a deadline to meet and time to him on any assignment is an important consideration.

If the photographer makes his request of the doctor during a medical meeting where the physician appears on the program, it might be more expedient and feasible for the matter to be referred to the organization's public relations man or press relations contact, both of whom are fully aware of the necessary protocol.

A short time ago a physician read an interesting paper before a national specialty society. The next morning a newspaper carried a story, based on his paper, on the first page. That afternoon a national news magazine requested his picture. The doctor refused on the basis of medical ethics; actually he felt that if the picture appeared, along with the story, in a national magazine he would be held up to ridicule by his colleagues. The photographer contacted the local medical society. Again permission to take the picture was turned down without even checking or weighing the facts with the society's publicity committee. The photographer's persistence finally got the matter to the attention of the society's publicity chairman who, without apparently being acquainted with the actual interpretation of the Principles of Medical Ethics, turned down the request, too.

It would not have been unethical for the committee to grant the photographer permission to take the doctor's picture. The whole matter could have been handled in a matter of minutes. As it turned out, the photographer was given a harmful runaround by people who apparently did not want to be bothered.

Since the doctor had given his paper before an open meeting of an ethical medical society, the photographer was within his rights in requesting the picture. If he had taken the picture while the doctor was delivering his address, neither the doctor nor the medical group could have done anything about it. In refusing permission in this case, the publicity committee was responsible for doing a disservice, publicitywise, to the medical profession as well as to the news magazine.

The committee apparently was unaware of the sentence in the Principles of Medical Ethics which reads: "Refusal to release the material may be considered a refusal to perform a public service . . ."

Generally speaking, the job of handling medical press relations can be boiled down to this:

It is considered unethical for a physician to furnish a story or picture on his own initiative to newspapers or magazines, as this smacks too much of self-seeking publicity. On the other hand, if data have been or are being presented publicly in lay or scientific meetings, it is considered ethical if the physician works through the publicity committee or public relations man of his local society. In that way, his colleagues are his judges and, in abiding by their judgment, he cannot be accused of being a publicity seeker.

Investigators associated with medical schools are obligated to conform to the procedures established by their institutions for dealing with reporters and publicity through public relations officials appointed by their institution. It must be emphasized, however, that under the existing code of ethics this procedure does not relieve the investigator of the responsibility of clearing the release with the publicity committee of the local society. Should the latter rule after publication that release of the data is unethical, the physician, not the medical school's public relations council, is held responsible for violation of the code.

Emergency situations often arise in which the time relation between the story already presented at a meeting and its "going to press" make such referrals impossible. In such a case, where decision on publication is no longer in the hands of the physician, he should still make every effort to assure accuracy of the data contained in the news release by working in conjunction with the publicity committee of his county society.

NEW AND NONOFFICIAL REMEDIES

The following additional preparations have been accepted as conforming to the rules of the Council on Pharmacy and Chemistry of the American Medical Association for admission to New and Nonofficial Remedies. A copy of the rules on which the Council bases its action will be sent on application.

R. T. Stormont, M.D., *Secretary.*

Estrone-U.S.P. (See New and Nonofficial Remedies 1952, p. 332).

Kremers-Urban Company, Milwaukee

Solution Estrugenone in Oil: 1 cc. ampuls and 10 cc. and 30 cc. vials. A solution in sesame oil containing 1 mg. (10,000 I.U.) of estrone in each cubic centimeter. Preserved with 0.5% chlorobutanol.

10 cc. vials. A solution in sesame oil containing 2 mg. (20,000 I.U.) of estrone in each cubic centimeter. Preserved with 0.5% chlorobutanol.

Suspension Estrugenone with Benzyl Alcohol 2%: 1 cc. ampuls and 5 cc. and 10 cc. vials. A suspension in 60% propylene glycol containing 5 mg. (50,000 I.U.) of estrone in each cubic centimeter.

Suspension Estrugenone with Procaine Hydrochloride 1%: 1 cc. ampuls and 10 cc. vials. A suspension in 15% propylene glycol containing 2 mg. (20,000 I.U.) of estrone in each cubic centimeter. Preserved with thimerosal 1:10,000.

Progesterone-U.S.P. (See New and Nonofficial Remedies 1952, p. 348).

The Bio-Intrasol Laboratories, Inc., Brooklyn

Aqueous Suspension Progesterone with Procaine Hydrochloride 1%: 10 cc. vials. An aqueous suspension containing 10 mg. or 25 mg. of progesterone in each cubic centimeter. Preserved with thimerosal 1:10,000.

Solution Progesterone in Oil with Benzyl Alcohol 2%: 10 cc. vials. An aqueous suspension containing 10 mg. or 25 mg. of progesterone in each cubic centimeter. Preserved with thimerosal 1:10,000.

The Upjohn Company, Kalamazoo, Mich.

Aqueous Suspension Progesterone: 1 cc. and 10 cc. vials. A suspension in isotonic salt solution containing 25 mg. of progesterone in each cubic centimeter. Preserved with thimerosal 1:10,000.

Mephenesin (See New and Nonofficial Remedies 1952, p. 432).

C. B. Kendall Company, Indianapolis

Tablets Mephenesin: 0.25 gm. and 0.5 gm.

Diphtheria Toxoid, Aluminum Phosphate Adsorbed.—Aluminum phosphate adsorbed diphtheria toxoid is a sterile suspension of diphtheria toxoid adsorbed on aluminum phosphate. It is detoxified and standardized for potency as described in the monograph on Diphtheria Toxoid, Aluminum Hydroxide Adsorbed. Diphtheria Toxoid, Aluminum Phosphate Adsorbed, complies with the official potency and other requirements of the National Institutes of Health of the United States Public Health Service.

Actions and Uses.—See monograph on Diphtheria Toxoid, Alum Precipitated. Because of the physical character of the adsorbed product, absorption is delayed.

Dosage.—See monograph on Diphtheria Toxoid, Alum Precipitated.

Parke, Davis and Company, Detroit

Diphtheria Toxoid, Aluminum Phosphate Adsorbed: 0.5 cc. vials (one injection), 1 cc. vials (one immunization; two 0.5 cc. injections), 5 cc. vials (five immunizations). Preserved with thimerosal 1:10,000.

Penicillin for Injection for Prolonged Action (See New and Nonofficial Remedies 1952, p. 126).

Irwin, Neisler & Company, Decatur, Ill.

Aqueous Suspension Procaine Penicillin G: 10 cc. vials. A suspension containing 300,000 units of procaine penicillin G in each cubic centimeter. Preserved with 0.18% methylparaben and 0.02% propylparaben.

Procaine Penicillin G in Oil: 10 cc. vials. A suspension in sesame oil containing 300,000 units of procaine penicillin G in each cubic centimeter with 2% aluminum monostearate.

Thiopental Sodium-U.S.P. (See New and Nonofficial Remedies 1952, p. 246).

Abbott Laboratories, North Chicago, Ill.

Pentothal Sodium: 10 gm. multiple dose ampuls. Buffered with 0.6 gm. of anhydrous sodium carbonate.

Penicillin for Oral or Sublingual Administration (See New and Nonofficial Remedies 1952, p. 122).

Premo Pharmaceutical Laboratories, Inc., South Hackensack, N. J.

Tablets Penicillin G (Buffered): 200,000 units. Buffered with calcium carbonate.

Citrated Normal Human Plasma-U.S.P. (See New and Nonofficial Remedies 1952, p. 200).

Junior League Blood Center of Milwaukee, Inc., Milwaukee

Normal Human Plasma (Citrated): Equivalent to 250 cc. of pooled plasma containing 5% dextrose.

***p*-Aminosalicylic Acid** (See New and Nonofficial Remedies 1952, p. 87).

Gold Leaf Pharmacal Company, New Rochelle, N. Y.

Tablets Para-Pas: 0.5 gm.

Estrogenic Substances—Conjugated (See New and Nonofficial Remedies 1952, p. 338).

Eli Lilly & Company, Indianapolis

Tablets Konogen: 0.625 mg., 1.25 mg., and 2.5 mg. U. S. Patent 2,429,398.

Penicillin for Inhalation (See New and Nonofficial Remedies 1952, p. 121).

Abbott Laboratories, North Chicago, Ill.

Soluble Tablets Potassium Penicillin G: 250,000 units.

Diiodohydroxyquinoline-U.S.P. (See New and Nonofficial Remedies 1952, p. 154).

G. D. Searle & Company, Chicago

Tablets Diodoquin: 0.65 gm. U. S. Trademark 336,484.

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METABOLISM OF FRUCTOSE IN THE DIABETIC

It has been known for some time that the administration of glucose to dogs or normal human subjects leads to an increase in the pyruvate concentration and a decrease in the level of inorganic phosphate in the blood. These changes also occur when a diabetic patient receiving insulin is given glucose. The increase in the blood pyruvate, coupled with a decrease in inorganic phosphate content, has been used as an index of the metabolism of administered sugar.

Recently, a series of studies[1] on the metabolism of intravenously administered fructose in normal and diabetic subjects has been reported. The changes in the blood concentration of fructose, pyruvate, and inorganic phosphate following an intravenous injection of fructose indicates to these investigators that the metabolism of fructose is different from that of glucose in both normal subjects and diabetic subjects without insulin. After giving fructose to either group there is an increase in pyruvate concentration in the blood with a concurrent decrease in inorganic phosphate level. While the intravenous administration of fructose to a diabetic subject without insulin invokes a large and rapid increase in the pyruvate concentration in the blood, glucose given in a similar manner causes either a slight or a delayed increase in pyruvate. This was interpreted as indicating that the diabetic subject without insulin can metabolize fructose given intravenously in a manner similar to a normal person.

By using the technique of hepatic vein catheterization, Craig and associates[2] studied the metabolism of fructose in the liver of diabetic and nondiabetic persons. Following the intravenous administration of fructose, they found that the livers of both groups of subjects removed large amounts of fructose from the blood. Also, livers of the diabetic and nondiabetic subjects produced increased amounts of pyruvic acid after the injection of fructose. In the absence of ketosis, the liver of the diabetic subjects was found to metabolize fructose in a manner similar to that in the nondiabetic person; however, when ketosis occurred there was a normal uptake of fructose but a decreased production of pyruvic and lactic acids.

The investigations on the metabolic aberration in diabetes have yielded promising information in recent years, particularly since the Embden-Meyerhof scheme for the anaerobic breakdown of glucose has been widely accepted. Since fructose normally enters the scheme of glycolysis subsequent to the point at which glucose is phosphorylated, these studies on the metabolism of fructose are consistent with the concept that the phosphorylation of glucose to glucose-6-phosphate is impaired in the diabetic. The phosphorylation of fructose, on the other hand, appears to proceed at its normal rate in the diabetic without insulin.

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[1] Miller, M., and others: Metabolism of Intravenous Fructose and Glucose in Normal and Diabetic Subjects, J. Clin. Investigation 31: 115-125, 1952.

[2] Craig, J. W., and others: Metabolism of Fructose by the Liver of a Diabetic and Non-Diabetic Subjects, Proc. Soc. Exper. Biol. & Med. 78: 698-702, 1951.

BLOOD TRANSFUSION IN IRRADIATION HEMORRHAGE

The threat of atomic warfare and the ever-increasing peacetime exploitation of atomic energy have focused attention on the necessity for developing methods for the control or prevention of irradiation injury in man. The mortality in animals exposed to high levels of irradiation has been reduced by the administration of the broad spectrum antibiotics,[1] by early exchange transfusion,[2] by parabiosis,[3] or by the implantation of fresh splenic tissue or the injection of spleen homogenates.[4] In contrast, while the use of frequent whole blood transfusions would seem a logical method of combating the thrombocytopenia and anemia in the abnormal bleeding syndrome of irradiation sickness, Allen and his associates have shown that in dogs such transfusions not only are ineffective but may in some instances actually be harmful.[5] The University of Chicago investigators exposed 173 dogs to single doses of total body x-radiation at dosage levels ranging from 175 to 450 r and compared the survival times of control animals and of animals given three transfusions a week with compatible blood. They found that the animals that received transfusions survived no longer than the controls and at autopsy displayed as many areas of gross hemorrhage. Furthermore, while blood transfusion did benefit to some extent the irradiation anemia, it did not correct the thrombocytopenia or leukopenia and failed to prevent or control the postirradiation increase in whole blood clotting time. In some animals, the transfusions appeared to exert a deleterious effect, possibly owing to transfusion reactions, which, according to the authors, occur much more frequently in irradiated than in nonirradiated dogs.

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[1] Miller, C. P.; Hammond, C. W., and Tompkins, M.: Reduction of Mortality from X-Radiation by Treatment with Antibiotics, Science 111: 719 (June 30) 1950. Allen, J. G.; Moulder, P. V., and Emerson, D. M.: Pathogenesis and Treatment of the Postirradiation Syndrome, J. A. M. A. 145: 704 (March 10) 1951. Furth, F. W.; Coulter, M. P., and Howland, J. W.: The Effect of Aureomycin on the Radiation Syndrome in Dogs, Am. J. Path. 28: 25 (Jan.-Feb.) 1952; The Effect of Aureomycin and Terramycin on the X-Radiated Rat, Am. J. Path. 28: 185 (March-April) 1952.

[2] Swisher, S. N., and Furth, F. W.: Effect of Early Small Exchange Transfusion on the X-Irradiated Dog, Proc. Soc. Exper. Biol. & Med. 78: 226 (Oct.) 1951.

[3] Brecher, G., and Cronkite, E. P.: Post-Radiation Parabiosis and Survival in Rats, Proc. Soc. Exper. Biol. & Med. 77: 292 (June) 1951.

[4] Jacobson, L. O., and others: Further Studies on Recovery from Radiation Injury, J. Lab. & Clin. Med. 37: 683 (May) 1951. Cole, L. J.; Fishler, M. C.; Ellis, M. E., and Bond, V. P.: Protection of Mice Against X-Irradiation by Spleen Homogenates Administered After Exposure, Proc. Soc. Exper. Biol. & Med. 80: 112 (May) 1952.

[5] Allen, J. G., and others: Blood Transfusion in Irradiation Hemorrhage, Science 115: 523 (May 8) 1952.

Allen and his co-workers point out that, since the status of blood types and transfusions in dogs is as yet ill-defined, the failure of frequent blood transfusions to prolong the survival time of irradiated dogs does not necessarily mean that such measures would be ineffective in man. Furthermore, they emphasize that their results do not relate in any manner to the use of blood in the treatment of shock incident to the early blast effects of an atomic burst or to blood needs in anoxic anemia. On the other hand, they feel that on the basis of their results a more cautious attitude toward the use of frequent transfusions alone as a therapeutic measure in the treatment of the latent symptoms of irradiation in man may be indicated.

MOVIE PORTRAYS DOCTOR AT WORK

RKO-Radio Pictures has just released a 15 minute documentary film called "Your Doctor" to commercial theaters throughout the country. The film portrays a story that is one of the finest tributes ever paid to the medical profession.

The picture, which was prepared with the assistance of the American Medical Association, reveals the high quality and ready availability of medical care in this country. In cities and towns American physicians may be virtually around the corner, but in the mountains of Hickory Nut Valley, N. C., the work of a 37-year-old general practitioner, George Bond, exemplifies what doctors are doing in hundreds of rural communities. The story behind the movie is built around Dr. Bond, who has realized a life-long ambition—the establishment of the Valley Clinic and Hospital, one of many institutions supplying the best in medical care to remote areas.

The movie ends with this conclusion: "In the lives of men and women who practice the art and science of medicine is realized the noblest of human aspirations. For long ago it was said that in no other act does man approach so near the gods as when he is restoring the sick to the blessing of health."

RKO, which is trying to book "Your Doctor" in as many local theaters as possible, estimates that the film will be shown during 1952 in about 3,000 theaters, reaching an audience of 10 to 15 million persons. However, that audience can be doubled if physicians throughout the country help publicize local showings. County and state medical societies are being asked to cooperate in any way possible with theaters showing "Your Doctor." If the film is being shown in your community, urge your patients and friends to see it. The theater manager will suggest other ways in which you can help.

"Your Doctor" is an inspiring story for the lay public, and, as a result, the medical profession will benefit immeasurably if a large segment of the U. S. population sees it. The picture dispels many misconceptions about doctors; for that reason it is imperative that everything possible be done to help local theaters build a sizable audience.

NUTRITION AND THE OLDER ADULT

As time passes and the life expectancy of our population slowly increases, some apprehension appears with respect to the physiological well-being of the older adults. This group is not characterized by age in years alone but rather by a discernible decline in physical vigor and activity. Because of the lack of precise evaluation of the problem, the nutrition during later years of life has attracted considerable attention, and one recent comment[1] presents a well-considered point of view. The general principles governing the planning of a diet for the aged are not different from those for other age groups. The subjects themselves often plan their dietary program in the pattern of their youth, a period before the foundations of good nutrition were firmly established. Furthermore, because there is diminished physical activity, less food is required, and interest in food lags when mastication is difficult or digestive activity is retarded. Chronic disease, combined with economic and psychological factors, operates to develop an over-simplified dietary program, one that is too frequently deficient in protective foods.

In the light of a decreased need for energy, the daily caloric value of the diet often can be surprisingly low; older women frequently are in nitrogen balance and maintain weight on 1,500 calories or less, and men maintain weight on 2,000 calories or less. Certainly, the commoner tendency toward obesity is to be decried. There is little evidence for the need for increased consumption of protein in the elderly. Because of the tendency toward a relationship between serum cholesterol and atherosclerosis and because of the parallelism between dietary fat and the level of serum cholesterol, it appears prudent to decrease the proportion of fat in the diet in the later years of life. While there is little evidence that older adults have different requirements for vitamins, it does seem wise to allow generous amounts of iron and of calcium in the diet of the aged.

In the aged, it is quite likely that under conditions of restricted activity and the associated diminished food intake the resultant intestinal residue will not stimulate the one-a-day bowel movement that has become the popular goal in this respect. In no other connection does habit and mental attitude play a larger part than with defecation. The same program of education that will protect the older person from the alluring claims of the food-faddist should reassure him that the natural roughage in the fruit, leafy vegetables, and whole grain cereals of a good diet will promote satisfactory intestinal evacuation. All the aspects of nutrition in the later years of life merit continued study.

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1. Keys, A.: Nutrition for the Later Years of Life, Pub. Health Rep. 67: 484 (May) 1952.

PENNSYLVANIA

Society News.—Effective Sept. 6 the name of the *Pittsburgh Medical Bulletin* will be changed to the *Bulletin of the Allegheny County Medical Society.*——The Pennsylvania Academy of General Practice recently installed as president Dr. Anthony T. Merski of Erie.

Hospital Retirements.—Twelve staff members, with a total of 428 years of hospital service, have retired from the medical and surgical staffs of Harrisburg Hospital. They will, however, remain as consultants of the hospital and will continue their private practice. They are Drs. Harvey F. Smith, aged 81, with 52 years of hospital service; George F. Gracey, 78; George H. Seaks, 75; George R. Moffitt, 73; Park A. Deckard, 71; Carson Coover, 70; Norman B. Shepler, 69; Joseph W. Shaffer, 69; Harry M. Kirkpatrick, D.D.S., 68; Allen Z. Ritzman, 67; Frank F. D. Reckord, 66; and George L. Laverty, 65.

Philadelphia

University News.—Dr. Victor C. Vaughan III, formerly assistant professor of pediatrics in the Yale University School of Medicine, New Haven, Conn., has been appointed associate professor of pediatrics in the Temple University School of Medicine.——Dr. John B. Levan, chief of the cardiology department of St. Joseph's Hospital, Reading, has been named as assistant professor of medicine at the Woman's Medical College in Philadelphia. Dr. Levan is president of the Pennsylvania Heart Association and has been supervising graduate courses in cardiology in 10 cities for the Medical Society of the State of Pennsylvania.

Pittsburgh

Society News.—The Pittsburgh Urological Association has elected the following officers for the coming year: Dr. Joseph G. Moore, president; Dr. William C. D. McCuskey, Wheeling, W. Va., president-elect; and Dr. Conrad A. Kuehn, secretary-treasurer.

TEXAS

Inaugurate Preceptor Plan.—Texas physicians are putting into practice a plan for placing senior medical students of the University of Texas Medical Branch in Galveston, in the homes of family doctors for 11 weeks. They will accompany their preceptor on hospital rounds, in the operating room, during office interviews, on house calls, and during emergency visits. All preceptors are members of the Texas Academy of General Practice.

Controlled Polio Experiment.—A 10 day controlled experiment was instituted on July 2 on 35,000 children, aged 1 to 6, half of whom received injections of gamma globulin. Of the eight clinics set up in Harris County to handle the experiment, five are in Houston. The $500,000 project, which is being conducted by Dr. William McD. Hammon, professor of epidemiology at the University of Pittsburgh, assisted by Dr. Lewis L. Coriell, medical director of the Camden (N. J.) Municipal Hospital for Contagious Diseases, and Helen Moore of the Detroit Department of Health, is financed by the National Foundation for Infantile Paralysis. Similar experiments were undertaken in Utah last summer with 5,768 children. According to the U. S. Public Health Service, for the week ended June 28 there were 206 cases of polio in Texas, as compared with 62 for the week ended June 30 last year.

HAWAII

American of the Week.—Dr. Robert B. Faus, Honolulu, chairman of the advisory council to the governor of civil defense, was recently selected as "American of the Week" by the American Way Committee of the Honolulu Chamber of Commerce.

Dr. Holmes Lectures in Orient.—Dr. William J. Holmes of Honolulu has returned from a tour of the Far East and India. He was guest speaker at the annual All India Ophthalmologic Conference, and in New Delhi and Bombay spoke before the local ophthalmologic societies; at Vellore he served as visiting professor of ophthalmology at the Christian Medical College. In Japan Dr. Holmes served as a special ophthalmic consultant to the Surgeon General.

GENERAL

Narcotics Bibliography.—A comprehensive list of books and journal articles dealing with drug addiction, compiled by the library of the New York Academy of Medicine, is being issued jointly by the academy and the Welfare Council of New York City. It is available from the council, 44 E. 23rd St., New York 10, for 50 cents.

Society Election.—At the annual meeting of the American Association for Cancer Research in Detroit, Dr. Stanley P. Reimann, director of the Institute for Cancer Research, Philadelphia, was elected president; Dr. Harold P. Rusch, professor of oncology, University of Wisconsin Medical School, Madison, vice-president; and Hugh J. Creech, Ph.D., of the Institute for Cancer Research, Philadelphia, secretary-treasurer.

Trudeau Society Desires Papers.—The American Trudeau Society, medical section of the National Tuberculosis Association, invites the submission of abstracts of scientific papers for representation at its next annual meeting in Los Angeles, May 18-22, 1953, at the Hotel Statler. For information write to the Chairman of the Medical Sessions Committee, American Trudeau Society, 1790 Broadway, New York 19.

Prizes for Cancer Research.—At its annual meeting the American Association for Cancer Research presented Anna Fuller Memorial Prizes "for outstanding and original research on the role of viruses in cancer" to Dr. F. Peyton Rous, Rockefeller Institute for Medical Research, New York; Dr. Francisco Duran-Reynals, Yale University School of Medicine, New Haven, Conn.; and Dr. Richard E. Shope, Merck Institute for Therapeutic Research, Rahway, N. J.

Ida Scudder International Grant.—The American Association of University Women announces the Ida S. Scudder International Grant of $500, a gift of the Scudder Association, to help a woman physician from India to study in the United States. Dr. Scudder, who in 1918 founded the Vellore Christian Medical School for women, celebrated her golden jubilee of medical work in India in 1950. The school, now affiliated with the University of Madras, has recently admitted men students.

Lecturers Wanted for Foreign Countries.—Through its secretary general, Dr. Louis H. Bauer of Hempstead, N. Y., President of the American Medical Association, the World Medical Association requests national medical associations to notify the secretariat when well-qualified physicians are planning visits to foreign countries and desire to give lectures in the countries they are visiting. Such information should be sent to Dr. Louis H. Bauer, Secretary General, The World Medical Association, 2 E. 103rd St., New York 29.

New Health Chief for Virgin Islands.—Gov. Morris F. de Castro recently announced the appointment of Dr. Roy A. Anduze as commissioner of health for the Virgin Islands and as chief municipal physician for the municipality of St. Thomas and St. John. Dr. Anduze served his internship at Freedman's Hospital, Washington, D. C., where he was assistant resident in gynecology and obstetrics. He was appointed to the staff of Fredericksted Hospital as municipal physician of St. Croix in 1940, and was made director of the venereal disease control program at St. Thomas in 1942.

Society News.—At the annual meeting of the American Pediatric Society at Old Point Comfort, Va., May 6-8, Dr. Irvine McQuarrie, Minneapolis, was elected president; Dr. Ernest J. Caulfield, Hartford, Conn., vice-president; Dr. Aims C. McGuinness, Philadelphia, secretary-treasurer; Dr. Stewart H. Clifford, Brookline, Mass., recorder; and Dr. Henry G. Poncher, Chicago, member of the council. Dr. Richard W. B. Ellis, Edinburgh, Scotland, and Sir James Spence, Newcastle upon Tyne, England, were elected honorary members of the society. The John Howland Award for distinguished service to pediatrics was bestowed on Dr. Edwards A. Park, Baltimore.

Elizabeth Blackwell Awards.—Five American women physicians have received the annual Blackwell awards of the New York Infirmary for significant contributions to the practice and teaching of medicine. The citations were bestowed on Ethel C.

Dunham, adjutant professor of pediatrics, Georgetown University School of Medicine, Washington, D. C., for study and improved care of premature infants; Edith L. Potter, assistant professor of pathology (obstetrics and gynecology), University of Chicago School of Medicine, for her work in infant pathology; Annie V. Scott of the American Presbyterian Mission, Cheeloo University School of Medicine, Tsinan, Shantung, China, for the practice and teaching of pediatrics in foreign missions; Martha M. Eliot, chief of the Children's Bureau, U. S. Dept. of Labor, Washington, D. C., for her contributions to child welfare and international public health; and to Catharine Macfarlane, research professor of gynecology, Woman's Medical College of Pennsylvania, Philadelphia, for achievements in the practice of gynecology, in cancer research, and in medical education.

Prevalence of Poliomyelitis.—According to the National Office of Vital Statistics, the following number of reported cases of poliomyelitis occurred in the United States, its territories and possessions in the weeks ended as indicated:

Area	June 28, 1952	June 21, 1952	June 30, 1951
New England States			
Maine	3	..	..
New Hampshire	..	1	..
Vermont	1	..	..
Massachusetts	1	1	7
Rhode Island	..	..	..
Connecticut	2	..	5
Middle Atlantic States			
New York	21	15	17
New Jersey	3	1	3
Pennsylvania	4	1	3
East North Central States			
Ohio	22	18	3
Indiana	9	6	4
Illinois	8	6	17
Michigan	13	4	9
Wisconsin	10	6	10
West North Central States			
Minnesota	2	3	2
Iowa	20	16	2
Missouri	5	3	..
North Dakota	1	1	5
South Dakota	..	..	..
Nebraska	2	5	3
Kansas	19	3	6
South Atlantic States			
Delaware	..	..	3
Maryland	..	..	1
District of Columbia	1	..	1
Virginia	..	5	3
West Virginia	6	5	2
North Carolina	9	9	5
South Carolina	1	1	1
Georgia	16	7	13
Florida	10	12	15
East South Central States			
Kentucky	4	3	2
Tennessee	9	2	5
Alabama	8	8	14
Mississippi	26	21	8
West South Central States			
Arkansas	9	4	9
Louisiana	36	17	22
Oklahoma	22	4	13
Texas	206	146	62
Mountain States			
Montana	5	7	..
Idaho	..	1	..
Wyoming	2	2	..
Colorado	5	2	2
New Mexico	6	2	5
Arizona	2	6	8
Utah	..	..	..
Nevada	..	..	..
Pacific States			
Washington	14	12	4
Oregon	2	2	2
California	39	63	48
Territories and Possessions			
Alaska	..	..	2
Hawaii	6	6	..
Puerto Rico	..	..	..
Total	590	427	346

CANADA

Cerebral Palsy Clinic.—The Edmonton Cerebral Palsy Association recently opened a clinic in Edmonton, Alberta, where children can be taken for therapy as well as school work. The clinic represents the cooperative efforts of the public, who contributed funds for its establishment; the Alberta Health Department, which supplied the staff and much of the equipment; an Edmonton lumber company, which constructed and donated special chairs; an Edmonton club, which provided a bus to transport the children to and from their homes; and city firemen who volunteered as drivers.

FOREIGN

Anti-Tuberculosis Campaign in Arabia.—A BCG vaccination campaign against tuberculosis in Aden, Arabia, has been successfully carried out by the United Nations International Children's Emergency Fund and the World Health Organization with the cooperation of the government, according to a report submitted to WHO's regional office in Alexandria. The first phase of the campaign, directed by international personnel, has been completed. Between January and May more than 31,000 children were tested for tuberculosis and 8,000 vaccinated, and the international team trained national personnel to carry on BCG work. The campaign is now being conducted by local health authorities with the aid of supplies and equipment that will be provided for another year.

Penicillin Treatment for Venereal Disease in Ethiopia.—Penicillin treatments for syphilis were scheduled to be introduced in Addis Ababa in July by the Ethiopian Ministry of Public Health and the World Health Organization. The venereal disease control project will include serologic testing of the inhabitants of Addis Ababa and will demonstrate the efficacy of penicillin in curing syphilis and other venereal diseases. A fully equipped WHO team of three experts will be sent to work with an Ethiopian team under P. Serra, chief of the venereal disease center at Addis Ababa. Facilities for the work will be provided by the government at the Filoha and Tekla Haimanot Clinics. Arrangements have been made to conduct blood tests at the laboratories of the Pasteur Institute, formerly the Imperial Ethiopian Medical Research Institute of Addis Ababa. In addition to blood testing and treatment the project will include investigation of the distribution of venereal disease throughout the country.

EXAMINATIONS AND LICENSURE

BOARDS OF MEDICAL EXAMINERS

ALASKA:* *Examination.* Juneau. On application in other towns where there are board members. *Reciprocity.* On application. Sec., Dr. W. M. Whitehead, Box 140, Juneau.

CALIFORNIA: *Written.* Los Angeles, Aug. 18-21; Sacramento, Oct. 20-23. Application must be received at least two weeks prior to date of examination. *Oral for Reciprocity Applicants.* Los Angeles, Aug. 16; San Francisco, Nov. 8. Applicants are requested not to arrange to come to an oral examination until they receive notice from the Credentials Committee. *Oral and Clinical for Foreign Medical School Graduates.* Los Angeles, Aug. 17; San Francisco, Nov. 9. Sec., Dr. Frederick N. Scatena, 1020 N St., Sacramento.

GUAM: The Commission on Licensure will meet whenever a candidate appears or submits his credentials. Ex. Sec., Dr. Austin W. Matthis, Agana.

ILLINOIS: *Examination.* Chicago, Sept. 23-25. Supt. of Regis., Mr. Charles F. Kervin, Capitol Bldg., Springfield.

MISSOURI: *Reciprocity.* Jefferson City, July 27. Ex. Sec., Mr. John A. Hailey, Box 4, State Capitol Bldg., Jefferson City.

MONTANA: Helena, Oct. 6-8. Sec., Dr. S. A. Cooney, 214 Power Block, Helena.

NEVADA:* Carson City, Aug. 4. Sec., Dr. George H. Ross, 112 Curry St., Carson City.

NEW HAMPSHIRE: Concord, Sept. 10. Sec., Dr. John S. Wheeler, 107 State House, Concord.

NORTH CAROLINA: *Reciprocity.* Raleigh, Aug. 2. Sec., Dr. Joseph J. Combs, 419 Professional Bldg., Raleigh.

Oregon:* *Examination.* Portland, July 7-9. *Reciprocity.* Portland, July 25-26. Final date for filing application was June 16. Exec. Sec., Mr. Howard I. Bobbitt, 609 Failing Bldg., Portland.

Texas: *Reciprocity.* Fort Worth, Aug. 3. Sec., Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth.

Wyoming: Cheyenne, Oct. 6. Sec., Dr. Franklin D. Yoder, State Office Bldg., Cheyenne.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

Alaska: *Examination.* On application. Juneau or other towns in Territory as decided by Board. *Reciprocity.* On application. Sec., Dr. C. Earl Albrecht, Box 1931, Juneau.

Colorado: *Examination.* Denver, Sept. 10-11. Sec., Dr. Esther B. Starks, 1459 Ogden St., Denver 18.

District of Columbia: *Examination.* Washington, Oct. 20-21. Sec., Dr. Daniel L. Seckinger, 4130 E. Municipal Bldg., Washington.

Michigan: *Examination.* Detroit and Ann Arbor, Oct. 10-11. Sec., Mrs. Anne Baker, 423 W. Michigan Ave., Lansing.

Nebraska: *Examination.* Omaha, Oct. 7-8. Director, Bureau of Examining Boards, Mr. Husted K. Watson, 1009 State Capitol Bldg., Lincoln.

Oklahoma: *Examination.* Oklahoma City, Sept. 13. Sec., Dr. Clinton Gallaher, 813 Braniff Bldg., Oklahoma City.

Oregon: *Examination.* Portland, Sept. 6 and Dec. 6. Sec., Dr. Charles D. Byrne, State Board of Higher Education, University of Oregon, Eugene.

Rhode Island: *Examination.* Providence, Aug. 13. Administrator of Professional Regulations, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

Texas: *Examination.* Austin, October. Sec., Brother Raphael Wilson, 407 Perry-Brooks Bldg., Austin.

Wisconsin: *Examination.* Madison, Sept. 13, Milwaukee, Dec. 6. Sec., Mr. W. H. Barber, 621 Ransom St., Ripon.

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* Basic Science Certificate required.

MEETINGS

Alaska Territorial Medical Association, Anchorage, Aug. 21-23. Dr. William P. Blanton, P. O. Box 2569, Juneau, Secretary.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, The Homestead, Hot Springs, Va., Sept. 4-6. Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Texas, Secretary.

American Congress of Physical Medicine, Roosevelt Hotel, New York, Aug. 25-29. Dr. Frances Baker, One Tilton Ave., San Mateo, Calif., Secretary.

American Physiological Society, New Orleans, Sept. 4-6. Dr. E. F. Adolph, University of Rochester School of Medicine and Dentistry, Rochester, N. Y., Secretary.

Biological Photographic Association, Hotel New Yorker, New York, Sept. 10-12. Mr. Lloyd E. Varden, Pavelle Color, Inc., 533 W. 57th St., New York 19, Secretary.

Medical Society of Delaware, Rehoboth, Sept. 8-10. Dr. Andrew M. Gehret, 822 North American Bldg., Wilmington, Secretary.

National Medical Association, Chicago, Aug. 11-15. Dr. John T. Givens, 1108 Church St., Norfolk 10, Va., General Secretary.

Nevada State Medical Association, Reno, Aug. 21-23. Dr. William A. O'Brien III, 505 Chestnut St., Reno, Secretary.

New Hampshire Medical Society, Mt. Washington Hotel, Bretton Woods, Sept. 7-9. Dr. Deering G. Smith, 44 Chester St., Nashua, Secretary.

Post Graduate Medical Assembly of South Texas, Shamrock Hotel, Houston, July 21-23. Dr. C. A. Dwyer, 229 Medical Arts Bldg., Houston, Secretary.

Reno Surgical Society Conference, Reno, Nevada, Aug. 21-23. Dr. Olin C. Moulton, 130 N. Virginia St., Reno, Nevada, Chairman.

Rocky Mountain Radiological Society, Denver, Aug. 7-9. Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Neb., Secretary.

U. S. Chapter, International College of Surgeons, Conrad Hilton Hotel, Chicago, Sept. 2-5. Dr. Arnold S. Jackson, 1516 Lake Shore Drive, Chicago, Executive Secretary.

Utah State Medical Association, Salt Lake City, Sept. 4-6. Dr. T. C. Weggeland, 42 S. Fifth East St., Salt Lake City, Secretary.

Vermont State Medical Society, Mt. Washington Hotel, Bretton Woods, N. H., Sept. 7-9. Dr. James P. Hammond, 128 Merchants Row, Rutland, Secretary.

Washington State Medical Association, Olympic Hotel, Seattle, Sept. 13-17. Dr. Bruce Zimmerman, 338 White-Henry-Stuart Bldg., Seattle, Secretary.

West Virginia State Medical Association, The Greenbrier, White Sulphur Springs, July 24-26. Mr. Charles Lively, P. O. Box 1031, Charleston, Executive Secretary.

INTERNATIONAL

American Congress of Industrial Medicine, Rio de Janeiro, Brazil, Sept. 20-28. General Secretarial Office: Calle Arenales No. 981 Buenos Aires, Argentina.

Australasian Medical Congress, Melbourne Victoria, Australia, Aug. 22-30. Dr. C. H. Dickson, Medical Society Hall, 426 Albert St., East Melbourne, Victoria, Australia, General Secretary.

European Congress of Cardiology, University of London, Bloomsbury, W.C.1, England, Sept. 10-12. Dr. K. Shirley Smith, 35 Wimpole St., London, W.1, England, Secretary.

European Society of Cardiovascular Surgery, Strasbourg, France, Oct. 3-4. Sir James Learmonth, Department of Surgery, University New Buildings, Edinburgh 8, Scotland.

Interamerican Cardiological Congress, Buenos Aires, Argentina, Aug. 31-Sept. 7. Dr. Blas Moia, Larrea 1132, Buenos Aires, Argentina, Secretary-General.

Inter-American Congress of Public Health, Havana, Cuba, Sept. 27-Oct. 1. Pan-American Sanitary Bureau, 2001 Connecticut Ave., Washington, D. C.

Inter-American Congress of Radiology, Mexico City, Mexico, Nov. 2-8. Dr. Guido Torres Martinez, Marsella No. 11, Mexico, D.F., Secretary-General.

International Congress of Anthropology and Ethnology, Vienna, Austria, Sept. 1-8. For information write: Secretaries, Royal Anthropological Institute, 52 Upper Bedford Place, London, W.C.1, England.

International Congress of Biochemistry, Paris, France, July 21-27. Prof. Jean Courtois, 4, Avenue de l'Observatoire, Paris 6^{e}, France, Secretary.

International Congress of Dermatology and Syphilology, London, England, July 21-26. Dr. Gordon B. Mitchell-Heggs, 5, Lisle St., London, W.C.2, England, General Secretary.

International Congress on Diseases of the Chest, Rio de Janeiro, Brazil, Aug. 24-30. Mr. Murray Kornfeld, 112 East Chestnut St., Chicago 11, Illinois, Executive Secretary.

International Congress of Haematology, Mar del Plata, Argentina, Sept. 21-26. Dr. Carlos Reussi, Anchorena 1710, Buenos Aires, Argentina, Secretary.

International Congress of the History of Medicine, Nice-Cannes, France, and Monaco, Sept. 8-14. Dr. F. A. Sondervorst, 124 Avenue des Allies, Louvain, Belgium, Secretary-General.

International Congress on Hydatid Disease, Santiago, Chile, Nov. 21-24. Organizing Committee, P. O. Box 9183, Santiago, Chile.

International Congress of Internal Medicine, Friends House, London, N.W.1, Eng. Sept. 15-18. Sir Harold Boldero, 12 Pall Mall East, London, S.W.1, England, Secretary.

International Congress of Medical Press, Venice, Italy, Oct. 4-5. Dr. Bruno de Biasio, S. Marco 4646, Venice, Italy, Secretary.

International Congress on Medical Records, London, England, Sept. 7-12. Miss Gwen Perkins, 120 C Street S.E., Washington, D. C., Chairman.

International Congress of Military Medicine and Pharmacy, Montevideo, Uruguay, March 1-7. Dirreccion General del Servico de Sanitad Militar, 8 de Octubre y Mariano Moreno, Montevideo, Uruguay, Secretariat of the Congress.

International Congress on Neuropathology, Rome, Italy, Sept. 8-13. Dr. Armando Ferraro, 722 W. 168th St., New York, N. Y., U. S. A., Secretary General.

International Congress of Otorhinolaryngology, Amsterdam, Netherlands, June 8-15, 1953. Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam, Netherlands, Secretary.

International Congress of Urology, The Waldorf-Astoria, New York, N. Y., U. S. A., Sept. 15-18. Dr. John A. Taylor, 2 East 54th St., New York, N. Y., U. S. A., Secretary-General.

International Hospital Congress, London, England, May 25-30, 1953. Capt. J. E. Stone, 10 Old Jewry, London, EC2, England, Hon. Secretary.

International Union Against Tuberculosis, Rio de Janeiro, Brazil, Aug. 24-27. Prof. Etienne Bernard, 66 Blvd. St. Michel, Paris 6e, France, Secretary-General.

Medical Women's International Association, Vichy, France, Sept. 12-16. Dr. Margaret M. Wurts, 27 East Wellesley Road, Montclair, N. J., U. S. A., National Secretary.

Neuroradiologic Symposium, Stockholm, Sweden, Sept. 17-20. Docent Ake Lindbom, Symposium Neuroradiologicum, Serafimerlasarettet, Stockholm K., Sweden, Secretary.

World Congress of Jewish Physicians, Jerusalem, Israel, Aug. 10-14. Secretariat: % Israel Medical Association, Rehov Mesilat Yesharim, Beth Kovatz, Jerusalem, Israel.

World Congress of Medicine, Montecatini, Italy, Oct. 16-18. Secretariat: Corso Trieste, 65, Rome, Italy.

World Federation of Mental Health, Brussels, Belgium, Aug. 25-Sept. 1. Dr. J. R. Rees, 19 Manchester St., London, W.1, England, Secretary.

World Medical Association, Athens, Greece, Oct. 12-16. Dr. Louis H. Bauer, 2 East 103d St., New York 29, N. Y., Secretary General.

DEATHS

Wood, Neal Naramore ⊕ Santa Clara, Calif.; born in Lamar, Mo., July 24, 1884; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1908; Army Medical School, 1912; commissioned in the regular Army Medical Corps and later resigned; served during World War I; formerly professor of public health (hospital administration) at College of Medical Evangelists, Loma Linda and Los Angeles, and on the faculty of his alma mater; formerly health director of Charlevoix, Emmet, Otsego, and Antrim counties in Michigan and in charge of the Bay County (Mich.) department of health; at one time superintendent of the Los Angeles County General Hospital in Los Angeles, and director of Starling-Loving University Hospital in Columbus, Ohio, and Hillman Hospital in Birmingham, Ala.; died in Agnews State Hospital in Agnew March 25, aged 67, of chronic myocarditis and arteriosclerosis.

Spangler, Davis ⊕ Dallas, Texas; born in Bonham, Texas, Sept. 30, 1892; University of Texas School of Medicine, Galveston, 1913; clinical assistant professor of radiology, Southwestern Medical School of the University of Texas; specialist certified by the American Board of Radiology; member of the Radiological Society of North America of which he was vice-president, American Roentgen Ray Society, and the American College of Radiology; past president of the Dallas County Medical Society; served in France during World War I; during World War II county chairman of procurement and assignment of medical personnel for the military services; affiliated with Texas Scottish Rite Hospital for Crippled Children, Children's Medical Center and Medical Arts Hospital; died in Gaston Hospital April 13, aged 59, of rheumatic heart disease and cononary sclerosis.

Booth, Thomas Fagan ⊕ Pulaski, Tenn.; Vanderbilt University School of Medicine, Nashville, Tenn., 1926; member of the American Academy of General Practice; died in Baptist Hospital, Knoxville, April 9, aged 50, of cerebral hemorrhage.

Brookie, Roger William ⊕ Flora, Ind.; Northwestern University Medical School, Chicago, 1908; served during World Wars I and II; died in Tifton, Ga., April 22, aged 69, of heart failure.

Caseley, Walter Norris, Long Beach, Calif.; Central Medical College of St. Joseph, Mo., 1895; affiliated with Seaside Hospital, where he died April 7, aged 78.

Cornell, Earl Hamilton, Oakland, Calif.; University of California Medical School, San Francisco, 1913; physician for the Matson Navigation Company for many years; died in Yreka April 23, aged 64, as the result of an automobile accident.

Harrod, George ⊕ Conway, Ark. (licensed in Arkansas in 1903); died April 11, aged 82, of heart disease.

Hawkins, Norman Louis ⊕ Watertown, N. Y.; Columbia University College of Physicians and Surgeons, New York, 1902; specialist certified by the American Board of Pediatrics; member of the American Academy of Pediatrics; affiliated with Good Samaritan and Mercy hospitals; died recently, aged 70, of cerebral embolism, arteriosclerotic heart disease, and chronic cholecystitis.

Hays, Thomas Walden, Cincinnati; Medical College of Ohio, Cincinnati, 1888; for many years on the staff of St. Mary's Hospital; died in St. Francis Hospital April 3, aged 88, of arteriosclerotic heart disease.

Henry, Taylor H., Columbus, Miss.; Memphis (Tenn.) Hospital Medical College, 1899; state senator; died in Jackson March 23, aged 76.

Herff, Adolph ⊕ Boerne, Texas; Jefferson Medical College of Philadelphia, 1880; fellow of the American College of Surgeons; formerly practiced in San Antonio, where he was affiliated with Santa Rosa Hospital; died recently, aged 93, of cancer.

Hoare, Harry James, Los Angeles; College of Physicians and Surgeons, Medical Department of the University of Southern California, Los Angeles, 1910; died recently, aged 68.

Hochwalt, Gustav A., Dayton, Ohio; Starling Medical College, Columbus, 1895; for many years affiliated with Miami Valley Hospital and St. Elizabeth Hospital, where he died recently, aged 79, of cardiovascular renal disease.

Jacobi, Felix ⊕ New York City; Kaiser-Wilhelms-Universität Medizinische Fakultät, Strasbourg, Germany, 1908; died April 1, aged 69, of leukemia.

Jacobs, Gustave Edgar ⊕ St. Louis; St. Louis College of Physicians and Surgeons, 1915; member of the American Academy of General Practice; died April 5, aged 68, of heart disease.

Kenoyer, William Ray ⊕ Hugoton, Kan.; University of Kansas School of Medicine, Kansas City, 1931; served during World Wars I and II; county coroner; died April 16, aged 54, of coronary heart disease.

Klaveness, Eivind, St. Paul; Kongelige Frederiks Universitat Medizinske Fakultet, Oslo, Norway, 1897; died April 10, aged 81, of coronary sclerosis.

Lee, Edwin Henry ⊕ Tuskegee, Ala.; Howard University College of Medicine, Washington, D. C., 1915; affiliated with Veterans Administration Hospital; died recently, aged 65, of melanosarcoma of the liver.

Leicht, Phillip ⊕ Lakemills, Wis.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1907; an Associate Fellow of the American Medical Association; served during World War I; died in Milwaukee recently, aged 69, of abscess of the lung and bronchopneumonia.

Leonard, Isaac Edward ⊕ Atlantic City, N. J.; College of Physicians and Surgeons, Boston, 1899; also a graduate in pharmacy; formerly member of the board of education; died March 31, aged 89, of cerebral thrombosis.

Levin, Arthur Aaron, Brooklyn; University and Bellevue Hospital Medical College, New York, 1921; died recently, aged 53.

Lowry, Casper Franklin, Dover, Fla.; Georgia College of Eclectic Medicine and Surgery, Atlanta, 1910; died March 30, aged 68, of carcinoma of the lung and liver.

McCarthy, Michael Joseph, Philadelphia; Medico-Chirurgical College of Philadelphia, 1916; died recently, aged 60, of carcinoma of the laryngopharynx.

McDonald, George Oliver ⊕ Detroit; University of Toronto Faculty of Medicine, Toronto, Ontario, Canada, 1920; died in Thamesford, Ontario, April 8, aged 60, of coronary thrombosis.

McLean, John Allan Cameron ⊕ Somerville, Mass.; Harvard Medical School, Boston, 1904; an Associate Fellow of the American Medical Association; served during World War I; veteran of the Spanish-American War; member of the senior consulting staff of Somerville Hospital, where he died April 8, aged 72, of cerebral hemorrhage.

McMillan, Robert C. ⊕ Monmouth, Ill.; the Hahnemann Medical College and Hospital, Chicago, 1905; served as city health officer; formerly on the staff of the Monmouth Hospital; died in Moline April 13, aged 89, of cerebral arteriosclerosis.

Meckstroth, Henry Louis ⊕ Mansfield, Ohio; Jefferson Medical College of Philadelphia, 1908; served during World War I; died March 12, aged 69.

Miller, Burton Jonathan, Massillon, Ohio; Homeopathic Hospital College, Cleveland, 1887; died March 18, aged 88.

Nanninga, John Benjamin ⊕ Newton, Kan.; Rush Medical College, Chicago, 1928; certified by the National Board of Medical Examiners; affiliated with Bethel Deaconess Hospital; died April 18, aged 53, of coronary occlusion.

⊕ Indicates Member of the American Medical Association.

Panaro, Peter Thomas ⊕ New York; University of Arkansas School of Medicine, Little Rock, 1928; died Feb. 7, aged 51.

Parkhurst, Jane Hunting Sutherland, San Francisco; New York Medical College and Hospital for Women, Homeopathic, New York, 1902; at one time health officer of Burlingame; served on the staff of Hahnemann Hospital, where she died April 18, aged 85, of coronary disease.

Parnell, Henry Elliott ⊕ Perrine, Fla.; Kentucky School of Medicine, Louisville, 1908; served overseas during World War I; died in Lake City April 20, aged 70.

Pectol, Charles F. ⊕ Spencer, Ind.; Louisville (Ky.) Medical College, 1898; for many years county health officer; died in St. Vincent's Hospital, Indianapolis, April 7, aged 85, of cerebral hemorrhage.

Ramstad, N. Oliver ⊕ Bismarck, N. D.; University of Minnesota College of Medicine and Surgery, Minneapolis, 1899; past president of the North Dakota State Medical Association; member of the founders group of the American Board of Surgery; died in St. Petersburg, Fla., recently, aged 76, of arteriosclerosis.

Ries, Gay K. Durbin, Wilmington, Del.; Woman's Medical College of the New York Infirmary for Women and Children, New York, 1892; at one time practiced in Chicago; died recently, aged 86, of myocardial infarction.

Rigby, Owen Compton ⊕ Shreveport, La.; Tulane University of Louisiana School of Medicine, New Orleans, 1920; member of the Southeastern Surgical Congress; fellow of the American College of Surgeons; on the staff of the Tri-State Hospital and North Louisiana Sanitarium; died recently, aged 63.

Sandifer, Vollie LaFayette ⊕ Logansport, La.; Atlanta Medical College, 1915; died recently, aged 62.

Scheer, George Henry, Sheboygan, Wis.; Rush Medical College, Chicago, 1903; served with the U. S. Public Health Service; affiliated with Sheboygan Memorial Hospital and St. Nicholas Hospital, where he died April 7, aged 74, of coronary thrombosis.

Schellhamer, William Heinly ⊕ York, Pa.; Medico-Chirurgical College of Philadelphia, 1916; member of the Industrial Medical Association; served during World War I; on the staff of York Hospital; died March 21, aged 59.

cherrer, Frederick William Jr. ⊕ St. Louis; St. Louis University School of Medicine, 1928; specialist certified by the American Board of Otolaryngology; on the staffs of the Barnes and Evangelical Deaconess hospitals; died April 7, aged 49, of cerebral hemorrhage.

Schindler, Hans Joachim ⊕ New York; Kaiser-Wilhelms-Universität Medizinische Fakultät, Strasbourg, Germany, 1905; died April 21, aged 70, of carcinoma.

Seville, David Walter ⊕ Bellevue, Pa.; Western Pennsylvania Medical College, Pittsburgh, 1900; on the staff of the Suburban General Hospital, where he died April 4, aged 82.

Seymour, Charles R., Binghamton, N. Y.; Albany (N. Y.) Medical College, 1892; member of the American Medical Association; twice president of the Broome County Medical Society; served during World War I; for many years connected with the National Guard; in 1944 was given a citation for distinguished service by the Alumni Association of Albany Medical College; on the staff of the Binghamton City Hospital, where he died April 28, aged 82, of coronary thrombosis.

Shaw, Manning Franklin, Omega, Ga.; University of Georgia Medical Department, Augusta, 1912; on the staff of the Vereen Memorial Hospital in Moultrie; died in John D. Archbold Memorial Hospital, Thomasville, April 2, aged 65, of carcinoma of the prostate.

Sheller, William Owen ⊕ Los Angeles; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1902; an Associate Fellow of the American Medical Association; affiliated with St. John's and Santa Monica hospitals in Santa Monica, and Culver City (Calif.) Hospital; died April 23, aged 78, of rupture of a dissecting aortic aneurysm, hypertension, and arteriosclerosis.

Sisk, Crete Nixon, Morganton, N. C.; University of Nashville Medical Department, 1905; district health officer; served as health officer of Vance County; died recently, aged 69, presumably of cardiovascular crisis.

Smith, Elisha Baker ⊕ Birmingham, Ala.; Birmingham Medical College, 1912; died recently, aged 64, of coronary occlusion and cerebral accident.

Smith, Frank Arthur ⊕ Westbrook, Maine; Medical School of Maine, Portland, 1915; died recently, aged 69, of cerebral hemorrhage and hypertension.

Smith, Robert Gillespie, Fayetteville, Ark.; Ohio State University College of Medicine, Columbus, 1940; served during World War II; died March 20, aged 40.

Smithwick, Marsena Parker ⊕ Boston; Harvard Medical School, Boston, 1895; formerly affiliated with the Boston City Hospital; medical inspector for the public schools; died March 30, aged 84.

Stephens, John Henry, Hendersonville, Tenn.; University of Nashville Medical Department, 1901; died recently, aged 81, of injuries received in an automobile accident.

Sweeney, John Joseph ⊕ Upper Darby, Pa.; Medico-Chirurgical College of Philadelphia, 1914; formerly trustee and councilor, Second Councilor District, Medical Society of the State of Pennsylvania; past president of the Delaware County Medical Society; affiliated with Misericordia, St. Agnes, and St. Joseph's hospitals, all in Philadelphia, and the Fitzgerald-Mercy Hospital in Darby, where he died April 18, aged 62, of gastric and duodenal ulcer and intestinal obstruction.

Talbot, Benjamin H., Ruston, La.; Medical Department of Tulane University of Louisiana, New Orleans, 1893; died April 27, aged 81, of cardiovascular accident.

Terrell, Leland Leslie, Woodbine, Ky.; University of Louisville (Ky.) Medical Department, 1910; for many years member of the school board; died in Lenoir City, Tenn., April 8, aged 62, of diabetes mellitus.

Thomas, Deleon Hale, French Camp, Miss. (licensed in Mississippi in 1911); died in Baptist Hospital, Jackson, April 2, aged 68.

Waller, Amos Orville ⊕ Eugene, Ore.; College of Physicians and Surgeons of San Francisco, 1916; past president of the Lane County Medical Society; served during World War I; formerly member of the state legislature; affiliated with Sacred Heart General Hospital, where he was chief of staff; died April 16, aged 69, of a heart attack.

Watson, James ⊕ Cleveland; Northwestern University Medical School, Chicago, 1925; specialist certified by the American Board of Psychiatry and Neurology; member of the American Psychosomatic Society, American Psychiatric Association and the Association for Research in Nervous and Mental Diseases; formerly chief neuropsychiatrist, Regional Office, Veterans Administration; served during World War II; an ordained minister; died April 27, aged 68, of myocardial infarction and arteriosclerosis.

Webb, William S. ⊕ Wheeling, W. Va.; College of Physicians and Surgeons, Baltimore, 1904; affiliated with Ohio Valley General Hospital and Wheeling Hospital, where he died April 11, aged 73, of acute suppurative pancreatitis.

Weighart, Joseph Benjamin ⊕ New York; Cornell University Medical College, 1901; for many years on the staff of French Hospital; died April 27, aged 79, of a heart attack.

Whitaker, Darel Wesley, Batesville, Miss.; University of Tennessee College of Medicine, Memphis, 1925; died March 22, aged 59, of coronary thrombosis.

Williams, Owen Hassell ⊕ Savannah, Tenn.; University of Nashville Medical Department, 1907; died March 22, aged 71, of a heart attack.

Wilson, Middleton Ford, New Orleans; Medical Department of Tulane University of Louisiana, New Orleans, 1903; died in Southern Baptist Hospital April 13, aged 73, of carcinoma.

Woltz, John Louis, Mount Airy, N. C.; Southern Medical College, Atlanta, 1897; died March 21, aged 79, of arteriosclerotic heart disease.

GOVERNMENT SERVICES

ARMY

Medical Service Celebrates 177th Anniversary.—The Army Medical Service will celebrate its 177th anniversary on July 27. From the small group of doctors and assistants organized as a medical department by the Continental Congress in 1775 at the behest of General George Washington, the Army Medical Service has grown to the largest military medical service in existence. Whether in combat or out, Army medical personnel are dedicated to the task of safeguarding and restoring the health of the individual soldier, and the combined efforts of its physicians, nurses, medical specialists, and enlisted men and women have created the healthiest combat force in history.

Society Election in Japan.—Col. George M. Powell is the newly-elected president of the Southern Honshu Medical Society, chosen during a society meeting at the 279th General Hospital. The colonel is chief of medical service at Osaka Army Hospital. Col. Sterrett E. Dietrich, commanding officer of the Kyoto Army Hospital, was elected first vice-president; Lieut. Col. John L. Mothershead, commanding officer, Nara Station Hospital, second vice-president; Major Carl Meeks, orthopedic clinic chief, 382nd General Hospital, secretary; and Lieut. Col. Edward P. Shannon, professional service chief, 279th General Hospital, treasurer.

ATOMIC ENERGY COMMISSION

Oak Ridge Institute Renews Contract.—The Atomic Energy Commission and the Oak Ridge Institute of Nuclear Studies, Inc., have signed a contract for the institute to continue its operation of certain programs for the AEC for another five years. This nonprofit educational corporation is composed of 30 member universities in the South and Southwest. The president of the institute is Dr. Paul M. Gross, vice-president of Duke University. The chief resident officer is Dr. William G. Pollard, executive director. Founded in 1946, with 14 charter member universities, the institute uses some of the unique atomic energy facilities in Oak Ridge to carry out research and training in the nuclear sciences.

The basic purposes of the institute are (1) to stimulate cooperation between the government and participating universities, in undertaking fundamental research in atomic energy; (2) to foster increased opportunities and improved programs of graduate study and education in nuclear energy in educational institutions of this region; (3) to aid in acquisition and training of scientific personnel; and (4) to use Oak Ridge's unique facilities for graduate research and instruction.

Specialized programs carried on by the institute include the conduct of courses in the handling of radioactive isotopes; the operation of a long-range research program on the treatment of malignant diseases; and the operation of the American Museum of Atomic Energy, together with its traveling exhibits. The institute also sponsors programs cooperatively with the Oak Ridge National Laboratory, which is operated by Carbide and Carbon Chemicals Co., and administers the AEC-sponsored fellowship program on a nationwide basis.

Member universities of the Institute are: Agricultural and Mechanical College of Texas, Alabama Polytechnic Institute, Catholic University of America, Clemson Agricultural College, Duke University, Emory University, Forida State University, Georgia Institute of Technology, Louisiana State University, Mississippi State College, North Carolina State College, Rice Institute, Tulane University of Louisiana, Vanderbilt University, Virginia Polytechnic Institute, University of Alabama, University of Arkansas, University of Florida, University of Georgia, University of Kentucky, University of Louisville, University of Maryland, University of Mississippi, University of North Carolina, University of South Carolina, University of Oklahoma, University of Puerto Rico, University of Tennessee, University of Texas, and University of Virginia.

DEPARTMENT OF DEFENSE

The Armed Forces Medical Library.—The appointment of Lieut. Col. Frank B. Rogers (MC) as director of the recently established Armed Forces Medical Library has been announced. The library, the largest institution of its kind in the world, will serve as a central medical library for the Army, Navy, and Air Force and as a national library for medicine and related sciences. The library's extensive facilities will also be available to medical research and development contractors, other governmental agencies, and civilian physicians and medical scientists of all countries.

Formerly known as the Army Medical Library, the newly-named establishment gained an international reputation through the publication of its Index-Catalogue, the most extensive bibliography of medicine in existence. In 1950 the decision was made to phase out the remaining activities of this monumental bibliography and to substitute for it a monthly publication, the Current List of Medical Literature. This publication enables the library to maintain an up-to-date bibliography of the world's medical literature.

Functioning mostly on a mail order basis, the library receives requests for books, references, and other material from all over the world. During the fiscal year 1951, more than 29,700 books were lent to borrowers in the United States. During that same period requests for information numbered 55,900. The library is able to furnish microfilms or photostatic copies of desired material for a very nominal sum. Also available are microfilm copies of publications which may be obtained on a loan basis. The photoduplication service received 56,000 orders requiring over 1,500,000 pages of film and print reproductions during the fiscal year 1951.

In 1942 because of space limitations, it became necessary for the library to move part of its collection to Cleveland, where a history of medicine division was established at the Allen Memorial Medical Library on the campus of Western Reserve University. To this location was transferred all of the medical literature written prior to 1850. The art section of the library has more than 21,000 pictures and almost 13,000 negatives.

PUBLIC HEALTH SERVICE

Fetal Deaths.—The 70,584 fetal deaths at the gestational age of 20 weeks or more registered in the United States for 1949 is the lowest number ever recorded, according to a recent report by the National Office of Vital Statistics. The fetal death ratio based on this figure is 19.8 per 1,000 live births. Although the 1949 figure is the lowest on record for the United States, it fails by a large margin as a measure of the pregnancy loss problem. The U. S. National Committee on Vital and Health Statistics has estimated that if fetal deaths of all gestational ages were recorded, the annual number in this country would be about half a million. However, until 1950, the definition of a fetal death (stillbirth) for registration purposes related exclusively to fetuses reaching the 20th week of gestation, and the definition and reporting requirements in many states varied from the national recommendation.

In 1950, the Third World Health Assembly recommended uniform adoption of the following, broader definition: "Fetal death is death prior to the complete expulsion or extraction from its mother of a product of conception, irrespective of the duration of pregnancy; the death is indicated by the fact that after such separation, the fetus does not breathe or show any other evidence of life such as beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles." The new definition has been adopted by numerous national organizations and during 1951 several states took action to place the definition in effect.

FOREIGN LETTERS

CHILE

Ten Years of Preventive Medicine.—The law concerning preventive medicine came into being in 1938 and applies to all workers who are included under the social security law. It consists of an annual examination, including serologic tests for syphilis, a chest roentgenogram, and a cardiovascular check-up (blood pressure and auscultation). If the person is suspected of having either syphilis, tuberculosis, or cardiovascular disease, he is sent for further investigation to a specialist. Free treatment and rest are provided if his condition can be improved. During the period of treatment the patient receives his usual salary, but it is paid to him by the government from a fund into which the employer pays 1% of the employee's salary. The study presented by Prof. B. Viel refers to the examination of 97,255 white collar employees of Santiago examined during the 10 years 1938-1947.

Syphilis.—Of the 97,255 persons examined the prevalency of persons with syphilis was 5%, there being 3 to 4 times more men afflicted than women. During the last 10 years the prevalence decreased by 53% in men and by 83% in women. The incidence, which was 1%, decreased by 50%. All this seems to prove that the number of cases of syphilis to be seen in the future will be fewer and fewer. The treatment did not produce epidemiological changes because in no case was the contact brought under examination. The drugs used were the arsenic-bismuth combinations, which are very antiquated. Now penicillin has radically changed the problem. The conclusion drawn by the author is that there is no sense in making a mass serologic test of the population partly because the vast majority of cases found in this manner are of the noncontagious type of syphilis and also because the number of syphilis victims is decreasing day by day.

Tuberculosis.—Of the persons examined for the first time between the years 1938 and 1947, 19% were sent to specialists in bronchopulmonary disease. The prevalency of tuberculosis was 5% and has decreased during the 10 years by 45%. The incidence was 1.5%, and its decrease is even greater than that of prevalency. Thus between the years 1943 and 1947 it dropped by 64%. During the 10 years of work, 4,741 persons were diagnosed as suffering from tuberculosis. Of these, 82% were diagnosed during their first check-up, 12.2% in the annual control examinations of persons previously considered as healthy, 2.9% in the annual check-up of inactive tuberculosis patients, and 3.1% during annual examination of patients who did not want to consult the specialist after their first check-up. Not taking into consideration this last group we may safely say that the most productive group in regard to the finding of tuberculosis patients, irrespective of the nature of their lesions, is that of the so-called inactive tuberculosis patients, followed by persons examined for the first time, and lastly by the annual check-up of the group of healthy persons. Of the 4,741 patients 39.4% had minimal tuberculous lesions, 34.5% moderate lesions, 9% advanced lesions, 8.6% extrapulmonary tuberculosis, and 8.5% pleurisy. Of the total of 4,741 patients 90.2% benefited from the law of preventive medicine. Of the 395 cases of pleurisy 96.4% were considered healthy after treatment, and 3.6% were incurable. Less than nine months of rest were necessary in 50%. Of the patients with minimal tuberculous lesions 97% were cured and 50% had less than eight months' rest. Of the 1,612 cases of moderate lesions 71% were cured. Analyzing these results the author concludes that the recuperation of the patient is better when the lesion is smaller and those with a negative bacilloscopy have a better prognosis than those with a positive bacilloscopy.

If we compare the percentage of persons who died after 10 years after having been treated with that of persons who were considered incurable from the very start we see that the mortality in the first group is 10% and in the second group 60%. The author points out that the surviving 40% of the incurable group presents a very serious problem in dissemination of the disease. Regarding the treatment, 4,276 underwent sanatorial rest, surgery, or pneumotherapy. The author points out that the sanatorial treatment was done with no sense of epidemiology. Thus only 41.1% of patients with a positive bacilloscopy were sent to sanatoriums, and 15% with a negative bacilloscopy and 20% in whom no search for Koch's bacillus was made were sent to sanatoriums. The author points out that, since there is such a shortage of beds, these must be used in a more practical manner and since those patients with a positive bacilloscopy are twice as dangerous from the point of view of contagion than those with a negative bacilloscopy the former group must be given preference for sanatorial rest.

Cardiovascular Diseases.—Of the patients examined 18.8% were sent to a specialist of cardiovascular diseases. The prevalency of cardiovascular disease was 5.8% and was higher than tuberculosis and syphilis. Whereas the latter two decreased considerably during the 10 years of observation the incidence of cardiovascular disease only showed a small drop. The incidence is 2%. At their first examination 77% of the total number of cases were diagnosed. The yearly examination of the so-called healthy group gave 18% and that of the suspected group gave the final 5%. Regarding the etiological diagnosis, hypertension accounted for 70% of the patients, including arteriosclerosis 35.8%, valvular lesions 8.2%, cardiovascular syphilitic lesions 6.6%, and myocardial infarctions 2.5%; dysthyroidism 1%; polyarticular rheumatism 1.6%; congenital heart disease 0.4%; and chronic pulmonary heart disease 0.3%.

The patients were catalogued into four groups according to their functional capacity: (1) those who were asymptomatic, (2) those who presented discrete symptoms but were still able to work, (3) those whose working capacity was decreased, and (4) those who presented symptoms even at rest. During the first check-up 61% belonged to the first group, 20.3% to the second, 16.8% to the third, and 1.3% to the fourth. This brings us to the conclusion that only 36% of the patients require therapy. The author points out that 70% of the hypertensive group were asymptomatic, and 68.8% of the arteriosclerosis patients, 11.2% of the polyarticular rheumatism patients, 48.5% of nonsyphilitic valvular lesions patients, 63.8% of cardiovascular syphilitic patients, and 34% of the congenital heart disease patients did not have symptoms. The average age was 48 years in patients with heart disease, 36.7 years in the syphilitic group, and 30.3 years in the tuberculosis group. Of the 6,724 diagnosed cardiopaths 1,330 were given the benefits of the law. The percentage of those to enjoy the privileges of the law was smaller in the hypertensive and the arteriosclerotic group. This was to be expected because approximately 70% of these patients were asymptomatic. The law was applied to persons of the second and third group of the above-mentioned classification. The first was excluded because the patients were asymptomatic, and the fourth was excluded because the patients were considered as incurable. Thus 30% of the patients in the second group were receiving benefits from the law and 70% of the third group. Thus we see that 19% of all diagnosed cardiovascular patients received active therapy. Eighty per cent of the patients were improved, and therapy failed in only 10.7% of the cases; however, the high percentage of improvement obtained should not give a false impression of the situation because during these 10 years 209 patients who were considered improved presented on one or more occasions symptoms of relapse. Of this unimproved group 50% were hypertensive patients, 20% with valvular lesions, 10% arteriosclerosis, 7.5% myocardial infarctions, and 6.3% rheumatism; 0.6% were victims of hyperthyroidism; and 2% had other diseases.

The items in these letters are contributed by regular correspondents in the various foreign countries.

Mixed Pathologic Conditions.—1. Syphilis and tuberculosis: Of the patients with active syphilis 3.2% had some type of tubercular lesion. If we would apply the prevalency of tuberculosis to syphilitic patients to obtain the number of patients with tuberculosis that should exist theoretically among them we would find that the number is 222 cases whereas the 3.2% represents 170 cases. Thus a superficial analysis might induce us to the erroneous conclusion of saying that syphilis decreases the risk of tuberculosis. The author rejects this affirmation by saying that we cannot apply the prevalency of the tuberculosis group to the syphilitic group because of the difference of age in each case. 2. Syphilis and cardiovascular disease: In 443 cases the diagnosis was a syphilitic cardiovascular lesion. In case the prevalency of cardiovascular disease was to be applied to syphilitic patients the number of heart disease cases should have been 302, instead of the 443; however, there is a difference of 12 years between the average age of the syphilitic patient, who is much younger, and the cardiac patient. 3. Tuberculosis and cardiovascular disease: Only 1% of the tuberculous patients had heart disease. Thus if we were to apply the prevalency of cardiovascular disease to the tuberculosis group we could expect 270 instead of the 47 cases. The age factor again plays an important part in the correction of the prevalence. Concerning mixed pathologic conditions the author points out that, except in cases of syphilis and cardiovascular lesions, there are very few cases in which there are more than one diagnosis.

ISRAEL

Rabies Campaign.—Rabies incidence has been declining steadily in Israel since the start of the intensive nationwide campaign in October, 1950, according to Dr. Martin Kaplan, chief veterinary officer of the World Health Organization. The antirabies campaign, which is being waged with the assistance of WHO, includes a mass vaccination of dogs with a new egg-adapted vaccine (Flury strain), which has proved harmless to dogs and seems to give greater immunity of longer duration than other vaccines so far produced. The number of rabies cases in animals has dropped from an average of 100 a year before the start of the campaign to 10 (1 bull, 1 jackal, and 8 unvaccinated dogs) in the course of the past 15 months. All dogs in Israel have been registered, and vaccinated animals have received suitable identification. All stray dogs are being destroyed, while newborn animals are vaccinated when they reach 6 months of age. Vaccination is compulsory and free of charge.

Although it is still too early to determine to what extent vaccination alone has brought about the fall in rabies incidence in animals, Dr. Kaplan believes that the concentrated attack has played an important part in this matter. The drop is mirrored also in the decline in human deaths from rabies, which were none in 1951, whereas they numbered 20 between 1948 and 1950. In addition, the number of persons receiving Pasteur treatment following dog bites, which averaged 4,000 a year before the start of the campaign, has been cut to about one-half.

The campaign, which will be continued for the next few years, is expected to serve as a demonstration of the most effective method of rabies control and to provide a guide for other countries where the incidence of rabies is high. At the same time, it is yielding valuable information on the vaccine in use, such as the duration of immunity conferred by it under field conditions. Laboratory studies have shown that one inoculation of the vaccine gives adequate protection for a minimum period of two years. No revaccination of dogs in Israel will be practiced until there are indications that the immunity in vaccinated animals is beginning to wane.

Of equal importance is the greater feeling of security now being experienced among the population of the agricultural settlements. Israel's Veterinary Services, in cooperation with the Ministry of Health, are in charge of the campaign, while the necessary vaccine is being supplied at present through the WHO but will be produced in Israel in the future. WHO technical guidance is being provided through Dr. Kaplan. On his last visit, Dr. Kaplan was accompanied by Dr. Ernest S. Tierckel, who was loaned to WHO by the United States Public Health Service, where he directs rabies control activities. Dr. Tierckel has just completed a three month survey of rabies problems in Northern and Southern Rhodesia, Ceylon, Iraq, Greece, and Israel for WHO.

Rh Blood Types in Jews in Jerusalem.—J. Gurevitch and his associates from the Hebrew University-Hadassah Medical School, Jerusalem, have just finished a study of the Rh blood types of Jews living in Jerusalem.

Relatively few studies have been reported on the incidence of the Rh factor in Jews. In 1945 Gurevitch found in a select group of 400 women from different parts of the country that 15% were Rh-negative. It was obvious that this figure could not be considered as representative of a population selected at random. In later studies of a larger series, similar results were found for Ashkenasi (11% Rh-negative) and Sephardi groups (11.8% Rh-negative), whereas a much lower rate of persons with Rh-negative type blood was found among Yemenites (2.3% Rh-negative) and Kurdistani-Persians (1.5% Rh-negative). No instances of hemolytic disease of the newborn infant have been observed among 310 Yemenite and 1,739 Kurdistani-Persian Jewish women at the time of delivery.

A total of 1,335 samples of blood were tested for Rh types in Jews in Jerusalem with the aid of the following serums: anti-rh' (anti-C), anti-Rh_o (anti-D), and anti-rh" (anti-E). The subjects have been divided into three main groups: (1) Ashkenasi Jews, i. e., Jews of European descent, most of whom are from eastern European countries, (2) Sephardi Jews, descendants of Jews from medieval Spain, including Jews from North Africa (Algiers, Tunis, and Morocco), from southern Europe (Bulgaria and Greece), and an indigenous group from Israel, and (3) an Oriental group, including Jews from countries east of Israel (Iraq, Syria, and Afghanistan) but not from Persian and Kurdistani communities.

Percentage of Rh Types in Jerusalem Jews

Communal Group	No. of Persons	Types of Blood							
		Rh_1	Rh_2	Rh_1Rh_2	Rh_o	rh	rh'	rh"	rh'rh"
Ashkenasi	946	57.7	10.9	13.0	3.7	13.5	1.2	—	—
Sephardi	252	67.0	5.2	7.5	7.1	12.3	0.8	—	—
Oriental	137	64.3	10.2	7.3	4.4	13.1	0.7	—	—

In all three communal groups, the frequent occurrence of the Rh_1 (CDe) type was noteworthy, and this was especially true in the Sephardi group (67%) and among Oriental Jews (64.3%). Similar high percentages of Rh_1 type have been reported in Chinese and in Asiatic Indians. The rate of Rh_1Rh_2 among the Sephardi (7.5%) and Oriental Jews (7.3%) is low as compared with its rate in the Ashkenasi group of Jews and in other races.

The frequency of Rh-negatives (cde/cde) in this series was higher than that reported in previous studies and approached the rate for Rh-negatives reported for Europeans. It is noteworthy that the percentage of Rh-negative persons was similar in all three communal groups.

Convention of Psychiatrists in Haifa.—The Fifth National Convention of the Association of Neuropsychiatrists was held during May, 1952, in Haifa. It was disclosed at the gathering that, whereas the generally accepted figure of beds for mental cases is four for every thousand of the population, there are only 2,200 beds in Israel instead of the required 6,000. Generally speaking, the proportion of mental cases is higher in this country than in others, a fact that is accounted for by the large immigration (among which the proportion of mental cases is higher than average), the economic conditions that prevail locally, the overcrowding in dwellings, and the special difficulties encountered in new points of settlement, in *ma'baroth*, and in immigrants' camps. The hospitalization position for mental cases is catastrophic, a long time having to elapse before beds can be found for such patients. Suicides and acts of violence have been the result.

The convention voiced its demand for the establishment of work villages for chronic patients, labor camps for mental defectives, and a suitable home for epileptic patients, there being no such thing in the country at present.

Cardiovascular Research Unit.—The newly established cardiovascular research unit of the Hadassah University Hospital is headed by Dr. K. Braun. At a meeting of the Jerusalem Medical Association in February, 1952, the first report on the results of cardiac catheterization in patients with mitral stenosis was delivered. The pertinent findings were a rise in arteriovenous oxygen difference, a decrease in cardiac output and stroke index, a marked elevation of pulmonary arterial pressure, and some rise in right ventricular pressure. The competency of the right ventricle as an important factor in the production of paroxysmal pulmonary edema was emphasized.

At the meeting, Dr. A. M. Cohen of the Rothschild Hadassah Hospital reported on 18 patients with rheumatoid arthritis, bronchial asthma, and severe urticaria, who were treated by an intravenous drip of corticotropin. They found that a dosage of 1 to 3 mg. daily resulted in satisfactory clinical improvement. Unfavorable side-reactions were extremely few. The economical aspects of this treatment were stressed.

Dr. K. Braun discussed the effects of corticotropin on the pulmonary function in patients with bronchial asthma and obstructive emphysema treated by the above method. In the majority of patients, there was an improvement in the ventilatory functions of the lungs.

JAPAN

BCG Vaccination.—The annual tuberculosis death rate in Japan was around 200 per 100,000 population since 1900 showing a gradual decrease during the period 1925-1935. Although the highest peak was presented in the last war, the rate has decreased ever since 1947, dropping to 146.8 in 1950 and 111.1 in 1951.

The incidence of tuberculosis has been highest in the age group 15 to 29 as compared with that of the other groups. Almost all of these cases were of primary tuberculosis, proved by extensive studies clarifying the time of primary infection by means of repeated tuberculin testing on the nonreactors and the x-ray examination on the inverters. Indeed, nearly all of the pathological changes discovered through x-ray examination had appeared within one year after the positive inversion. As one of the chief measures for the prevention of the disease, stress was placed on the BCG vaccination of the tuberculin negative patients together with x-ray examination on the positive reactors.

The first attempt of BCG vaccination in Japan was made in 930 on the student nurses by Prof. Arao Imamura of the Osaka University. In 1938, the National Health Administration Committee of the Japan Association for the Promotion of Sciences organized an antituberculosis committee, the chief object of which was to make a study of the testing method of tuberculin reaction and of the use of BCG vaccination. The results of the five year research of this committee were published in April, 1943, proving the safety and efficacy of BCG vaccine.

In the year 1942, the welfare ministry recommended vaccination for all children applying for a position after leaving the primary school. The number of vaccinated persons increased year after year chiefly among such groups as the 7 to 25 year age group, workers in offices and plants, students, and families of tuberculous patients, totaling more than 7 million within 1948.

The Vaccination Law was put in force in July, 1949. 1. All of the tuberculin negative reactors under 30 years of age were to be included in the mass vaccination program. 2. Selection of the group to receive vaccination was to be done by tuberculin testing. Five toxic units of old tuberculin were injected intracutaneously into the flexor surface of the forearm, and after a 48 hour reading, BCG vaccination was made on the negative reactors with local redness less than 9 mm. in diameter. 3. Choice of groups for revaccination were to be made in the same way as the above. The number of vaccinated persons in 1950 was 13,941,855.

Because of its strong local reaction, ulceration and abscess, the subcutaneous injection was replaced in 1940 by the intracutaneous method: that is, intradermal injection of 0.1 ml. of the vaccine containing 0.4 mg. BCG per ml. Meanwhile, studies on scarification and multiple puncture methods are in progress. For the compulsory vaccination, the dried vaccine is to be employed, the products of which are carefully assayed to insure the safety and efficiency of the vaccine. The production of frozen-dried BCG vaccine is conducted by the Antituberculosis Association and its production plants located in Tokyo, Sendai, and Osaka. The total production of the vaccine will amount to 45 million doses annually.

PARIS

Treatment of Status Asthmaticus with ACTH and Cortisone.—Since 1950, Prof. Pasteur Valéry-Radot, C. L. Laroche, and their associates have treated with corticotropin (ACTH) and cortisone 33 patients with severe status asthmaticus at the medical clinic of the Broussais Hospital. The patients remained under observation for three months with the following results. Seventeen patients experienced a functional recovery lasting for some weeks; several of these, formerly invalids, were enabled to resume their occupations, and four had no crisis for two to eight months; but in six asthma reappeared after a remission of two months. Two complete failures were noted. In favorable cases, signs of improvement were observed from the second or third injection (12 to 24 hours). Expectoration diminished or disappeared in 22 patients. Eosinophils disappeared from the second day of treatment. The authors began the treatment with 75 to 100 mg. of corticotropin daily in divided doses for three to eight days, the dosage being then diminished. The treatment lasted for 3 to 23 days. In nine patients it was supplemented with a few injections of cortisone, without any noticeable change in the results. In eight patients multiple cures were undertaken. Studies with Thorn's test (fall of the eosinophil level) were made on 19 patients, and in 10 cases only was a fall of less than 50% obtained. There was no parallelism between the fall in eosinophils and the degree of therapeutic improvement; five patients with negative tests had excellent results with hormonal treatment. As regards toxic incidents, no outbreak of hypertension was noted, but six patients experienced hypotonia with marked asthenia; to prevent this a daily dose of from 1 to 2 gm. of potassium chloride was given during the treatment. In order to avoid infections (three pulmonary infections having been observed) the patients were given 1 gm. of aureomycin daily. Three deaths occurred in the course of treatment; in one case, the patient showed signs of cardiac deficiency, and the treatment, giving no results, was discontinued after 775 mg. had been administered; the other two patients died in severe asthma on the 27th and 34th day of treatment. The autopsy of one of them revealed an obstruction of the bronchi by secretion. The authors believe that, because of the possibility of an aggravation of the patients' condition following discontinuation of the treatment, medical supervision is required during the period called "hormonal weaning"; moreover, corticotropin and cortisone do not modify the evolution of the disease, being only symptomatic medications, and, immediately on the disappearance of critical phenomena, the patient should be given etiological treatment.

Hyaluronidase in the Toxicosis of Nurslings.—The value of intravenous perfusion in the treatment of toxicosis of nurslings is indisputable, but there are technical difficulties as well as the deadly risk of pulmonary edema, owing to the difficulty of regulating the drip. The substitution of subcutaneous for intravenous administration failed to produce the anticipated results because of the too slow resorption of the serum. Professor Turpin and his associates report that 250 cc. of an isotonic sodium chloride solution with hyaluronidase, injected subcutaneously, is absorbed within 10 minutes, without pain or distention of tissues. No dressing of hyaluronidase is used, and no incident is to be feared; there is only one contraindication to be noted: a serious skin infection that might be spread by hyaluronidase. In a 14-day-old child, treated by an intravenous perfusion, an extensive, hard subcutaneous infiltration, feeling like cardboard, appeared 24 hours after the perfusion in the lower limbs. Hyaluronidase was administered and, paradoxically, succeeded in bringing about the resorption of the edema, a reduction of weight, and an improvement in the general condition. It appears, therefore, that hyaluronidase has a general regulating action on the water exchanges between circulation and subcutaneous tissues, but this hypothesis requires verification. In the other cases, the authors observed that, with perfusion with hyaluronidase, definitive success was obtained after failure of intravenous transfusion. They believe that intravenous perfusion will become a useless technique, save in the exceptional cases of collapse, and that subcutaneous perfusion may be substituted.

TURKEY

Activities of Institute of Hygiene.—The Ankara Refik Saydam Institute of Hygiene was established in 1932 with an initial budget of 700,000 Turkish pounds from a special fund provided by the Ministry of Health and Social Assistance. The Rockefeller Foundation gave an $80,000 grant, which was spent on scientific equipment for the microbiology laboratory, the institute's first building; a plaque in the hall commemorates the philanthropic gift. At present the institute has three main buildings, in which are located departments of microbiology and chemistry, serology, virology, pharmacodynamics, departments that study vaccines and bacterial vaccines, antigens, and control of disease, and a kitchen for the study of nutrition. A vaccination center was opened in 1947.

The scientific staff of the institute consists of 16 specialist physicians, 4 veterinarians, 8 pharmaceutical chemists, 52 laboratory assistants, and 41 technicians.

In the *Bulletin of Hygiene and Experimental Biology*, Dr. Niyazi Musa Erzin and Dr. Orhan Hulusi Balkan reported on the activities of the institute from 1933 to 1949. Research concerned such public health problems as malaria, tuberculosis, trachoma, brucellosis, food-carried diseases, insects and insect-borne diseases, cholera, plague, and epidemics that may be introduced from neighboring countries. Until 1934, smallpox and bacterial vaccines and serums were prepared in Istanbul, and much was imported. They are now prepared at the institute, and during the last war Turkey sent cholera and plague vaccines as gifts to Egypt and Far-Eastern countries. Smallpox and bacteria vaccines are prepared in two departments, and BCG vaccine in a separate division. Cholera vaccine is obtained from five different strains received from Basra, Pasteur Institute, Marseilles, Egypt, and Istanbul. In 1947, because of the cholera epidemic in Egypt, 9,256 liters of vaccine were prepared and 8,295 liters were distributed; that year the vaccine contained 4 to 8 million germs in 1 cc., and the whole population in the south and in the southeastern provinces of Turkey was vaccinated. In 1948, 6,344 liters were prepared and 6,165 liters were distributed. Plague vaccine is prepared from six different strains, three of these are of old Turkish origin and three originated in Cairo. In 1947 the production was 4,416 liters, of which 3,854 were distributed. Polyvalent dysentery vaccine is prepared from Flexner's Y and E Turkish strains from Bucharest and the Pasteur Institute, and Shiga's R and S strains. Production in 1948 was 254 liters, of which 148 liters were distributed. Antimeningococcus vaccine is prepared from different Turkish strains. Since the introduction of penicillin and the sulfonamides its production has been reduced. Production in 1948 was 78 liters, and 73 liters were distributed. Combined typhoid and paratyphoid A and B vaccines are prepared for prophylactic use from selected strains of high antigenic value: 1 cc. contains 500 million typhoid and 250 million each of paratyphoid A and B strains. Production in 1948 was 2,856 liters, of which 2,100 liters were distributed. The production of antipneumococcus vaccine, types 1, 2, and 3, has been discontinued since 1941 in favor of penicillin and sulfonamides. From 1933 to 1948 BCG vaccine was prepared for oral administration only. The 1948 production was 14,800 doses, of which 7,309 doses were distributed. In 1948, 370 cc. of BCG vaccine for intracutaneous injections were prepared and distributed. The production of pertussis vaccine for prophylactic and therapeutic use was begun in a new laboratory during the latter months of 1948. It is prepared from Turkish and American strains and contains 1,000 million germs in 1 cc.; of 18 liters 3 liters were distributed. Staphylococcus vaccine is prepared from Staph. albus, Staph. aureus, and Staph. citreus; 22 liters were prepared in 1948, and 13 liters were distributed. Brucella vaccine is prepared for therapeutic use; it is obtained from more than 30 strains of Turkish and foreign origin. There are 1,000 million germs in 1 cc.; the vaccine is preserved in a dry state. Nine liters were prepared in 1948. Rabies vaccine for distribution to the 82 rabies centers is prepared according to the Semple method; it contains 5% brain tissue. That vaccine used at the institute vaccination station is prepared according to the Högyes method; 815 liters were prepared in 1948, of which 752 liters were distributed. Smallpox vaccine, after passage through two calves, is given an additional passage through a young donkey to strengthen the virus; the results have been very satisfactory. The smallpox vaccination law, which has been strictly enforced for 67 years, requires that vaccination be given during the first six months of life; it is required on entering schools and military or civil service, and in the event of an epidemic the whole population of that locality is revaccinated. In 1948 3 kg. of smallpox virus stock were prepared, and 26 million doses were distributed. In 1948 over a thousand liters of typhus vaccine were prepared according to the Cox method, and as a precautionary measure, a substantial amount was forwarded to the provincial health departments. The production of polyvalent bacteriophage for the use of summer diarrhea amounted to 730 liters; 536 liters were distributed. Very good results have been obtained with bacteriophage therapy combined with sulfaguanidine. Combined vaccines prepared during 1948 were: typhoid-tetanus 6,360 liters; diphtheria-tetanus 8,250 liters; typhoid-typhus 4,288 liters; typhoid-diphtheria 70 liters; plague-cholera 13 liters; typhoid-cholera-typhus 100 liters.

The preparation of toxins and anatoxins in Ankara was begun in 1934. For the preparation of diphtheria toxin and anatoxin, modified Müller-Wadsworth medium is used. There were 998 liters of diphtheria toxin and 864 liters of anatoxin prepared in 1948. Tetanus toxin and anatoxin is prepared from Pasteur Institute and Stick Dorne strain; 1,173 liters of toxin and 309 liters of anatoxin were prepared in 1948. Scarlatina toxin is prepared from hemolitic streptococci grown on media modified after the Ramon method; 139 liters were prepared in 1948.

Until 1933, serums were prepared in Istanbul, but much was imported; this is no longer necessary. The institute has no farm yet, but there are modern stables for serum and experimental animals. For the preparation of serums, horses and oxen are used; rams, young donkeys, and, sometimes, goats are used for rabies serum. The animals are quarantined for 20 days; they are tested for glanders and other diseases and are given tetanus inoculations. For the immunization toxins, anatoxins, germ emulsions, and stock strains are used. Tetanus serum for prophylactic use is prepared from oxen. It contains 1,000 and 3,000 units in 1 cc. For therapeutic use it is prepared from horses and contains 3,000 units, 10,000 units, and 30,000 units in 1 cc. There were 803 liters prepared in 1948. Diphtheria serum for prophylactic use is prepared from cattle and contains 1,000 units in 1 cc.; for therapeutic use it is prepared from horses, and it contains 3,000 units, 10,000 units, and 30,000 units in 1 cc.; 495 liters were prepared in 1948. The suspension of the organisms used for the immunization of horses from which polyvalent dysentery serum O is prepared contains 50% Shiga's bacillus. The serum of animals that have a minimum of 300 units in 1 cc. is regarded as satisfactory. The finished product contains 3,000 and 10,000 units in 1 cc.; 247 liters were prepared in 1948. Meningococcus serum is prepared from four foreign and Turkish strains; 176 liters were prepared in 1947. With the introduction of penicillin and sulfonamides, its production has been greatly reduced. Scarlet fever serum production was 58 liters in 1948, and polyvalent gangrene horse serum production was 331 liters. Scorpion antivenom is obtained from horses immunized with toxin of a scorpion found in Turkey; during 1945 to 1948, 55 liters of it were produced and almost all were distributed. Of 92 liters of rabies serum prepared in 1948, 16 liters were distributed. There were 934 liters of antianthrax serum prepared in 1948, of which 430 liters were distributed. Normal serum is of two kinds: one is for oral administration, the other for use in bacteriology laboratories; 134 liters were prepared in 1948.

Antigens prepared during 1948 included: Wassermann antigen (Organ) 11,600 cc.; Kahn antigen 8,260 cc.; Sachs antigen, 1933-1938, 8,500 cc., since 1938 discontinued; hemolitic serum 2,540 cc.; tuberculin 3,700 cc.; metylic antigen, bovine and human type, 2,570 cc.; diluted metylic antigen 3,330 cc.; and brucellargen for Brucella allergy 400 cc.

CORRESPONDENCE

GASTRIC LESIONS OF LOEFFLER'S SYNDROME

To the Editor:—I am very much interested in the article in The Journal, June 7, 1952, entitled "Gastric Lesion of Loeffler's Syndrome," reported by Ruzic, Dorsey, Huber, and Armstrong, and the findings at operation. I have seen the same lesions of the stomach with the comparable roentgenologic observations due to aureomycin. When therapy with the drug had been discontinued after a period of five to six weeks, the stomach completely returned to normal.

In one patient who came under my observation in July, 1949, and who had been given a copious dose of aureomycin, on roentgenologic examination edematous, pseudopolypoid diffused changes were found. These changes were so severe that we were considering gastrectomy. The gallbladder could not be visualized. X-ray examination was again performed on Aug. 9, 1949, and there was a complete disappearance of these pseudopolypoid changes of the stomach. The patient was operated on at the Johns Hopkins Hospital on Sept. 7, 1949. A cholecystectomy was performed, and the stomach gave no evidence of any lesion at operation. Gastroscopy one month before had revealed no evidence of any changes in the gastric mucosa, and this was verified on Aug. 9, 1949, by a repeat x-ray examination of the stomach.

Could not the lesion described in this so-called gastric lesion of Loeffler's syndrome be due to the changes that can occur in the gastrointestinal tract in association with the intake of aureomycin?

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THE FUTURE OF OTOLARYNGOLOGY

To the Editor:—The editorial entitled "The Future of Otolaryngology," which appeared in the April 26 issue of The Journal, page 1508, was a true summary of the information now available in connection with otolaryngological practice today. However, more should have been said about meeting the challenge in spite of gloomy forecasts by some and bright predictions by others. It should be emphasized strongly that there always was, to a certain extent, the medical otolaryngologist whose chief interest lay in local rhinological and otological diseases (now allergic, psychosomatic, and dermatological) and the surgical otolaryngologist who quickly took up plastic surgery, bronchoesophagology, laryngoscopy, and even gastroscopy. Some mastered fenestrations, laryngeal surgery, neck dissection, esophageal surgery, and thyroid and cancer surgery; some also mastered ophthalmology and diseases of the chest.

Some of those who still practice the conventional field of otolaryngology, especially in smaller communities, call the surgeons "super-specialists," overlooking the greater opportunities available in our larger urban areas with the numerous postgraduate facilities.

I should like to call attention to part of a paper published by Dr. C. Gale in the *Eye, Ear, Nose and Throat Monthly* (30:601 [Nov.] 1951): "In the last few years otolaryngology has rapidly lost ground as a specialty. There are several reasons for this trend. The use of antibiotics and sulfonamide drugs has in great measure reduced the number of surgical procedures formerly commonplace. Surgeons who previously have boasted of using up a complete set of mastoid chisels every year, today rarely have the opportunity of doing a simple mastoidectomy. The general practitioner now armed with the newer antimicrobial agents has in large measure taken over the treatment of the acute and chronic infections of the sinuses, pharynx, and larynx. Even the fields of allergy, in all of its ramifications, have to a certain degree been taken from the specialist's domain by the introduction of the antihistamine drugs."

The above-mentioned author dwells on the reaction on the part of the otolaryngologist who was prompted to investigate and pick up all loose ends of his specialty. The first of the loose ends that became integrated into the specialty was plastic and reconstructive surgery. More important was the realization that the nose and throat surgeon could also be a head and neck surgeon, that there is nothing to prevent the otolaryngologist from doing cancer operations of the head and neck, thyroid surgery, or, for that matter, esophageal surgery. The importance of psychosomatic medicine is emphasized in the armamentarium of the specialty. Thus speaks an otolaryngologist in the largest city of our nation.

In conclusion, aside from expensive experimentation, especially in connection with private practice, there is no reason for anyone with surgical skill in certain techniques not to take up or advance in the various fields (unless handicapped by economic means). There is no reason for the nonsurgical physician to neglect such studies as allergy, audiology, physical medicine, and psychosomatic medicine. Besides, who prevents him from using the antibiotics and sulfonamide drugs? The various papers in the medical press and the excellent editorial of The Journal will do the problem more good than mere criticism or superficial suggestion.

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THE CERVICAL CAP

To the Editor:—I question the advisability of publishing such inconclusive articles on the delicate field of infertility as "The Cervical Cap in the Treatment of Male Infertility" (*J. A. M. A.* **149:**427-431 [May 31] 1952). The authors state that the use of cervical cap is unquestionably indicated. I question this, for they do not adequately describe relative semen inadequacy and they do not adequately describe the sequence of events that preceded the use of the cap. They state that decreased metabolic function in one or both partners or other endocrine disturbances were corrected promptly. I beg to point out that these disorders are not corrected promptly. Furthermore, 15 cases of primary sterility with conception achieved in 10 (67%) hardly warrants publication, particularly when only 7 of their successes are true oligospermia as defined by Dr. John MacLeod of Cornell University.

The authors state that ovulatory time is determined by the vaginal smear technique. Again, this technique has not been proved to determine ovulatory time in any manner satisfactory, accurate, or informative. Many disagree as to the time of ovulation as determined by this technique. Further, there is no control over the patient's practices at home. In summary, I question advocating the cervical cap in the cases described.

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PRESSURE TREATMENT OF MAXILLARY SINUSITIS

To the Editor:—Regarding the article "Pressure Treatment of Maxillary Sinusitis" by J. Mathews Robison in The Journal (page 436, May 31), I would like to say that this article is interesting and worth reading from the academic and philosophical viewpoint, but I doubt that it has any real practical application for the operating room.

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BUREAU OF MEDICAL ECONOMIC RESEARCH

THE COST OF LIVING AND MEDICAL CARE PRICES

Frank G. Dickinson, Ph.D.

In the past year, the index of the prices of all items in the consumers' budget has risen almost twice as many points as the price of medical care. The Consumers' Price Index rose 13.7 points in 1951, and the index of medical care and drugs rose 7.1 points. In 1951, the Consumers' Price Index reached an all-time high of 185.6 while the index of the prices of medical care and drugs stood at 155.0. While the Consumers' Price Index has been considerably higher than the index of medical care prices in recent years, the spread between the two indexes became even more pronounced during 1951. The Consumers' Price Index had risen 86% since the base period, while the cost of medical care rose only 55%. In terms of purchasing power of the consumer dollar, medical care was 16% cheaper in 1951 than in 1935-1939, that is, 155.0 is 84% of 185.6.

As explained in our earlier publications,[1] the Consumers' Price Index, which is compiled by the U. S. Bureau of Labor Statistics, measures monthly the changes since 1935-1939 in the prices of fixed quantities of goods and services normally purchased by moderate-income families in large cities. The indexes of the medical care items are a small part of the entire index. This index is considered our best general measure of the purchasing power of the consumer dollar and is widely used to determine wages in labor negotiations.

How have the prices of the individual medical care items behaved? As shown in the accompanying table, the index of medical care excluding drugs was 160.9 in 1951 and the index of prescriptions and drugs, 128.4. The index of physicians' fees (general practitioner, surgeon, and specialist) was 145.2; of general practitioners' fees, 145.2; and of surgeons' and specialists' fees, 144.3. The index of dentists' fees was 160.0 and of hospital rates, 260.7. The price index of group hospitalization, which was recently added to the items priced by the Bureau of Labor Statistics (December, 1950=100), was 103.1 in 1951; thus group hospitalization (Blue Cross premium rates) rose 3.1% during 1951, the first full year covered by this newly added index. Again, with the exception of hospital rates, the indexes of each medical care item were considerably below the Consumers' Price Index. In fact, if the hospital portion of the index of medical prices were excluded, this index would be even lower than the reported level of 155.0.

As the Bureau has previously pointed out, hospitals as buyers of large quantities of food and labor feel the full impact of inflationary forces. They must adjust their rates to cover the vastly increased cost of the materials they purchase. The hospital's costs are not stabilized by such customary accounting items as depreciation and taxes, because most hospitals are charitable institutions. The hospital index may also be uniquely affected by the choice of the base period. While 1935-1939 is considered a relatively normal period by most economists for the purpose of general comparisons, in the case of hospitals it may have some shortcomings. For example, the shortening of the nurses' working day to eight hours, which would considerably affect the labor costs of hospitals, was not fully realized until after 1935-1939. Other changes may have counterbalanced this factor. It is, of course, difficult to judge the appropriateness of a base period for individual items.

Although it is somewhat outside the scope of an article on prices, the decrease in the average length of stay in hospitals should be noted. While hospital rates have increased sharply, the decreased length of stay in hospitals has reduced the average patient's hospital bill considerably. If more information were available, it probably would be possible to make volume or quantity adjustments in the other medical care indexes—adjustments that would carry the discussion into the field of medical care expenditures and away from the topic of prices.

Consumers' Price Index and Price Indexes for Medical Care for Moderate-Income Families in Large Cities for 1949-1951 (1935-1939=100)

	1949	1950	1951
Consumers' Price Index (cost of living)	170.2	171.9	185.6
Medical care and drugs	144.9	147.9	155.0
I. Medical care, excluding drugs	149.7	153.1	160.9
A. Physicians' fees (general practitioner, *surgeon, specialist)*	137.9	140.0	145.2
1. *General practitioners' fees*	137.7	139.8	145.2
(a) Office visit	139.0	140.9	146.0
(b) House visit	131.4	133.9	138.3
(c) Obstetric case	155.6	157.3	167.3
2. *Surgeons' and specialists' fees*	138.4	140.6	144.3
(a) Appendectomy, adult	134.2	137.4	141.6
(b) Tonsillectomy, child	142.8	144.0	147.2
B. Dentists' fees	150.6	154.3	160.0
1. *Fillings*	150.9	154.5	159.5
2. Extractions	152.9	156.9	164.5
C. Optometrists' fees, eyeglasses	127.6	128.9	134.0
D. *Hospital rates*	226.8	235.3	260.7
1. *Men's pay ward*	253.5	265.5	297.2
2. *Semiprivate room*	221.7	229.6	253.6
3. *Private room*	207.7	213.7	233.7
E. Group hospitalization*	.....	.....	103.1
II. Prescriptions and drugs	123.3	124.8	128.4
A. Prescriptions	137.1	140.7	147.7

* December, 1950 = 100.

Source: for Consumers' Price Index, Monthly Labor Review, Bureau of Labor Statistics, U. S. Department of Labor, 74:615 (May) 1952; for medical care price indexes 1949-1951, Indexes *of Retail Prices of Apparel,* Housefurnishings, and Services and Miscellaneous Goods to Moderate-*Income Families in Large Cities,* Quarterly Reports, Bureau of Labor Statistics, U. S. Department of Labor, March, 1950, December, 1951, and March, 1952.

The index of hospital prices is for room rates only. The prices of laboratory and other services are not sampled. The index of prescriptions and drugs does not cover any of the "wonder drugs" of recent years. Realizing the necessity for adding such items and for reflecting certain population changes, the Bureau of Labor Statistics has undertaken a thorough revision of its price index series. The data presented in this article have undergone an interim revision. The complete revision, however, will be presented in the indexes published in 1953. These data will have a new base period, 1947-1949. When the new indexes, which are expected to be more representative of today's cost of living, are issued, the Bureau of Medical Economic Research will present an explanation of the revision and a more complete analysis of medical prices.

Director, Bureau of Medical Economic Research.

[1] *Bulletin* 62, Comparative Increases in the Costs of Medical Care and in the Costs of Living, 1948; *Bulletin 66,* The Cost and Quantity of Medical Care in the United States, 1948; Medical Care Prices vs. Cost of Living, J. A. M. A. **139**: 591 (Feb. 26) 1949, reprinted in Bulletin 67, Four Recent *Articles on Medical* Economics, 1949; Medical Care Prices vs. Cost of Living, ibid. **143**: 1198 (July 29) 1950, reprinted as Bulletin 76, 1950; Medical Care Prices and the Cost of Living, ibid. **147**: 258 (Sept. 15) 1951, reprinted as Bulletin 86, 1951; *Bulletin* 87, Medical Care Expenditures, Prices and Quantity, 1930-1950, 1951. All are publications of the Bureau of Medical Economic Research, American Medical Association.

BUREAU OF LEGAL MEDICINE AND LEGISLATION

MEDICOLEGAL ABSTRACTS

Blood: Admissibility of Evidence Relating to Blood Type.—The defendant was convicted on a charge of second degree murder and appealed to the supreme court of New Jersey. The defendant was accused of having stabbed the deceased through the heart with a bread knife and then pushing her through a plate glass window, and the issue arose as to whether the fatal wounds were actually caused by the knife or the broken glass. He was arrested and, prior to his trial, was examined by a physician associated with the Board of Health who extracted a specimen of blood for laboratory analysis to determine the presence or absence of venereal disease. The Board of Health, after making these tests, turned over to the police authorities an unused portion of the blood, which was analyzed by them to determine the defendant's blood type. On appeal, the defendant challenged the state's right to use his blood, which it is contended was taken against his will and without his consent, as evidence against him at his trial to show that it was of the same type as that found on the handle of the knife in question. The basis for this challenge was that such an act was a violation of the defendant's privilege against self incrimination, a denial of the process of law, an unreasonable search and seizure, and a violation of the state and federal constitutions.

The defendant alleged that he never gave his blood with the understanding that it was to be used against him at his trial for murder, and, before a consent to such use could be spelled out, the state must show affirmatively that he had full knowledge of all the facts when he permitted his blood to be taken. Although the identical question raised here has never been passed on in this state, said the supreme court, it has been decided in a recent Pennsylvania case. In that case the blood of the accused had been taken without his consent for typing, and he was subsequently convicted of assault and robbery with intent to commit rape. The defendant's blood type was presented at the trial, and objection was made that this violated the defendant's privilege against self incrimination. The Pennsylvania court said that in other jurisdictions it has been uniformly held that the constitutional immunity from self incrimination does not apply to a compulsory examination to determine the prisoner's physical or mental condition for the purpose of testifying in regard thereto, provided, of course, that he be not compelled to answer any questions propounded to him by those making the examination, since the purpose of the immunity is to prohibit the compulsory oral examination of the prisoner, either before or at trial, to prevent his being required to incriminate himself by speech or the equivalent of speech. Taking into consideration the origin and history of the rule of privilege against self incrimination, its scope and purpose and the protection to be afforded thereby, said the supreme court of New Jersey, we have no hesitancy in subscribing to the doctrine enunciated by the above case and other cases that permitted the use in evidence of photographing and fingerprinting and the results of mental examinations. The evidence relating to the defendant's blood type, concluded the court, was not a denial or invasion of the defendant's common law, constitutional, or statutory right or privilege against self incrimination. It was also contended that the taking of the blood against his will for the purpose of using it in evidence was a denial and an invasion of his rights against unreasonable search and seizure. It would seem, said the court, that after arrest is made the person of the accused may properly be examined without a search warrant in order to find evidence of his guilt and that such an examination would not be deemed an unreasonable search. The defendant's further contentions that the state's conduct also violated due process of law and the state and federal constitutions were also overruled, and the use in court of evidence of the defendant's blood type was approved. Accordingly, the judgment of conviction was sustained. *State v. Alexander, 83 A. (2d) 441 (New Jersey, 1951).*

MEDICAL MOTION PICTURES

Seizure: The Medical Treatment and Social Problems of Epilepsy: 16 mm., black and white, sound, showing time 45 minutes. Produced in 1951 by and procurable on loan from Presentation Division, Veterans Administration, Washington, D. C.

This film portrays the problems of a man in whom epilepsy develops following combat experience in the army. In spite of a prepossessing personality, an understanding wife, and other important advantages, his difficulty in holding jobs reduces him to despair and he returns to the Veterans Administration for help. The first 5 or 10 minutes of the film, and many of the later portions, are superb in their sincerity and emotional impact.

There is a scene near the end in which a rather immature physician is trying to comfort the defeated man and the physician seems unable to grasp what really has hurt and worried him. In trying to reassure his patient regarding the inheritability of epilepsy, he fails to emphasize one definite point that should always be made: that epilepsy resulting from physical trauma, chemical poisoning, and organic lesions is not inheritable. This would relieve the worries of a large group of patients, and he could go on to explain to the rest how the need for exact diagnosis justifies the formidable program of history taking, examining, and testing to which the patient is being subjected. At the end, the film bogs down entirely. Surely something has been learned by this time about safe occupations for persons with epilepsy in or out of institutions, and the vocational adjustments that have been found feasible should be suggested by a swift series of brief closing shots.

The photography generally is satisfactory. The animated sequences explaining causes of seizures are well done. The sound on the print reviewed was not consistently of high quality. As the film stands now, with its depressing close, it can be recommended to research workers in the medical and social sciences as a stimulus to better handling of the epileptic patient's problems. If it were provided with a positive, constructive ending it could be shown with telling effect to any mature audience, both lay and professional.

A New Beginning: 16 mm., black and white, sound, showing time 34 minutes. Presented by the United States Army with the cooperation of the Institute of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center, the United Mine Workers of America Welfare and Retirement Fund, the J.O.B. Committee. Produced in 1951 by Unity Films, Inc., New York. Procurable on purchase from United World Films, Inc., 1445 Park Ave., New York 29.

This documentary film tells the story of Charles Moore, a paraplegic coal miner, whose back was broken in a mine accident. After 10 years of helplessness and inactivity, the United Mine Workers Welfare and Retirement Fund provided means for his rehabilitation. The film shows how Moore, through long and arduous work at the Institute of Physical Medicine and Rehabilitation of the New York University-Bellevue Medical Center, manages to regain a remarkable degree of independence and usefulness. After his training is completed, he obtains a job, "working alongside other men," doing equal work for equal pay.

This is not a training film. Rather, it shows the layman and the physician uninitiated in physical medicine and rehabilitation the processes of rehabilitation in a general way. For that reason it does not cover the details of technique nor the professional aspects of the medical and surgical care of the paraplegic patient. It will be of value in inspiring any disabled person to attempt rehabilitation. The photography and narration are excellent. Except for the fact that it is a bit slow moving at the beginning and at times is overdramatized, the story is well told. The primary audience for this film is the disabled person, but it should also be of interest to any adult who is not familiar with the advantages of physical medicine and rehabilitation.

MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A.M.A. Am. Journal Diseases Child., Chicago

83:421-572 (April) 1952

*Herpes Simplex Virus as Cause of Fulminating Visceral Disease and Hepatitis in Infancy: Report of Eight Cases and Isolation of Virus in One Case. W. W. Zuelzer and C. S. Stulberg.—p. 421.
Corticotropin (ACTH) in Treatment of Acute Laryngotracheitis. J. A. Turner and E. A. Morgan.—p. 440.
*Kerosene Intoxication. R. B. Olstad and R. M. Lord Jr.—p. 446.
Accelerated Rehabilitation in Rheumatic Fever. A. J. Simon, I. Mack and P. Rosenblum.—p. 454.
Prematurity in the Negro Infant. E. P. Crump, C. Wilson-Webb and M. B. Pointer.—p. 463.

Herpetic Hepatitis in Infancy.—The virus of herpes simplex caused herpes viremia in five newborn infants who did not have pronounced cutaneous lesions. The diagnosis was supported by clinical evidence and postmortem studies, and by isolation and identification of the virus from the liver of one infant whose signs and symptoms were the same as those of the others. The clinical picture of this disease is determined in part by the portal of entry, which may be the conjunctiva, oral mucosa, or skin. Exposure may occur through placental transmission or during passage through an infected birth canal. A systemic reaction to the infection occurs on the fifth to seventh day of life, with temperature elevation or hypothermia, increasing jaundice, lethargy, respiratory distress, vomiting, dyspnea, cyanosis, circulatory collapse, and enlargement of the liver and sometimes the spleen. Characteristic symptoms are accumulation of yellow mucus in the pharynx, a severe bleeding tendency, and hypoprothrombinemia. The essential features of herpes lesions in the internal organs are necrosis and typical intranuclear inclusions. Even if maternal antibodies are present, premature infants are particularly susceptible to this infection, possibly because of a special susceptibility of embryonic tissues. Specific herpetic hepatitis was observed in three older infants (3 weeks, 22, and 18 months old, respectively.) The virus of herpes simplex might have invaded the blood stream of these infants during herpetic stomatitis. It is suggested that viremia is probably a frequent, if not a regular, phenomenon during primary herpetic infections of infancy.

Kerosene Intoxication.—Kerosene intoxication is reported in 71 infants and children. The amount of oil ingested was not always known, but the recorded amounts varied from 5 to 240 cc. Respiratory symptoms were found in all patients, gastrointestinal symptoms in 35, central nervous symptoms in 28, leukocytosis in 39, elevation of temperature in 53, and pneumonia in 29. Proteinuria and/or glycosuria was present in 37 of the 57 patients whose urine was examined. Roentgenograms were taken of 44 patients, 26 of whom had positive evidence of pneumonia in the lower lobes. In 19 patients with positive roentgenographic findings no positive auscultatory findings were recorded. In general, although the period of hospitalization was affected considerably by the presence of pneumonia and fever, it was proportional to the time that had elapsed from ingestion of kerosene to admission. The use of a stomach tube and copious lavage are recommended. The incidence of pneumonia was lower among the children who had vomited and had also received lavage. None of these patients died, but in 325 previously reported cases the mortality rate was 5.85%. Since most of these cases were due to accidental ingestion of kerosene in homes with kerosene stoves, a good preventive measure is instruction of families with this type of stove concerning its potential dangers.

A.M.A. Arch. Dermat. and Syphilol., Chicago

65:261-378 (March) 1952

Local Depilatory Action of Some Unsaturated Compounds. P. Flesch and M. Hunt.—p. 261.
Comparative Study of Histology and Cytology in Vesiculating Eruptions. L. H. Winer and C. E. Lipschultz.—p. 270.
Correlation of Emotional Status and Reactivity to Cutaneous Stimuli in Functional Dermatoses. M. E. Obermayer, R. Blair, C. Fiske and others.—p. 291.
Hidradenitis and Other Variants of Acne. H. A. Brunsting.—p. 303.
*Medical Aspects of Home Cold Waving. M. J. Brunner.—p. 316.
Natural Steroid Complex: Evaluation of Use in Collagen Diseases and Chronic Dermatoses. W. W. Duemling and M. Millman.—p. 327.
Bullous Eruption Due to Pediculosis Pubis. A. B. Kern.—p. 334.
Syringocystadenoma Papilliferum and Nevus Sebaceus (Jadassohn) Occurring as Single Tumor: Report of Case. J. L. Grund.—p. 340.
Effect of Cortisone upon Hypersensitivity Due to Lymphogranuloma Venereum. A. W. Grace, L. Frank and R. J. Wyse.—p. 348.

Medical Aspects of Home Cold Waving.—Almost all home cold wave solutions use ammonium or sodium thioglycolate, or mixtures of the two salts, in aqueous solution. Home cold wave solutions are not primary irritants under normal conditions, but irritant dermatitis can occur and is generally traceable to deviations in the method of application. Occasionally the wave solution is left on overnight without rinsing, or the suggested method of neutralization is incomplete. Irritant dermatitis of the hands from home cold wave solution is seen mainly after repeated contact without protection of the hands, as in women who give one or several home waves weekly to friends or relatives. In these subjects an ordinary "housewives' eczema" due to detergents may become exacerbated, or there may be maceration and exfoliation of the skin of the thumb and forefinger bilaterally, due to mechanical trauma incident to rolling up the plastic curlers while the skin is wet with waving solution. The dermatologist may be called on to investigate patchy or diffuse hair breakage, abnormalities in hair texture, or abnormalities in color or odor. Patchy hair breakage may result from excessively tight winding of the tress on the rod. Diffuse hair loss may result in bleached or otherwise damaged hair when failure to follow directions results in overprocessing, and it is occasionally seen following a severe eczematous dermatitis of the scalp. Discoloration of the hair after waving is seen almost entirely in women with dyed hair. Abnormal hair odor is due basically to inadequate application of neutralizer. The odor is particularly noticeable when the hair is rewetted and may be controlled by repeating neutralization. There were no authentic cases of systemic intoxication following percutaneous application of waving agents in the series covered by this report. Mild irritation may be expected after accidental entry of thioglycolate or neutralizer solutions into the eye, particularly if the agent is not flushed out with water.

A.M.A. Arch. Indust. Hyg. & Occup. Med., Chicago

5:311-414 (April) 1952. **Partial Index**

Studies on Toxicity and Skin Effects of Compounds Used in Rubber and Plastics Industries: I. Accelerators, Activators, and Antioxidants. F. S. Mallette and E. von Haam.—p. 311.
Toxicity of Sulfuric Acid Mist to Guinea Pigs. M. O. Amdur, R. Z. Schulz and P. Drinker.—p. 318.
Hazards from Thermal Decomposition of Motor-Insulating Materials. J. Sendroy Jr., J. D. O'Neal and G. C. Pitts.—p. 330.
Man, Submarines, and Carbon Dioxide. H. J. Alvis.—p. 344.
Disinfective Action of Methyl Bromide, Methanol, and Hydrogen Bromide on Anthrax Spores. R. W. Kolb, R. Schneiter, E. P. Floyd and D. H. Byers.—p. 354.
Influenza-Virus Vaccination in Industry. C. F. Yeager.—p. 365.
The Physician in Industry and the "Alcoholic" Worker. L. J. Wade.—p. 368.
Some Colloidal Properties of Inert Dusts. R. F. Hounam.—p. 375.
Concentrations of Particulates Found in Air. M. W. First and P. Drinker.—p. 387.

Periodicals on file in the Library of the American Medical Association may be borrowed by members of the Association or its student organization and by individual subscribers, provided they reside in continental United States or Canada. Requests for periodicals should be addressed "Library, American Medical Association." Periodical files cover only the last 11 years, and no photoduplication services are available. No charge is made to members, but the fee for other borrowers is 15 cents in stamps for each item. Only three periodicals may be borrowed at one time, and they must not be kept longer than five days. Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted.

A.M.A. Arch. Otolaryngology, Chicago

55:261-404 (March) 1952

Allergy of Upper Respiratory Tract: Cure by Surgery Plus Antiallergic Methods. M. F. Jones.—p. 261.
Symptoms and Treatment of Pituitary Tumors. O. Hirsch.—p. 268.
Herpes Zoster of Cephalic Extremity. F. H. McGovern and G. S. Fitz-Hugh.—p. 307.
Management of Recent Nasal Fractures. S. Fomon, A. Schattner, J. W. Bell and others.—p. 321.
Response of Otosclerosis and Adhesive Otitis to Treatment with Corticotropin. N. Riskaer.—p. 343.
Anesthesia for Long or Technically Difficult Ophthalmologic and Otolaryngologic Surgical Procedures. D. C. Moore and J. F. Tolan.—p. 350.
Role of Allergy in Otology. R. E. Jordan.—p. 363.
*Pathology of Otosclerosis. H. Brunner.—p. 369.
Diagnostic Value of Latency Period After Caloric Stimulation of Vestibular Apparatus. E. Wodak.—p. 381.
Aberrant Salivary Gland Tissue in Tonsil Fossa. B. A. Sanderson.—p. 387.
Salivary Gland and Duct Calculi. J. H. Melant.—p. 389.

Pathology of Otosclerosis.—Based on a 25 year experience the author summarizes the present concept of the pathology of otosclerosis by distinguishing between preotosclerotic and otosclerotic changes as follows: The preotosclerotic changes consist of extensive degeneration of the cartilage at the frame of the windows, and overproduction of both precollagenous substance and primitive bone. The broadened marginal sheaths of the blood vessels that may be observed in the preotosclerotic stage, for which the term "blue mantles" has been coined by Manasse, consist of precollagenous substance. These changes are probably hereditary and do not produce clinical symptoms. They are called "preotosclerotic" for the following reasons: they are frequently associated with actual otosclerosis; they occur also without actual otosclerosis; they are not associated with other diseases of the bone of the labyrinth; and they are, as far as the "blue mantles" are concerned, distributed all over the petrous bone. The preotosclerotic changes turn to actual otosclerosis if there are signs of activity, i. e., definite osteoclasis and/or osteogenesis in these areas. By studying the biology of the otosclerotic focus in addition to its histological features, the author concluded that the actual otosclerotic nidus is a genuine tumor of the bone of the labyrinth capsule and shows various microscopic features according to the stage of activity. As in all tumors, the beginning of the disease is scarcely demonstrable. Although otosclerotic tumors have certain features in common with osteoid osteoma, they differ from these tumors in that they are not self-limited and do not grow by expansion, that is, by pushing aside the adjacent structures. The atrophy of the basilar portion of the cochlea, frequently associated with otosclerosis, is probably caused by the same (unknown) factor that causes the formation of the preotosclerotic changes.

A.M.A. Arch. Pathology, Chicago

53:197-280 (March) 1952

Some Local Factors in Restoration of Rat's Liver After Partial Hepatectomy: I. Glycogen; Golgi Apparatus; Sinusoidal Cells; Basement Membrane of Sinusoids. K. Aterman.—p. 197.
Id.: II. "Watery Vacuolation": Its Relation to Vacuolation of Anoxia. K. Aterman.—p. 209.
Renal and Vascular Lesions Elicited by "Renin" in Rats with Desoxycorticosterone Hypertension. G. M. C. Masson, A. C. Corcoran and I. H. Page.—p. 217.
Intrinsic Hemangiomas of Peripheral Nerves: Report of Two Cases and Review of Literature. E. J. Losli.—p. 226.
Massive Fat Embolism Following Marrow Infarction in Sickle Cell Anemia. J. P. Wyatt and M. D. Orrahood.—p. 233.
Pathologic Lesions in Nervous System of Duck Fed Ration Deficient in Vitamin A. R. H. Rigdon.—p. 239.
Analysis of Hyperchromatism in Histologic Diagnosis of Carcinoma of Cervix Uteri, with Special Reference to Intraepithelial and Invasive Squamous Cell Carcinoma and Squamous Metaplasia. A. G. Foraker.—p. 250.
Senile Fibrosis of Stomach of Albino Rat. F. E. Shaffer and F. R. Dutra.—p. 257.
Extramedullary Lesions in Multiple Myeloma: Review of Literature and Pathologic Studies. D. W. Hayes, W. A. Bennett and F. J. Heck.—p. 262.
Vitamin-A Acceleration of Bone Growth Sequences in Hypophysectomized Rats. S. B. Wolbach and C. L. Maddock.—p. 273.

American Journal of Psychiatry, New York

108:641-720 (March) 1952

Psychological Response to ACTH, Cortisone, Hydrocortisone and Related Steroid Substances. H. P. Rome and F. J. Braceland.—p. 641.
Nor-Epinephrine-Like and Epinephrine-Like Substances in Psychotic and Psychoneurotic Patients. D. H. Funkenstein, M. Greenblatt and H. C. Solomon.—p. 652.
Lipoproteins in General and Cerebral Arteriosclerosis: Preliminary Report. A. Simon, J. W. Gofman, N. Malamud and others.—p. 663.
Recent Biochemotherapeutic Developments in Psychiatry. M. D. Sackler, R. R. Sackler, A. M. Sackler and J. H. W. van Ophuijsen.—p. 669.
The First Traveling Psychiatric Clinic in Quebec: Report of Experiences with One Year's Operation. G. E. Reed and A. Silver.—p. 676.
Sociological Motivations of Delinquency. P. W. Tappan.—p. 680.
Parental Deprivation and "Broken Home" in Dementia Praecox and Other Mental Disorders. J. E. Oltman, J. J. McGarry and S. Friedman.—p. 685.
Application of Principles of Child Psychotherapy in Residential Treatment. O. Krug.—p. 695.

American Journal of Public Health, New York

42:115-232 (Feb.) 1952. Partial Index

Civil Defense from a Health Officer's Viewpoint. H. D. Chope.—p. 115.
Federal Aspects of Health Emergency Planning. M. A. Pond.—p. 119.
Sanitation in Local Civil Defense Program. C. L. Senn.—p. 131.
Methods of Payment for Physicians' Services in Medical Care Programs. F. Goldmann.—p. 134.
Relation Between Recent Injections and Paralytic Poliomyelitis in Children. M. Greenberg, H. Abramson, H. M. Cooper and H. E. Solomon.—p. 142.
Association of Parenteral Injections with Poliomyelitis. R. F. Korns, R. M. Albrecht and F. B. Locke.—p. 153.
Public Health Aspects of Campaign Against Yellow Fever in Central America. N. W. Elton.—p. 170.
Further Studies of New Serologic Test in Tuberculosis. E. R. Maillard.—p. 175.
Food Plant Sanitation. P. E. Laughlin.—p. 182.

42:233-352 (March) 1952. Partial Index

Biological Warfare Defense: I. Introductory Remarks. W. L. Wilson.—p. 233.
Id.: II. Epidemic Intelligence Service of Communicable Disease Center. A. D. Langmuir and J. M. Andrews.—p. 235.
Id.: III. The State Sanitary Engineer's Viewpoint. J. M. Hepler.—p. 239.
Coccidioidin Sensitivity Among School Children in Phoenix (Skin Test and X-Ray Survey). J. Emmett.—p. 241.
Multiple Screening. W. G. Smillie.—p. 255.
*Accidental Carbon Monoxide Poisoning Due to Domestic Gas Appliances and Gas Refrigerators: Problem in New York City and Its Control. J. B. Trichter and M. Helpern.—p. 259.
Measuring Quality of Medical Care Through Vital Statistics Based on Hospital Service Areas: I. Comparative Study of Appendectomy Rates. P. A. Lembcke.—p. 276.
Medical and Public Health Man Power. J. G. Wilson.—p. 287.

Carbon Monoxide Poisoning Due to Domestic Gas Appliances.—Carbon monoxide poisoning has increased in recent years as the result of incomplete combustion of illuminating gas in gas flame refrigerators. The users of gas flame refrigerators are not generally aware that such appliances require periodic adjustment and servicing. They may appear to be operating satisfactorily, without odor, and with a gas flame of the proper color, and at the same time may be discharging dangerous amounts of carbon monoxide. Spot surveys of water-cooled refrigerators in New York City, now fueled with mixed gas and formerly supplied with manufactured gas, have shown that they may be a grave hazard. Of 330 water-cooled refrigerators tested, 156 (59%) were producing dangerous amounts of carbon monoxide. Of 745 air-cooled models, 304 (41%) were operating improperly or were producing dangerous amounts of carbon monoxide. These tests were carried out after the change from manufactured to mixed gas. Carbon monoxide poisoning from gas refrigerators usually occurs more slowly than poisoning due to other gas appliances. With the slow accumulation of carbon monoxide in the blood, the victim usually has headache, dizziness, and muscular weakness. Even before mental confusion develops, the victim may become nauseated and vomit. Mental confusion and helplessness becomes extreme. The victim staggers about; he may overturn furniture and injure himself on broken utensils, frequently leaving a trail of blood stains. In many cases the circumstances give rise to a suspicion of homicide. Health departments and medical examiners' offices should assume leadership in combatting carbon monoxide poisoning resulting from use of gas appliances in the home, and the resources of all official agencies should be utilized.

Am. Practitioner & Digest of Treatment, Philadelphia

3:265-348 (April) 1952

*Treatment of Temporal Arteritis and Other Vascular Headaches: Use of Procaine Infiltration and Surgical Excision. J. W. Fleming and L. A. Grossman.—p. 265.
Pediatric Problems of Prognostic Importance. W. M. Block.—p. 272.
*Use of Anion-Cation Exchange Resin in Edematous States Contraindicating Mercurial Diuretics. M. M. Best.—p. 274.
Clinical Implications of "Infantile Personality." H. H. W. Miles, H. C. Shands and S. Cobb.—p. 280.
Some Diagnostic Problems of Pleural Effusions. J. B. Andosca.—p. 285.
Effective Therapy of Severe Penicillin Reactions with Sodium Dehydrocholate: Clinical Studies of Liver Dysfunction—XII. L. Pelner and S. Waldman.—p. 293.
Prescribing Physical Treatment in General Practice. O. F. von Werssowetz.—p. 297.
Clinical Physiology of Kidney. G. E. Wakerlin.—p. 302.
Use of Experimental Suppositories in Treating Refractory Migraine. E. E. Reisman Jr.—p. 308.
"Restricted Activity." W. T. Gibb Jr.—p. 311.
Management of Carcinoma of the Female Breast. L. M. Trauner.—p. 315.

Treatment of Vascular Headaches.—Two patients, aged 70 and 57, with the uncommon clinical syndrome termed "temporal arteritis" are described. This syndrome is characterized by severe throbbing headache, swelling, and often nodularity of the temporal artery, with erythema of the overlying skin and pain on mastication. There is usually a systemic reaction, with temperature elevation, malaise, muscular aching, anorexia, and weight loss. Blindness due to involvement of the retinal artery may occur in one-third of the cases, as it did in one of the authors' two patients. The pathological picture is one of panarteritis, with necrosis of the media and formation of granulomatous tissue containing foreign body giant cells and epithelioid cells. The intima becomes thickened, and thrombosis is sometimes present. Blindness persisted, but all other manifestations subsided after an illness of two months' duration in the older patient. In the younger patient complete relief of pain and headache promptly followed periarterial infiltration with 1% procaine hydrochloride. It was necessary to repeat this procedure several times, the relief obtained lasting six to eight hours. Nevertheless increasing erythema and brownish discoloration were noted over the right temporal artery, and brief periods of irrational behavior were observed. A 2 cm. segment of the right temporal artery was then excised. Microscopic study of the vessel showed only thickening of the intima. Recovery was considered complete, since the patient remained asymptomatic during a two year follow-up period. Five additional patients are described who were between the ages of 23 and 44 years; they had vascular, usually unilateral, headache of relatively localized origin, with an afebrile course. A small area of hyperesthesia was observed over a segment of the temporal or other extracranial arteries. This headache was dramatically relieved by periarterial procaine hydrochloride infiltration. It is concluded that infiltration with procaine hydrochloride is a valuable therapeutic aid in treating certain headaches, regardless of their etiology. Surgical excision of segments of involved extracranial arteries may be indicated in some cases.

Treatment of Edema with Anion-Cation Exchange Resins.—Of eight patients with moderate to severe edema in whom organic mercurial diuretics were ineffective or contraindicated, seven were treated with a mixture of anion and cation exchange resins of the carboxylic type. This mixture contained approximately 12% anion exchanger, 29% potassium cycle cation ion exchange resin, and 59% hydrogen cycle cation exchange resin. One patient received two types of cation exchange resins; she was first given a resin consisting of 80% ammonium cycle and 20% hydrogen cycle and later the hydrogen-potassium cycle resin. Each resin was administered in equally divided amounts at 10 a. m., 3 p. m., and bedtime to a total of 45 gm. per day. The anion-cation exchange resin mixture was effective in restoring responsiveness to organic mercurial diuretics in three of five patients. The two unresponsive patients may have had edema unassociated with sodium retention. Typical symptoms and electrocardiographic changes suggestive of potassium depletion developed in the patient in whom the two types of cation exchangers were used. Acidosis and over-depletion of sodium and potassium are the commonest hazards associated with the use of any cation exchange resin. In one patient with intercapillary glomerulosclerosis, the cation resin administered alone caused such a rapid decrease in the carbon dioxide combining power that its further use was considered inadvisable. The anion-cation mixture resulted in less acidosis and better control of edema. The presence of renal disease need not be considered an absolute contraindication to resin therapy. Periodic laboratory determinations of serum sodium, potassium and chloride levels, and carbon dioxide content or combining power, are desirable in all patients and essential in those with preexisting renal disease. It would seem that the use of cation exchange resins, as adjuvants to an anion exchanger, will assume a prominent place in the management of selected cases of edema, since sodium depletion can be achieved and the action of organic mercurial diuretics potentiated or restored by the use of these resins.

Annals of Internal Medicine, Lancaster, Pa.

36:751-932 (March) 1952

*Effect of Cortone and ACTH on Histopathologic Lesions of Rheumatoid Arthritis. B. M. Norcross, L. M. Lockie, A. G. Constantine and others. —p. 751.
Benign and Pathologic Albuminuria: Study of 600 Hospitalized Cases. S. E. King and C. Gronbeck Jr.—p. 765.
Mechanism Involved in Polyneuritis as Exemplified by Postdiphtheritic Polyneuritis. M. R. Brown.—p. 786.
Effects of Intravenously Administered Veratrum Viride in Hypertensive and Normotensive Subjects. A. P. Shapiro and E. B. Ferris.—p. 792.
Comparative Study of Effects of Veratrum Viride and Tetraethylammonium Chloride in Hypertension. A. P. Shapiro, A. A. Brust and E. B. Ferris.—p. 807.
Choice of Precordial Leads in Diagnosis of Coronary Artery Disease. M. Pomerance, J. B. Hoffman and G. J. Ashe.—p. 811.
*Diffuse Interstitial Fibrosis of Lungs. E. H. Rubin, B. S. Kahn and D. Pecker.—p. 827.
Hemopneumothorax: Reëvaluation of Treatment. G. A. Beatty and R. W. Frelick.—p. 845.
*Nocardiosis: Three Case Reports. I. L. Bernstein, J. E. Cook, H. Plotnick and F. J. Tenczar.—p. 852.
Cardiac Asthma: Its Pathogenesis and Response to Aminophylline. C. Schuman and H. G. Simmons.—p. 864.

Effect of Cortisone and Corticotropin on Lesions of Rheumatoid Arthritis.—Muscle biopsies were performed on 30 patients with rheumatoid arthritis prior to and during hormone therapy. Both inflammatory and degenerative lesions were found in the muscles of 18 of the 30 patients, but in only 11 did the changes seem suitable for the evaluation of hormonal therapy. In 8 of the 11 patients there was definite improvement in both types of lesions following administration of cortisone (cortone®) and corticotropin (ACTH). The inflammatory nodules disappeared in five and were improved in the other three patients. Degenerative muscle changes improved less than the inflammatory lesions in eight patients. The greater response of the inflammatory lesions seems understandable in view of the short periods of treatment. Subcutaneous rheumatoid nodules were altered considerably in the 12 patients in whom they were studied during the period of hormone administration. Many small nodules almost disappeared, while the large nodules became smaller, less painful, and less inflamed. There was a definite histological change in two of the three patients from whom comparable nodules were removed before and during therapy. Synovial inflammation decreased in both patients in whom articular biopsies were performed before and during hormone therapy. No delay occurred in the clinical healing of the surgical wounds of this group of patients during cortisone or corticotropin administration.

Diffuse Interstitial Pulmonary Fibrosis.—In 1944 four cases were reported of a disease characterized by relatively sudden onset of dyspnea, cough, cyanosis, elevation of temperature, and early signs of cardiac decompensation. Death resulted within a short time from acute asphyxia or heart failure. At postmortem examination the lungs contained diffuse interstitial fibrotic changes. One similar case had been described in 1940 and subsequently reported cases, including the one reported here, bring the total to 14. The patients included six men and eight women ranging in age from 21 to 68 years. The main symptoms were cough, expectoration, hemoptysis, chest pain, fever, dyspnea, and cyanosis. In a few cases the onset was insidious, but in most it was acute. With the appearance of dyspnea and signs of heart failure, the disease progressed rapidly, with death resulting from suffocation or cardiac decompensation. The duration varied from one month to three and a half years. Clubbing of fingers and toes was noted in several cases. Miliary, nodular, or mottled infiltrations, involving diffusely both lungs, were seen frequently, and often were associated with conglomerate areas of consolidation and occasionally pleural effusion. The differential diagnoses included tuberculosis, atypical pneumonia, chronic pulmonary infection, pulmonary edema with pneumonia or cardiac disease, pulmonary manifestations of systemic disease, such as blood dyscrasia, amyloidosis, and allergic conditions, as well as metastatic neoplasm. In the case described here roentgenographic findings suggested sarcoidosis. At autopsy diffuse interstitial fibrosis of both lungs was found. The progressive nature of the disease was indicated by the presence of different stages of the fibrotic reaction in various parts of the lungs. Associated cardiac enlargement affected chiefly the right heart. It is suggested that the disease is caused by a virus. Recovery may take place in some cases, but this cannot be proved, since the diagnosis depends on a complete autopsy.

Three Cases of Nocardiosis.—Nocardiosis is an acute or chronic infectious disease of granulomatous or suppurative nature, caused by an aerobic, gram-positive, inconstantly acid-fast species of Actinomycetaceae. Nocardia asteroides is the organism most frequently isolated. The authors add 3 cases to the 43 recorded in the literature. In case 1 the diagnosis was established before death, but treatment was ineffectual. Whether the preexisting silicosis predisposed to nocardiosis can only be conjectured. Case 2 illustrates the frequent association of pulmonary and neurological manifestations; in contrast to case 1, antemortem studies failed to reveal the etiological agent. The third patient was cured, and there has been no evidence of recurrence during a four year follow-up. The similarity of the signs and symptoms to those of pulmonary tuberculosis was striking. Since nocardiosis may mimic pulmonary tuberculosis, it is important to know that fragmented acid-fast mycelia in sputum may be mistaken for Mycobacterium tuberculosis. In the cases reported to date, the organisms have been more sensitive to sulfadiazine than to antibiotics. Surgical drainage should be employed when indicated.

Annals of Western Medicine & Surgery, Los Angeles

6:131-194 (March) 1952

Management of Gastric Ulcer. W. H. Bachrach.—p. 145.
*Baseball or Mallet Finger. L. Cozen and A. Waxman.—p. 154.
Infectious Mononucleosis and Guillain-Barré Syndrome: Report of Case with Recovery. W. F. Owen Jr.—p. 156.
Struma Ovarii: Report of Case. E. A. Pearson.—p. 157.
Psychosomatic Aspects of Early Postoperative Ambulation. N. Shure. —p. 159.
Varicose Veins: Part VII. Differential Diagnosis of Varicose Ulcers. A. Fields.—p. 162.
Medical Malpractice: What About Experimentation? E. L. Cady Jr. —p. 164.
Systematic Approach to Identification of Toxic Organic Compounds Isolated from Tissues and Fluids: Part III. Aquo-Ferriate and Stannic Chloride Tests. C. J. Umberger and A. Stolman.—p. 171.
Management of Gastric Hemorrhage and Hematemesis. R. G. Majer. —p. 175.
Domiciliary Care of Epilepsy. J. W. Shuman.—p. 179.

Baseball or Mallet Finger.—Avulsion of the distal attachment of an extensor tendon may result from a blow to the tip of the finger, forcing the finger into sudden flexion, or of a blow to the dorsum of the finger, injuring the extensor aponeurosis of the distal phalanx. A chip of bone may be detached with the end of the tendon. The trauma may be caused by a ball striking the end of the finger (hence the term "baseball finger"), and another name for the condition is "mallet finger." Two closed methods of treatment have been described. One is to place a long splint, such as a Lewin splint, on the finger with both interphalangeal joints extended. Usually, however, since the flexor tendons are so much stronger than the extensor tendons, the splint fails to keep the extensor relaxed enough to effect a union of the torn aponeurosis. The other, described by Smillie and by Watson-Jones, employs a plaster cast. This is applied to the finger with the proximal interphalangeal joint flexed and the distal interphalangeal joint extended. It has great merit theoretically, but unfortunately, the cast has the disadvantage of sliding on the finger. The method with which these authors obtained good results in all of nine cases in which it was used utilizes a very short splint, which must not extend proximally farther than the interphalangeal joint. This joint will remain flexed, since a finger that is not used is constantly in the position of relaxation, in this case, flexion. Thus the goal of Smillie and of Watson-Jones is achieved in that the proximal interphalangeal joint is in flexion while the distal interphalangeal joint is in extension. Since the splint is short, it can be worn with little discomfort for the eight weeks necessary to insure union of the aponeurosis. The patient's comfort is increased by removal of the splint at two week intervals. The only one of the nine patients who did not obtain full extension did not start to wear the splint until three weeks after injury, yet even he obtained a satisfactory result.

Blood, New York

7:289-386 (March) 1952

Studies on Platelets: III. Absence of "Selective Sequestration" and Destruction of Platelets by Spleen in "Idiopathic" Thrombocytopenic Purpura. M. Stefanini, J. B. Chatterjea, W. Dameshek and others. —p. 289.
Metabolic Displacement of Folic Acid by Aminopterin: Studies in Leukemia Patients. M. E. Swendseid, A. L. Swanson, S. Miller and F. H. Bethell.—p. 302.
*Nutritional Status of Folic Acid in Persons with Leukemia and Its Possible Relation to Effects of Aminopterin Therapy. M. E. Swendseid, A. L. Swanson, M. C. Meyers and F. H. Bethell.—p. 307.
Roles of Prothrombin Activity, Heparin-Protamine Titer and Platelet Concentration in Bleeding of Leukemia. G. Freeman and J. S. Hyde. —p. 311.
Certain Endocrine Influences Governing Leukocytic Response to Fever. W. L. Hubler, G. M. Higgins and J. F. Herrick.—p. 326.
Blood Destruction in Polycythemia Induced by Hypoxia. K. R. Reissmann, W. L. Burkhardt and B. Hoelscher.—p. 337.
Chronic Hemolytic Anemia Associated with Thalassemia and Sickling Traits. P. Sturgeon, H. A. Itano and W. N. Valentine.—p. 350.
Intravenous Use of High Molecular Ferric Carbohydrate Compound in Treatment of Hypochromic Anemia. J. E. Lucas and A. B. Hagedorn. —p. 358.
Drying of Hemoglobin. W. E. Smith and R. B. Pennell.—p. 368.
Effect of Dextran on Blood Typing and Crossmatching. P. Roche Jr., R. A. Dodelin and W. L. Bloom.—p. 373.
Influence of Dimethylaminoazobenzene on Leukocyte Production in Rat. J. S. Dinning, L. D. Payne and P. L. Day.—p. 376.

Folic Acid in Leukemia.—Reports on the use of metabolic antagonists of folic acid in leukemia revealed that in some patients a partial, temporary remission follows the administration of a folic acid antagonist. These remissions occur for the most part in patients with acute leukemia, particularly those in the younger age group, whereas the results of treatment in chronic leukemia are in general unsatisfactory. Moreover, in patients with chronic leukemia, the administration of an antagonist is usually accompanied by early severe toxic manifestations such as erythema, stomatitis, hemorrhage, and anorexia. Therefore, it appears that a general systemic factor that determines the individual response to folic acid antagonists must vary in different types of leukemia. It seemed possible that the metabolic availability of folic acid itself might be such a factor, and an attempt was made to determine the nutritional status with respect to folic acid in cases of chronic and acute leukemia. Folic acid excretion following test doses of the vitamin was measured in 31 cases of leukemia. Evidence was obtained that depletion of body stores of folic acid occurs in many cases of leukemia. It is suggested that the patient with leukemia has an increased requirement for the vitamin due to his hypermetabolic state, or perhaps more precisely, to the production of large numbers of immature leukocytes with relatively high folic acid concentrations. Since patients with chronic leukemia more commonly show evidence of greater folic acid depletion than those with acute leukemia, it appears that duration of the disease may be a factor in exhausting tissue reserves of the vitamin. The degree of folic acid nutritional depletion seems related to the varying clinical results obtained with antagonist therapy in chronic and acute leukemia.

Cancer Research, Chicago

12:165-242 (March) 1952

Numerical Proportions of Different Cells in Mouse Epidermis During Early Methylcholanthrene Carcinogenesis. B. Davibhadhana.—p. 165.
Effect of Irradiation on Universal Reaction in Cancer. R. L. Kahn, F. J. Hodges, I. Lampe and O. W. Doyle.—p. 170.
Effect of Irradiation on Universal Reaction in Polycythemia Vera. R. L. Kahn, W. H. Bullock and F. H. Bethell.—p. 176.
Differential Invasion of Embryonic Chick Tissues by Mouse Sarcomas *180 and 37.* J. Kautz.—p. 180.
Incorporation and Turnover of Radiophosphorus in Mouse Mammary Tumors (dbrB and C3H). H. Albaum, A. Goldfeder and L. Eisler. —p. 188.
Progressive Microscopic Alterations in Livers of Rats Fed Hepatic Carcinogens 3′-Methyl-4-dimethylaminoazobenzene and 4′-Fluoro-4-dimethylaminoazobenzene. J. M. Price, J. W. Harman, E. C. Miller and J. A. Miller.—p. 192.
Tumor Incidence and Lethal Mutation Rate in Drosophilia Treated with 20-Methylcholanthrene. W. J. Burdette.—p. 201.
Comparative Effects of Various Steroids on Lymphoid Tissue of Rat. W. L. Money, J. Fager and R. W. Rawson.—p. 206.
Protective Action of Stock Diets Against Cancer-Inducing Action of 2-Acetylaminofluorene in Rats. R. W. Engel and D. H. Copeland. —p. 211.
Studies on Methylcholanthrene Induction of Tumors in Scorbutic Guinea Pigs. W. O. Russell, L. R. Ortega and E. S. Wynne.—p. 216.
Influence of Blockage of Nipples on Occurrence of Hyperplastic Nodules in Mammary Glands of C3H Mice. E. Fekete, C. C. Little and F. L. Richardson.—p. 219.
Neoplasia Induced in Rat Embryos by Roentgen Irradiation. J. G. Wilson, R. L. Brent and H. C. Jordan.—p. 222.
Comparative Uptake of Free Amino Acids by Mouse-Ascites Carcinoma Cells and Normal Tissues. H. N. Christensen and M. E. Henderson. —p. 229.
Inhibition of Pasteur Effect in Yeast by Tumor Extracts and Differences in Lability of Sulfur in Normal and Tumor Tissues. D. Ghosh and H. A. Lardy.—p. 232.
Failure to Observe Pentolysis by Serum of Rats Bearing Malignant Tumors. J. H. Roe, J. W. Cassidy, A. C. Tatum and E. W. Rice. —p. 238.

Industrial Medicine and Surgery, Chicago

21:147-196 (April) 1952. **Partial Index**

*Pole Top and Other Manual Resuscitation Methods—Comparison Study. A. S. Gordon, M. S. Sadove, F. Raymon and A. C. Ivy.—p. 147.
Sequelae in Case of Electric Shock. L. H. Cotter and B. H. Cotter. —p. 153.
Ultrasound and Industrial Medicine. H. O. Parrack.—p. 156.
Standards of Noise Tolerance. J. H. Sterner.—p. 165.
Industrial Loss of Hearing: Medical Aspects. C. S. Nash.—p. 171.
Medical Care in Surface Transportation Company. H. Brandaleone and G. J. Friedman.—p. 174.
Therapeutic Use of Oxygen in Industrial Medicine. T. W. Nale.—p. 179.
Use of Hemo-Irradiation (Knott Technique) in Eye Infections. D. F. Farmer, C. P. Sullivan and W. B. Sullivan.—p. 183.

Resuscitation Methods.—Gordon and associates studied resuscitation methods on five men made apneic by means of intravenous thiopental (pentothal®) sodium and *d*-tubocurarine chloride. In pole top resuscitation, the rescuer ascends to the victim. He secures his safety strap around the pole and places the victim astride it between himself and the pole. He encircles with his arms the waist of the victim, placing both hands on the abdomen, thumbs below the lower ribs and fingers touching. With his arms and hands, he compresses the victim's abdomen in an upward motion. At the finish of the stroke, the hands are cupped with the fingers depressing the abdomen under the rib margin. The pressure is quickly released and reapplied 12 to 15 times per minute. The pole top method of resuscitation gives adequate ventilation, and should be applied to linemen who are shocked on the poles. However, since the vertical position predisposes to peripheral stasis and circulatory embarrassment, and impedes drainage of fluids that may collect in the oropharynx, the victim should be lowered to the ground and one of the more efficient push-pull methods begun as soon as he can be placed in the prone position. Comparative tests of the manual resuscitation techniques revealed that push-pull methods are two to three times as efficient, as regards pulmonary ventilation, as either the push or the pull methods alone. For general purposes the arm lift, back pressure method (modification of the Danish Nielsen method) should be used, since it is easy to teach, can be performed by most operators for prolonged periods, and gives excellent pulmonary ventilation without circulatory embarrassment. Trained personnel should also be acquainted with the hip lift, back pressure and Silvester arm lift, chest pressure methods to be used as alternatives in special circumstances.

Journal of Clinical Investigation, Cincinnati

31:131-244 (Feb.) 1952

Hemodynamic Alterations in Acute Myocardial Infarction: I. Cardiac Output, Mean Arterial Pressure, Total Peripheral Resistance, "Central" and Total Blood Volumes, Venous Pressure and Average Circulation Time. E. D. Freis, H. W. Schnaper, R. L. Johnson and G. E. Schreiner.—p. 131.
Determination of Thyroidal and Renal Plasma I^{131} Clearance Rates as Routine Diagnostic Test of Thyroid Dysfunction. S. A. Berson, R. S. Yalow, J. Sorrentino and B. Roswit.—p. 141.
Absorption and Excretion of Tocopherol in Laennec's Cirrhosis. G. Klatskin and D. W. Molander.—p. 159.
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Relationship of Streptolysin S Inhibitor to Phospholipids in Serum of Human Beings and Experimental Animals. G. H. Stollerman, B. B. Brodie and J. M. Steele.—p. 180.
Alveolar-Arterial Oxygen Pressure Gradient in Anemia. J. M. Ryan and J. B. Hickam.—p. 188.
Incorporation of C^{14} Glycine into Protein by Human Liver Slices. C. M. Grossman and J. D. Hauschildt.—p. 192.
Distribution of Sodium and Potassium in Man. N. Deane and H. W. Smith.—p. 197.
Distribution of Total Body Chloride in Man. N. Deane, M. Ziff and H. W. Smith.—p. 200.
Oxygen Tension of Tissues by Polarographic Method: IV. Skin Oxygen *Tension vs. Arterial Oxygen Saturation; Relationship to Oxyhemoglobin* Dissociation Curve. R. Penneys.—p. 204.
Comparison of Estimation of Basal Cardiac Output from Linear Formula and "Cardiac Index." H. L. Taylor and K. Tiede.—p. 209.
Some Structural Requirements for Prevention of Leukopenia Induced by Nitrogen Mustard. A. S. Weisberger, R. W. Heinle and B. Levine. —p. 217.
Immunochemical Study of Albumins of Serum, Urine, Ascitic Fluid and Edema Fluid in Nephrotic Syndrome. D. Gitlin and C. A. Janeway. —p. 223.
Spontaneous Fluctuations in Glucose Content of Hepatic Venous Blood in Resting Normal Human Beings. P. K. Bondy.—p. 231.
Agglutination of Sensitized Sheep Erythrocytes and Collodion Particles by Tuberculous and Normal Sera. C. A. Colwell and G. Pitner. —p. 238.

Journal-Lancet, Minneapolis

72:175-228 (April) 1952

What's Past Is Prologue. R. J. Anderson.—p. 176.
Antimicrobial Treatment of Pulmonary Tuberculosis. F. W. Pitts and C. W. Tempel.—p. 180.
Bronchography in Chronic Disease of Lung. R. M. Fawcett.—p. 185.
Routine Photoradiography in General Hospital: Its Place in Detecting Unsuspected Pulmonary Tuberculosis. S. Jacobs.—p. 189.
Emotional Problems in Tuberculosis. H. A. Wilmer.—p. 192.
Tuberculin Test as Evaluation of Future of Tuberculosis. J. B. Novak. —p. 198.
Report of Committee on Tuberculosis, American College Health Association W. T. Palchanis.—p. 199.
**Prolonged Intercostal Nerve Block in Upper Abdominal Surgery.* W. J. Puderbach and H. E. Shaftel.—p. 200.

Prolonged Intercostal Nerve Block in Upper Abdominal Surgery.—Pulmonary complications of upper abdominal surgery have been attributed to decreased respiratory excursion due to postoperative pain. Intercostal nerve block with 1 to 2 cc. of a stable solution of procaine and butyl aminobenzoate in a water miscible nonoily vehicle ("efocaine") was given a therapeutic trial for control of postoperative pain in 30 patients between the ages of 36 and 64. Of these 30 patients, 18 submitted to cholecystectomy, 1 to cholecystotomy, 1 to gastrectomy, 2 to mastectomy, 5 to operations for epigastric hernia, and 3 to operations for ventral hernia. Postoperative pain control was achieved to a high degree, permitting freedom of movement, deeper breathing, and coughing. Twelve patients had no local postoperative pain and required no narcotic or sedative medication. Fourteen had mild pain or were uncomfortable for the first 24 to 30 hours and required one to two doses of sedative medication. Three received three doses of sedative medication and one required five doses. The patients who had had intercostal nerve block received an average of 0.7 doses of postoperative medication as compared to 5.2 doses required by a control group of patients undergoing similar surgical intervention by the same surgeons. Local anesthesia lasted for from 8 to 21 days, the mean duration being 14.2. There were no local or systemic toxic manifestations. There was no interference with healing of the incision.

Journal of Neurophysiology, Springfield, Ill.

15:87-162 (March) 1952. Partial Index

Projection of Cortical Area 6 to Brain Stem in Monkey. E. W. Peterson and D. S. Bickers.—p. 87.
Selective Action of Anesthetics on Synapses and Axons in Mammalian Sympathetic Ganglia. M. G. Larrabee and J. M. Posternak.—p. 91.
Splanchnic Afferent Pathways in Central Nervous System. O Aidar, W. A. Geohegan and L. H. Ungewitter.—p. 131.
Distribution of Potentials Following Stimulation of Olfactory Bulb in Cat. C. M. Berry, W. D. Hagamen and J. C. Hinsey.—p. 139.

Journal of Pediatrics, St. Louis

40:269-404 (March) 1952

Ameliorative Effect of Antibiotics on Nonlipoid Reticuloendotheliosis (Letterer-Siwe Disease) in Identical Twins. H. R. Bierman, J. T. Lanman, K. S. Dod and others.—p. 269.
Flush Method for Blood Pressure Determinations in Newborn Infants. D. Goldring and H. Wohltmann.—p. 285.
Clinical Report on Amebiasis in Infants Under One Year of Age. C. Goldenberg, W. H. Shlaes and S. Mintzer.—p. 290.
Evaluation of "Seton" Procedure in Treatment of Infantile Communicating Hydrocephalus. H. J. Svien, J. G. Love, H. W. Dodge Jr. and H. M. Keith.—p. 298.
Hereditary Ectodermal Dysplasia of Anhidrotic Type: Report of Case in Negro. B. F. Metson and B. K. Williams.—p. 303.
Congenital Entero-Umbilical Fistula Due to Patent Vitelline Duct. H. E. Scalettar, M. M. Mazursky and H. Rascoff.—p. 310.
Experimental Glutamic Acid Treatment in Mentally Retarded Children. V. Oldfelt.—p. 316.
Bacteriostatic, Germicidal, and Sanitizing Action of Quaternary Ammonium Compounds on Textiles: II. Sanitization of Cotton Fabrics. M. A. Latlief, M. T. Goldsmith, J. L. Friedl and L. S. Stuart. —p. 324.
Infantile Cortical Hyperostosis: Report of Case Responding to Treatment with Corticotropin. L. G. Bush and O. E. Merrell.—p. 330.
Duplication of Stomach. H. F. Berg and K. Marx.—p. 334.
Case of Perforated Gastric Ulcer Manifested by Hematemesis in 8-Week-Old Infant. E. B. Rubell, F. Leix and R. A. Clelland.—p. 337.
Improved Examining Pad for Infants. G. E. Stafford.—p. 339.

40:405-538 (April) 1952

Acute Phase Reactants: I. Serum Nonglucosamine Polysaccharides in Patients with Rheumatic Fever and Related Conditions. V. C. Kelley. —p. 405.
Id.: II. Serum Hexosamines in Patients with Rheumatic Fever and Related Diseases. V. C. Kelley.—p. 413.
Tables for Predicting Adult Height from Skeletal Age: Revised for Use with Greulich-Pyle Hand Standards. N. Bayley and S. R. Pinneau. —p. 423.
Incontinentia Pigmenti: Report of Three Additional Cases. A. B. Loveman, M. T. Fliegelman, A. I. Weidman and C. R. Rein.—p. 442.
*Selenium and Dental Caries: Investigation Among School Children of Oregon. D. H. Hadjimarkos, C. A. Storvick and L. F. Remmert. —p. 451.
Gastric Response of Normal Children to Urecholine. J. A. Jones, B. Margolis and T. Potterfield.—p. 456.
Variations in Blood Pressure in Hospitalized Children. G. W. Clayton and J. G. Hughes.—p. 462.
Treatment of Pinworm Infection (Enterobius Vermicularis) with Egressin. R. F. Goddard and H. W. Brown.—p. 469.
Intestinal Obstruction in a Premature Infant Treated by Resection and Primary End-to-End Anastomosis. C. W. Bierman, J. K. Davis and F. C. Biehusen.—p. 474.
Internal Prolapse of Sigmoid Colon in Infants and Children. J. M. Babich, F. Burns and C. P. Lynxwiler.—p. 479.
Primary Tuberculous Inoculation of Ear Lobe: Report of Unusual Case and Review of Literature. L. G. Morgan.—p. 482.
Generalized Congenital Phlebectasia. J. M. Humphries.—p. 486.

Selenium and Dental Caries.—Since "bad teeth" was reported to be one of the most pronounced symptoms in populations living in a seleniferous area, the authors of this paper decided to collect further data on the relationship between selenium and dental caries. Urinary selenium concentrations were determined in native-born school children reared in the counties of Clatsop and Klamath of Oregon where high and low rates, respectively, of dental caries were known to exist. Previous studies indicated that fluoride content of the drinking water, availability of dental services, and quantity of candy bars and carbonated beverages consumed were not the factors for the observed wide differences in caries incidence. A direct relationship was found between the prevalence of dental caries and the urinary selenium concentrations, suggesting that, in an as yet unknown manner, the trace element selenium may render the teeth more susceptible to caries.

Michigan State Medical Society Journal, Lansing

51:265-400 (March) 1952

Civil Defense is Self Help. M. L. Lichter.—p. 301.
Development of Program for Uniform Medical and Surgical Care in Disaster. R. M. McKean and R. J. Noer.—p. 303.
Psychiatric Viewpoint on Civil Defense. P. A. Martin.—p. 304.
Casualty Evacuation Plan. J. A. Witter and R. Altman.—p. 310.
Shock and Hemorrhage. J. L. Posch and R. J. Noer.—p. 313.
Training of Blood Procurement and Distribution Teams. D. H. Kaump and E. R. Jennings.—p. 314.
Management of Burned Patients. J. L. Posch.—p. 318.
Treatment of Soft Tissue Injuries. D. A. Cameron, H. M. Smathers and A. M. Large.—p. 320.
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Management of Chest Injuries from Atomic Bombing. D. W. Myers and W. M. Tuttle.—p. 329.
Trauma of Nervous System. E. S. Gurdjian and J. E. Webster.—p. 331.
Resuscitation and Anesthesia. F. E. Greifenstein.—p. 335.
Emergency Treatment of Genito-Urinary Tract Injury. G. E. Chittenden. —p. 336.
Emergency Care of Eye Injuries. W. S. Davies.—p. 338.
Ear, Nose and Throat—Emergency Treatment. J. E. Croushore.—p. 340.
Acute Radiation Injury. L. Berman, A. H. Price and L. D. Stern. —p. 341.
Blood Clotting Mechanisms and Acute Radiation Injuries. W. H. Seegers. —p. 344.
Industrial Medical Services and Civil Defense. A. F. Lecklider.—p. 345.
Public Health in Civil Defense. P. T. Salchow.—p. 346.
Formula Works. J. R. Hainline.—p. 348.

Nebraska State Medical Journal, Lincoln

37:93-136 (April) 1952

Leukemia Cases at the University of Nebraska Hospital: Statistical Analysis and Review of All Records of Leukemic Cases Treated at the University Hospital from Jan. 1933 to Jan. 1951. A. S. Rubnitz. —p. 95.
Value of Peritoneoscopy in Diagnosis of Obscure Abdominal Conditions. J. C. Kennedy.—p. 99.
*Surgical Treatment of Duodenal Ulcer: Comparison of Results of Treatment With and Without Vagotomy. G. Crile Jr.—p. 104.
Bronchogenic Carcinoma—Statistical Survey of Symptoms and Diagnostic Procedures. W. P. Kleitsch and W. Van Hazel.—p. 107.

Duodenal Ulcer With and Without Vagotomy.—During the past five years over 400 patients have been treated at the Cleveland Clinic by vagotomy and gastroenterostomy. Seven of the first 154 patients could not be traced, but the remaining 147 have been followed for from two to four years. Good results have been obtained in 90% of the patients followed. Another 3% have had no recurrence of ulcer symptoms, are better than before operation, but have side-effects of the operation or gastrointestinal symptoms arising from causes other than an ulcer severe enough to require some form of dietary or medical treatment. In 10 patients (7%) the results are classified as failures. The 90% of patients in whom satisfactory results were obtained are following no diet, taking no medicine, and are permitted to eat, drink, and smoke as they please. They have been informed that they no longer have the ulcer diathesis and that they can forget that they ever had an ulcer. The majority of these patients make the unsolicited comment that they have never felt better in their lives. A recurrence after vagotomy and gastroenterostomy can be dealt with by resection, with a mortality rate no higher than that of a primary resection, but there is no known cure for the gastric cripple who does poorly after a radical gastrectomy. Because the mortality rate of vagotomy and gastroenterostomy is less than 0.5% in over 400 cases, because the morbidity and incidence of side-effects are lower than with gastric resection, and because in 95% of the cases there is no evidence of recurrent ulceration for from two to four years after operation, Crile believes that vagotomy combined with gastroenterostomy is the best operation for patients with complicated duodenal ulcer.

New Orleans Medical and Surgical Journal

104:343-384 (March) 1952

Thrombosis and Embolism. W. H. Parsons.—p. 343.
Present Status of Military Psychiatry. R. E. Chambers.—p. 348.
"Spells": Causes and Diagnosis of Common Complaint. I. Stevenson. —p. 352.
Unusual Case of Carcinoma of Cervix. R. W. Dickenhorst.—p. 359.
Mental Health Angle in Obstetrical Cases. L. W. Rowland.—p. 360.
Adoption. J. K. Avent, D. S. Buell, G. S. Knox and others.—p. 365.
Systemic and Laboratory Investigation of Intraocular Disease. N. L. Hart.—p. 372.

Oklahoma State Medical Assn. J., Oklahoma City

45:87-124 (March) 1952

Carcinoma of Thyroid Gland. P. J. Rosenbaum, H. C. Dodson and L. J. Starry.—p. 90.
Management of Acute Perforated Peptic Ulcer. W. P. Fite and A. H. Krause.—p. 93.
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Postgraduate Medicine, Minneapolis

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Use of Cortisone and Antibiotics in Resistant Pelvic Infections. A. Hurtig. —p. 196.
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Psychoanalytic Quarterly, Albany, N. Y.

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Public Health Reports, Washington, D. C.

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*Lead Poisoning in Young Children. H. Williams, E. Kaplan, C. E. Couchman and R. R. Sayers.—p. 230.
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Expenditures of Health Departments in Large Cities. I. Seeman.—p. 279.
Morbidity Reporting—Basis of Communicable Disease Control. W. G. Smillie.—p. 287.
Units of Radiation and Radioactivity. E. E. Anderson.—p. 293.
Effect of Radioactive Materials on Environmental Health. C. P. Straub. —p. 298.

Lead Poisoning in Young Children.—Chewing of paint from cribs, toys, furniture, and window sills, and eating of painted plaster and fallen paint flakes causes lead poisoning in childhood. Putting things in the mouth, though normal in the first year of life, is abnormal if continued much later and is referred to as pica, or perverted appetite. Children of teething age living in old, run-down houses where lead paint has been used for years are especially likely to have lead poisoning. Weeks or months following repeated ingestion of small amounts of lead, there may appear irritability, fretfulness or disturbed gastrointestinal function, such as lack of appetite, constipation, vomiting or cramps; there may also be pallor as the result of secondary anemia. More severe intoxication results in lead encephalitis, which may terminate in death. Central nervous system involvement or encephalopathy, rarely seen in adults with lead poisoning, is common in children, whereas peripheral neuritis, a lead line on the gums, and colic are usually absent in children. A history of pica and examination of blood smears for stippling of the red blood cells are important aids in diagnosis. Porphyrinuria is frequently found, and the demonstration by roentgenogram of an increased density in the growing ends of the long bones is a cardinal sign of lead poisoning. The quantitative determination of lead in blood or urine is helpful in diagnosis. Williams and associates review the efforts made in the Baltimore City Health Department to diagnose and treat plumbism in infants. Education of the public combined with "lead consciousness" in physicians and pediatric clinics and with a blood-lead laboratory service has resulted in a marked increase in case recognition. It is hoped that education and the enforcement of measures regulating the use of lead-containing paints will result in a material reduction and eventual elimination of lead poisoning in children.

Texas State Journal of Medicine, Fort Worth

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Infant Mortality in Texas. F. P. Helm.—p. 127.
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U. S. Armed Forces Med. J., Washington, D. C.

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Review of Tuberculosis Cases, United States Navy, 1949: Epidemiologic Implications. S. A. Britten.—p. 441.
Duodenal Ulcer in Military Personnel: Studies on Military Effectiveness of the Ulcer Patient: I. The Problem. E. D. Palmer and B. H. Sullivan Jr.—p. 455.
Treatment of Leukemia in Children. O. C. Bruton and W. S. Price. —p. 461.
Lymphoblastoma in Infant. M. Kurzrok and F. Marshall.—p. 469.
Traumatizing Foreign Body in Esophagus. R. T. Gants.—p. 473.
Mediastinal Emphysema. W. E. Klein, W. H. Neil, J. B. Stotler and R. L. Eslick.—p. 479.
Pronestyl in Treatment of Ventricular Tachycardia. C. L. Leedham and W. H. Isham.—p. 487.
Fluid Balance in Pediatrics. F. C. Biehusen and C. W. Bierman.—p. 491.
Myocardial Infarction Following Electric Shock. L. E. Imboden and C. B. Newton.—p. 497.

Cortisone in Frostbite.—Inasmuch as no satisfactory treatment for frostbite in the stage after rewarming was available, the metabolic research facility studied the effect of cortisone. Patients were placed on a standard diet with a fixed calculated intake of calories, nitrogen, sodium, potassium, and phosphorus. Balance studies were conducted on these elements. Antibiotics were used when indicated, and physical activity about the ward was encouraged. The treated patients and controls were "paired" as to severity of injury as far as possible. The treated group of eight patients received 100 mg. of cortisone in one intramuscular dose daily. Because no satisfactory placebo was found, none was used in the untreated group. *Cortisone exerted no significant clinical effect on the progress of the frostbite injuries; no significant metabolic effects were noted in the treated group.*

Virginia Medical Monthly, Richmond

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Rehabilitation of Tuberculous Patient. A. R. Dawson and B. B. Bagby Jr.—p. 179.
New Technique for Radiation in Carcinoma of Cervix. G. Cooper Jr. and J. N. Clore.—p. 184.
Contemporary Concepts in Diagnosis and Management of Epilepsy. I. S. Zfass.—p. 188.
Acne—Fifty Year Review. T. W. Murrell and T. W. Murrell Jr.—p. 198.
Silent Gallstone: Significance and Management. F. R. Whitehouse. —p. 202.
Changing Fashions in Treatment of Influenzal Meningitis. G. C. Hall Jr. and W. W. Waddell Jr.—p. 206.
Surgical Treatment of Glaucoma: Study of Seventy-Five Consecutive Cases. E. G. Gill.—p. 209.
Acute Shoulder Bursitis as Encountered in General Practice. J. B. S. Perrow.—p. 212.
Carcinoma of Second Breast: Case Report. G. J. Levin.—p. 216.

FOREIGN

Acta Medica Scandinavica, Stockholm

142:315-398 (No. 5) 1952. **Partial Index**

Chronic Cor Pulmonale in Pulmonary Tuberculosis. S. Samuelsson. —p. 315.
Administration of Testosterone Propionate in Bronchial Asthma. H. Arnoldsson and U. Pipkorn.—p. 325.
Incidence of Spondylarthritis Ankylopoetica in a Norwegian County. *H. Holst* and P. F. Iversen.—p. 333.
Relationship of Vegetative Nervous System to Uremia, with Remarks on Pathogenesis of "Renal Rickets." L. Hess.—p. 339.
Interrelationship of Folic Acid and Vitamin B_{12} *in Macrocytic* Anemia Associated with Linitis Plastica. M. R. Beyers, W. C. L. Diefenbach, H. Mark and L. M. Meyer.—p. 351.
*Case of Arteritis Temporalis Treated with ACTH. H. P. Østergård Kristensen.—p. 391.

Corticotropin in Treatment of Temporal Arteritis.—Temporal arteritis is characterized by chronic arteritis and periarteritis. Histologically it is manifested by formation of granulation tissue in the intima and media, with exudation in the adventitia and round-cell infiltration around the vasa vasorum. Postmortem microscopy of the arteries has now established that the lesion is a general chronic arteritis, as characteristic changes have been found in almost all the arteries of the body. Thus the lesion in the temporal arteries is to be regarded as the symptom that most frequently leads to the diagnosis. A woman, aged 71, with the typical symptoms of this disorder is described. For months she had had pains in the occipital region and lately also in both temporal regions. Her temperature was elevated. The temporal arteries were almost as thick as a pencil and of bluish color; they felt sclerotic. A segment of the frontal branch of the left temporal artery was removed, and it showed pathological changes. After several therapeutic measures, including administration of antihistamines and antibiotics, had proved largely ineffective, treatment with corticotropin was started and continued for about a month. The effect was prompt; the pain subsided, the temperature and sedimentation rate decreased, and the patient's general condition improved. When this therapy was discontinued, the temperature again increased and the sedimentation rate rose to the same level as prior to the institution of treatment, but the patient had no recurrence of pain. The author suggests that corticotropin treatment, possibly intermittent, combined with bishydroxycoumarin therapy, might improve the prognosis of temporal arteritis by preventing progression of the arteritis in some vital regions.

Brain, London

75:1-108 (March) 1952. **Partial Index**

Racemose *Form* of Cerebral Cysticercosis. E. R. Bickerstaff, P. C. P. Cloake, B. Hughes and W. T. Smith.—p. 1.
Factors Influencing Course of Regeneration and Quality of Recovery After Nerve Suture. S. Sunderland.—p. 19.
*Altered Consciousness Associated with Brain-Stem Lesions. M. Jefferson. —p. 55.
Intraventricular Medication in Catatonic Stupor (Preliminary Communication). S. L. Sherwood.—p. 68.
*Neurological Manifestations of Idiopathic Hypoparathyroidism. J. A. Simpson.—p. 76.

Altered Consciousness in Brain Stem Lesions.—Jefferson points out that alterations of consciousness may arise from lesions of the brain stem and describes in detail six patients who had disturbances of consciousness associated with signs of mesencephalic disorder. The first patient had a history of nine attacks of pathological sleep, each lasting up to a week. Initially they occurred without focal signs, but later were associated with indications of disorder of function in the mesencephalic and upper pontine tegmentum. The causal lesion seemed to be an angioma. In the second patient disturbances had started two and a half years before, with variable bilateral ptosis. After a year there was a sudden episode in which consciousness was lost for an hour; simultaneously further ocular signs and rigidity of trunk and limbs developed, all indicating a disorder at the mesencephalic level. After this she remained torpid for a week. In subsequent months she had three similar attacks. The third patient had symptoms of mesencephalic derangement, including fixed pupils, ptosis, external ocular palsies, nystagmus, bilateral pyramidal and cerebellar signs, some disturbance of postural and vibration sense, and dysarthria, together with evidence of spontaneous subarachnoid bleeding. He was somnolent for three days, and lethargic a fortnight longer. There was incomplete recession of neurological signs. A congenital anomaly of blood vessels in the region of the upper brain stem was suspected. The symptoms in the fourth patient also indicated a midbrain tegmental lesion, which, judging from the family history, seems to be caused by angiomatous malformations. The fifth patient had two attacks (at an interval of 10 years) of midbrain disorder, the first known to have been associated with subarachnoid bleeding and the second presumptively so. In the second attack there was loss of consciousness for 36 hours followed by pathological drowsiness and resultant amnesia of five weeks' duration. A brain stem vascular anomaly seems likely. The sixth patient had a pulmonary infarct and a few days later had brain stem embolism. She was stuporous for 18 hours, and in a state of somnolence for a week, with clinical signs of a mesencephalic lesion. Physiological studies suggest that when parasomnic symptoms occur clinically with brain stem disorders, they indicate encroachment of disease on the reticular substance. This has to be proved or disproved by pathological study.

Neurological Manifestations of Hypoparathyroidism.—That deficiency of parathyroid hormone secretion results in hyperexcitability of the peripheral nervous system (tetany or spasmophilia) is well known, but the other neurological manifestations of the tetany syndrome are less familiar. This paper presents the histories of two patients with idiopathic hypoparathyroidism. The syndrome consisted of tetany, trophic ectodermal lesions, skeletal abnormalities, and biochemical changes in the blood. Both patients had hemitetany for several months before this sign became bilateral. There was no evidence of gross cerebral disease. In case 1 the child also had epilepsy, acute hallucinatory psychosis, mental deficiency, a transient choreiform state, separation of cranial sutures, and calcification of the choroid plexus at an unusually early age. The electroencephalogram in case 1 showed a diffuse slow rhythm that increased to alpha frequency (with occasional delta activity) when the serum calcium was restored to normal. In case 2 the electroencephalogram of an adult showed mild dysrhythmia that was probably within normal limits. Attention is drawn to headache, excessive muscle weakness, and fatigue as symptoms of incipient tetany. Tests for latent tetany are reviewed and their mechanisms discussed.

British Journal of Radiology, London

25:113-168 (March) 1952. **Partial Index**

ARTHROGRAPHY: SYMPOSIUM

I. Comparative Study of Arthrography of Knee Joint. P. P. Hauch. —p. 120.
II. Evaluation of Pneumoarthrographs. L. S. Borow.—p. 129.
III. Arthrography of Shoulder. *D. H. Nelson.—p.* 134.
IV. Peripheral Ischaemia as Basis of Joint Disease (Serial Angiography of Periarticular Blood Vessels. A. Leb.—p. 140.
Radiological Studies of Alimentary Tracts of Undernourished German *Children.* F. R. Berridge and K. M. Prior.—p. 145.
Internal Irradiation of *Antral and* Mastoid Cavities. H. A. Hughes and R. H. Archer.—p. 152.

British Journal of Urology, Edinburgh

24:1-88 (March) 1952. **Partial Index**

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Biochemical Changes After Ureterocolic Anastomosis. A. W. Wilkinson. —p. 46.
Kidneys in Tourniquet Shock, with Particular Reference to Circulatory Changes (Experimental Study). S. R. Mukherjee.—p. 52.
Carcinoma of Rete Testis Occurring 10 Years After Orchidopexy. C. Dundon.—p. 58.
Arterial Supply of Male Urinary Bladder. J. L. Braithwaite.—p. 64.

British Medical Journal, London

1:615-670 (March 22) 1952

Professional Prospects: Comparison of Position of Doctors with That of Other Professional People. R. Lewis.—p. 615.
Hormone Assays in Normal and Abnormal Pregnancies. G. I. M. Swyer. —p. 619.
Changes in Basal Metabolism, Serum-Protein-Bound Iodine, and Cholesterol During Treatment of Hypothyroidism with Oral Thyroid and *l*-Thyroxine Sodium. J. D. Robertson and H. F. W. Kirkpatrick. —p. 624.
Persistent Urinary Salmonella Dublin Carrier with Bilharziasis. W. S. Miller.—p. 628.
Balantidiasis in South Persia. A. G. McCarey.—p. 629.
Exchange Transfusion in Severe Anaemia. T. Ward.—p. 631.
Homolateral Pyramidal Signs with Tumours of Posterior Fossa. L. C. Oliver and J. R. Gibbs.—p. 634.

1:671-724 (March 29) 1952

Some Medical Aspects of Tobacco-Smoking. R. B. Scott.—p. 671.
*Retrolental Fibroplasia: Problem of Prematurity. B. A. Bembridge, M. Coxon, A. C. L. Houlton and others.—p. 675.
Depression of Tuberculin Sensitivity in Guinea-Pigs by Certain Antituberculous Surface-Active Agents. P. D. Hart, D. A. Long and R. J. W. Rees.—p. 680.
*Tuberculosis in Families with B.C.G.-Vaccinated and Non-Vaccinated Children. A. W. Dickie.—p. 682.
Head-Dropping Test. R. Wartenberg.—p. 687.
Side Effects of Preparation of Vitamin B_{12}. P. D. Bedford.—p. 690.
"Epidemic" Abdominal Colic Associated with Steatorrhoea. M. E. M. Thomas.—p. 691.
Death Following Barium Enema in a Child with Hirschsprung's Disease. A. Jolleys.—p. 692.

Retrolental Fibroplasia.—A team from the Oxford Eye Hospital, the pediatric department of the United Oxford Hospitals, and the Nuffield Laboratory of Ophthalmology has been concerned in a joint investigation of retrolental fibroplasia since June, 1950. All prematurely born babies of low birth weight were subjected to routine ophthalmologic examinations. The infantile optic disk is always extraordinarily pale, and the central fundus often has a patchy distribution of pigment. The retinal periphery constantly has a peculiar edematous-looking greyness, gradually merging into the pinkness of the posterior fundus. These appearances have been seen in both full-term and premature babies, and they gradually disappear during the first weeks of life. The periphery of the infant lens is often doubly refractile, and this may lead one to suspect a detachment of the retina when in fact there is none. The authors are sure that diagnosis of retrolental fibroplasia cannot be made without the presence of a retinal detachment or thickening, or of some retrolental mass near the periphery. In addition to these signs engorgement of the retinal vessels and formation of "fans" of new vessels from the retinal vessels, often coming forward into the vitreous in front of the thickened or detached retina, are important diagnostic points. They do not regard retinal vascular dilatation alone as sufficient to warrant a diagnosis of retrolental fibroplasia. A total of 22 cases of retrolental fibroplasia have been observed at the Oxford hospitals since 1947. The authors gave corticotropin by intramuscular injection in four doses daily, totaling 20 to 25 mg., to six infants with retrolental fibroplasia and felt that the disease was arrested by this treatment. Despite gross intraocular damage, many of the eyes retain sufficient vision to enable the child to avoid obstacles and to walk about unaided. Excision of the eye is unnecessary, and is to be avoided in view of this possibility.

Tuberculosis in Families with BCG-Vaccinated and Non-Vaccinated Children.—Many workers have reported surveys demonstrating the protective value of BCG vaccine, but others found little evidence to support this view. The problem of carrying out controlled investigations to cover all possible variables has made it difficult to assess its true value. A search was made for families in which some children had been vaccinated with BCG and others had not, and which had been exposed to a known source of tuberculous infection. This paper presents data on 16 families with 22 vaccinated and 22 nonvaccinated children. The source of infection, while not the same for all children, was at least similar. When the source of infection was in the home, it may be assumed that each child was exposed to the maximum possible risk. The social conditions, especially overcrowding, varied between families, but the children in each family lived under the same conditions and their standard of nutrition tended to be the same. The reason that some of the children were vaccinated and others were not was probably that the mother was offered vaccination for some of the children on one particular occasion, for example, at a school medical examination, a child welfare center, or a maternity hospital, but did not trouble to have the remaining children vaccinated. Primary tuberculosis developed in 21 of the 22 children in the nonvaccinated group. In the BCG-vaccinated group, two at most showed some evidence of superinfection. The protective value of BCG vaccination is striking, both when the source of infection is within the family (with the maximum risk for each child) and when it is outside. The difference in the incidence of tuberculosis in the vaccinated and nonvaccinated children admitted to a hospital is recorded. The absence of tuberculous meningitis and miliary tuberculosis in the vaccinated group is noted.

Deutsche medizinische Wochenschrift, Stuttgart

77:413-444 (April 4) 1952. Partial Index

Progress in Gastroscopic Diagnosis of Gastric Carcinoma. H. Stollreiter. —p. 415.
*Methionine Tolerance as Liver Function Test. K. Schreier and H. Schönsee.—p. 418.
*Does Cytodiagnosis Signify Progress in Early Diagnosis of Malignant Tumors, Particularly in Carcinoma? H. J. Mohr and A. Többen. —p. 420.
Present Status of Treatment of Crural Ulcer and of Varicose Veins. F. Jaeger.—p. 422.
Chemotherapy in Tuberculous Diabetic Patients. G. Vieth.—p. 425.

Methionine Test for Liver Function.—Amino acids, particularly methionine, are increased in the serum of patients with liver damage. This suggests that it is difficult for the damaged liver cell to metabolize or store methionine. In preliminary tests the authors determined that after a tolerance test with methionine the serum of patients with liver disease showed higher methionine values than the serum of healthy persons. Since, however, determination of methionine in the serum requires complicated microbiologic or colorimetric tests, it was regarded as unsuitable for a routine liver function test. It was decided to determine the methionine level in the urine, especially since a simple as well as specific colorimetric method is available for this purpose, namely, the McCarthy-Sullivan reaction. The authors used this test in a modified form, which corresponded largely to a modification described by Horn, Johnes, and Blum. In this liver function test methionine is given by mouth, and its excretion in the urine is ascertained over the next 24 hours. Healthy adults excrete in their urine from 11.5 to 13% of orally administered methionine. The authors regard the elimination of from 15 to 19% as abnormally high and over 19% as definitely pathological. In children, excretion of over 15% is to be regarded as definitely pathological. The authors hope that, in view of the simplicity and cheapness of the test, it will find wide application.

Cytological Studies in Early Diagnosis of Malignant Tumors.—Since Mohr's report in 1949, he and Többen have been able to study an additional 317 cases in which carcinoma was suspected on clinical grounds by means of Papanicolaou's cytological technique. Gynecologic and bronchial carcinomas accounted for most of the cases. They found that under unfavorable conditions correct results will be obtained in 37% of the cases and under favorable conditions in 93%. Doubtful results are to be expected in from 3 to 28% of the cases and false diagnoses in from 2 to 25%. Caution is therefore necessary, since a negative or doubtful result never excludes the possibility of a carcinoma. Only because of its simplicity can the cytological method be regarded as an advance in the early diagnosis of carcinoma. Its use can be recommended in material that can be obtained by a direct smear. Indirectly obtained material, such as puncture fluids, gastric juice, or bronchial, or prostatic secretions, do not provide reliable results. Although the method provides helpful indications, it must be verified by biopsy. It must be used cautiously as a basis for therapeutic measures and for prognostic evaluation.

Glasgow Medical Journal

33:71-106 (March) 1952

*Total Colectomy. A. B. Kerr.—p. 71.
Value of Intermittent Venous Occlusion: Its Effect on Local Muscle Circulation as Determined by Clearance of Radioactive Sodium (Sodium 24). E. M. McGirr.—p. 83.
Failure to Influence Blood Coagulation Time by Sublingual Administration of Heparin. A. C. Kennedy and A. Brown.—p. 89.
Old-Time Quacks and Quackery. H. S. Carter.—p. 92.

Total Colectomy.—Kerr demonstrates on the basis of three case histories that total colectomy has a place in the treatment of both ulcerative colitis and familial polyposis; that with the aid of modern anesthesia, antibiotics, and blood transfusions the risks are not high; and that in the presence of a permanent ileostomy, life can be both useful and happy. The surgical program is a formidable one, and is usually carried out in three stages at intervals of about three months. The first stage consists of division of the ileum and formation of a permanent ileostomy. Removal of the distal ileum, cecum, ascending colon, transverse colon, and descending colon represents the second phase, and removal of the sigmoid colon, rectum, and anal canal the third phase. Discussing the management of ileostomy Kerr says that within a few days of the establishment of an ileostomy its products become less fluid, but they do not acquire the solidity seen with a sigmoid colostomy and cannot be satisfactorily managed with a simple dressing and belt. The problem for most patients is solved by the Koenig-Rutzen bag, a soft rubber bag attached to a rubber collar, which in turn is secured to the skin with a special adhesive solution. None of the patients had continuing distress in handling the ileostomy. Familial polyposis of the colon is the less controversial of the two conditions for which Kerr employed total colectomy. In this patient carcinoma developed low in the rectum so that there could be no conservation of the rectum or anal canal. The minimal operation therefore was excision of the rectum and sigmoid colon. Some would have advocated watching the remaining colon, but this would not have been safe, since the patient had extensive papillomas and continued to bleed from her colostomy. The two patients with ulcerative colitis raise the question under what circumstances ileostomy for ulcerative colitis should be followed by total colectomy. The indications in these two patients were the continued diarrhea and blood loss. The threat of carcinoma must also be considered, because the surviving epithelium at the margins of ulceration is shaggy, edematous, and often hyperplastic, changes which some regard as precancerous.

Journal of Pathology and Bacteriology, Edinburgh

64:1-256 (Jan.) 1952. **Partial Index**

Pathogenesis of Herpetic Encephalitis Following Corneal and Masseteric Inoculation. E. J. Field.—p. 1.
Focal Lesions in Skeletal Muscles and Peripheral Nerves in Rheumatoid Arthritis and Other Conditions. B. Cruickshank.—p. 21.
Effect of Sclerosis of Nerve Trunk on Ascent of Tetanus Toxin in Sciatic Nerve of Rabbits and on Development of Local Tetanus. J. H. Baylis, J. Mackintosh, R. S. Morgan and G. P. Wright.—p. 33.
Portal Circulation in Experimental Cirrhosis of Liver. P. M. Daniel, M. M. L. Prichard and P. C. Reynell.—p. 53.
Active Immunity in Mice Following Intranasal Injection of Sub-Lethal Doses of Living Haemophilus Pertussis. G. N. Cooper.—p. 65.
Effect of Antibiotics on Growing Cultures of Bacterium Coli. R. J. V. Pulvertaft.—p. 75.
Stimulation of Eosinophil Leucocyte. J. Vaughn.—p. 91.
*Significance of Atheroma of Renal Arteries in Kimmelstiel-Wilson's Syndrome. G. F. M. Hall.—p. 103.
Acute Necrotising Glomerulonephritis. J. D. Blainey.—p. 121.
Observations on Agglutination and Haemolysis of Red Cells Treated with Extracts of Mycobacterium Tuberculosis: Evaluation of Methods. A. W. Pound.—p. 131.
Renal Lesions in Leptospira Canicola Infection in Dogs. W. I. M. McIntyre and G. L. Montgomery.—p. 145.
Hyperplasia and Neoplasia of Sertoli Cell. A. L. Stalker and W. T. Hendry.—p. 161.
Recognition of Pathogenic Staphylococci: With Notes on Non-Specific Staphylococcal Haemolysin. J. Marks.—p. 175.

Atheroma of Renal Arteries in Kimmelstiel-Wilson's Syndrome.—The fact that more severe atheroma at a much earlier age develops in many diabetics than in the general population has been known for many years, but no particular significance appears to have been attached to excessive atheroma of the renal arteries in diabetics. It is the purpose of this paper to show that such atheroma may produce extensive structural changes in the kidney, which are of considerable significance in relation to Kimmelstiel-Wilson's syndrome. Two types of glomerular lesions occurring in diabetic glomerulosclerosis, the nodular lesion and the exudative lesion, are briefly described on the basis of the findings in a series of 135 diabetics. The clinical and postmortem findings in eight cases of Kimmelstiel-Wilson's syndrome in which death resulted from uremia are recorded. Gross atheroma with much narrowing of the renal arteries was present in all eight cases. Atheromatous involvement of the large and small branches of the renal artery gives the kidney a characteristic macroscopic appearance. The histological picture in all eight cases was distinctive, the predominant feature being ischemic obliteration of vast numbers of glomeruli. Atheroma of the renal arteries of comparable degree was seen in only one other case in which similar clinical and histological features were not present. In 43 other cases of diabetic glomerulosclerosis there was no evidence that the renal lesion, uncomplicated by renal artery atheroma, was responsible for death. The pathogenesis of the massive obliteration of glomeruli is discussed, and it is concluded that obliteration results from atheromatous narrowing of the renal arteries superimposed on severe arteriolosclerosis.

Lancet, London

1:623-678 (March 29) 1952

Surgery of Spleen. L. Whitby.—p. 623.
Splenectomy: Indications and Technique. R. Maingot.—p. 625.
Incidence and Mechanism of Early Dumping Syndrome After Gastrectomy: Clinical and Radiological Study. J. C. Goligher and T. R. Riley.—p. 630.
Routine Testing of Sensitivity of Bacteria to Antibiotics. J. R. May and C. W. Morley.—p. 636.
Treatment of Parkinsonism in a Child. J. W. Farquhar.—p. 638.
Studies in Aetiology of Pneumonia in Glasgow, 1950-1951. N. R. Grist, J. B. Landsman and T. Anderson.—p. 640.
Cat-Scratch Fever: Case in England. O. F. Garai.—p. 646.

Maandschrift voor Kindergeneeskunde, Leyden

20:37-68 (March) 1952

*Resection in Primary Tuberculosis. C. Dijkstra.—p. 37.
Therapy of Congenital Cataract. W. A. Manschot.—p. 52.
Corticotropin (ACTH) and Gold in Treatment of Lupus Erythematosus Disseminatus. J. Engelhardt.—p. 60.

Resection in Primary Tuberculosis.—Dijkstra discusses the indications for pulmonary resection in primary tuberculosis on the basis of five cases in which the continued discharge of tubercle bacilli indicated resection of the lung. Surgical treatment is rarely necessary in the beginning phase of the primary infection, because in most cases the pathological process seems to become arrested, although repeated sputum examination and gastric lavage may reveal the intermittent discharge of bacilli. In other cases there seems to be a long period of clinical arrest (from two to five years), after which the sputum cultures again become positive. Caseation of the lymph nodes with rupture into the bronchi, frequently associated with specific changes in the walls of the bronchi, was the commonest cause of the continued discharge of tubercle bacilli. In such cases conservative treatment is not adequate and resection is advisable. The sputum should be cultured frequently and over long periods in patients with primary pulmonary tuberculosis.

Medical Journal of Australia, Sydney

1:313-348 (March 8) 1952. **Partial Index**

Potassium Deficiency in Entero-Colitis in Infancy: Its Clinical Features, Treatment and Prevention. H. Williams.—p. 313.
Some Aspects of Pre-Eclampsia and Eclampsia. R. B. C. Stevenson. —p. 317.
"Pheniodol" Excretion: Observations on Urine of Patients with Hepatic Disease. C. R. B. Blackburn, H. L. Webster and D. K. Grant.—p. 321.
Value and Limitations of X-Ray Examination in Assessing Disease of Gallbladder. S. Goulston.—p. 323.

Minerva Medica, Turin

43:189-210 (Feb. 2) 1952

*Disodium Salt of Congo Red and Streptomycin in Tuberculous Infections. G. Pescetti and E. Destefanis.—p. 189.
Thiosemicarbazone in Extrapulmonary Tuberculosis. G. Oppia.—p. 196.

Streptomycin Combined with Congo Red in Tuberculous Infections.—The failure of streptomycin to cure tuberculous infections has been attributed to the inability of the drug to penetrate and diffuse into the specific foci. To test this hypothesis, the authors combined streptomycin with congo red and obtained a double disodium salt that in vitro maintained, and sometimes enhanced, the antibiotic properties of streptomycin. In addition, because of phagocytosis of congo red, the antibiotic could penetrate and concentrate into the foci. Four groups of guinea pigs, of which one served as a control, were inoculated intraperitoneally with Mycobacterium tuberculosis. One month later one group was treated with intracardiac injection of congo red every other day, one group with intramuscular injection of 0.01 gm. of streptomycin in 1 cc. of isotonic sodium chloride solution every day, and the third with intramuscular injection of 0.01 gm. of streptomycin combined with 1 cc. of congo red every day. The experiment lasted 257 days, and the observation period after the treatment 135 days. All the control guinea pigs, all those that received congo red, and 25% of those treated with streptomycin died. There was no death among those treated with the salt. During the treatment reaction to tuberculin was positive in 90% of the animals treated with congo red, negative in 40% of those treated with streptomycin, and negative in all those treated with the salt. In some of these it became positive when treatment was interrupted. All the guinea pigs lost weight after the inoculation, regained it during treatment, and lost it again after the treatment was discontinued. Only those that were treated with the salt began to regain weight after a few days and exceeded their initial weight. The authors also made histological studies of these animals, and concluded that streptomycin combined with congo red arrests the infection and attenuates the toxic phenomena in tuberculous guinea pigs more than streptomycin alone. Moreover, if the infection reactivates after treatment is interrupted, it is a mild process with productive, fibrotic, and cicatricial tendencies. The authors, encouraged by these results, are now using the salt to treat patients with tuberculosis. The effects seem encouraging, but a definitive evaluation is still premature.

Philippine Medical Association Journal, Manila

28:61-104 (Feb.) 1952

Trial with Clostridium Tetani Toxoid in Inhibition of Malaria Relapse. J. B. Mendoza.—p. 61.
Fetal Salvage Through Prenatal Care. H. Acosta-Sison.—p. 67.
Report of Case of Left-Sided Homonymous Hemianopsia with Macular Sparing. P. de la Paz.—p. 74.
Acute Yellow Atrophy: Case Report. S. Sotto-Yuvienco.—p. 78.
Neglected Problems in Leprosy Control. C. B. Lara.—p. 82.

Presse Médicale, Paris

60:313-328 (March 5) 1952. **Partial Index**

*Remarks on Ligation of Inferior Vena Cava in Cardiac Decompensation; Results, Indications and Contraindications. C. Lian, H. Welti and F. Morgenthaler.—p. 313.
Nephritis After Ingestion of Mussels; Role of Allergy. M. Dérot.—p. 316.
General Anesthesia and Cerebral Surgery; Significance of Respiration Under Anesthesia. G. Lazorthes and L. Campan.—p. 319.
Study of Blood Volume in 60 Patients With Pulmonary Tuberculosis; Its Significance in Surgical Treatment. Cornet and Duhamel.—p. 322.

Ligation of Inferior Vena Cava in Cardiac Decompensation.—Ligation of the inferior vena cava was performed on 15 patients between the ages of 13 and 61 years with cardiac decompensation. There was no operative death. Immediate improvement of the respiratory embarrassment was obtained by all the patients, but despite immediate satisfactory results, one patient died from cardiac decompensation and uremia three weeks after the operation. Three patients died within the first three postoperative months; two of these patients had mitral lesions associated with asystole, and the third had asystole complicating persistent ductus arteriosus. Three additional patients died three, five, and nine months respectively after the operation; two of these had mitral lesions associated with asystole, and one had cor pulmonale and angina pectoris. Of the eight surviving patients, two were still incapacitated four and six months after the operation. Satisfactory late results were obtained in the remaining six patients; two of them had no functional disturbance and resumed their normal activities five and three months after the intervention. The others were considerably improved, but the functional signs of their heart disease had not subsided completely. These results corroborated those reported by other workers, and showed that ligation of the inferior vena cava induces immediate improvement that may be followed by death within the first six operative months, while permanent improvement may be obtained by 50% of the patients operated on. Use of local anesthesia and a retroperitoneal rather than transperitoneal approach was considered responsible for the satisfactory results. Poor general condition is the only contraindication to the operation in young patients. Lesions of the aortic valve are not considered contraindications. Indications are most favorable in patients whose stasis is limited to the lungs; they are less favorable in those with stasis of the lungs and the liver, and least favorable in those with visceral stasis associated with edema. Ligation of the inferior vena cava should be considered even in a cardiac patient with mitral lesions and considerable dyspnea on effort in the absence of rales at the base of the lungs and of enlargement of the liver, after treatment with mercurial diuretics, salt-free diet, and thevetin has failed.

Revista Médica de Chile, Santiago

80:79-134 (Feb.) 1952. **Partial Index**

Treatment of Ectopic Testes. J. Zañartu and E. C. Hamblen.—p. 79.
*Regression of Pulmonary Metastases of Breast Cancer with Androgen and Estrogen Treatment. A. Lucchini and L. Vargas Fernández.—p. 94.
Aureomycin and Terramycin Treatment of Human Enteroprotozooses. J. Faiguenbaum, M. Miranda, A. Neghme and M. Agosin.—p. 99.

Regression of Pulmonary Metastases with Androgens and Estrogens.—The authors have treated more than 80 patients with breast cancer since 1944 with implants of androgens (methyltestosterone, testosterone propionate, and testosterone); 9 of the patients also had estrogen implants (diethylstilbestrol or hexestrol). A review of 64 of these cases showed that the patients had had 304 hormonal implants (252 of methyltestosterone), amounting usually to 300 mg. every three to four months (average duration of treatment, 24 months with a total dose of 1.07 gm. of hormone). Pulmonary metastases usually occur in the final stage of the disease and end in death within a short time. Fifteen patients, all past the menopause, in this series had pulmonary metastases; in 11, the response to hormone therapy was favorable. This exceeds the figures given by the Council on Pharmacy and Chemistry of the American Medical Association, which reports improvement in only 13% of such cases. Treatment was given for an average of 27 months to these patients, about 300 mg. of androgens being given every three to four months. Larger doses do not seem to give better results; length of treatment is more important than size of dose. Striking regression of pulmonary metastases was obtained in two cases with small doses of synthetic estrogen, 100 mg. per implant, a fact requiring further study. Average survival from start of treatment was 19.3 months, which is longer than generally reported (radiotherapy, 12 months; hormone therapy, 8.9 months). Most of the patients were treated with methyltestosterone, which may be more effective than other androgens in antitumoral properties, especially when administered parenterally. The change of hormones, or the influence of an estrogen following an androgen, may also have affected the favorable outcome; in many cases, improvement was observed after the antitumoral effectiveness of the androgen had been exhausted.

BOOK REVIEWS

Calculation of Industrial Disabilities of the Extremities. By Carl O. Rice, M.D., M.S., Ph.D., Clinical Associate Professor of Surgery, University of Minnesota Medical School, Minneapolis. Cloth. $10.50. Pp. 289, with 160 illustrations by Jean E. Hirsch. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto 2B, 1952.

In this monograph the author has developed a detailed objective approach to the evaluation of industrial disabilities limited to the extremities. In the preface, it is stated that the information is intended to be of specific assistance to the physician in determining disability, a guide to the industrial commissioner in setting weekly indemnity values, and an aid to the insurance adjuster in estimating insurance reserves. The well-delineated table of contents and index provide easy reference to the subject matter.

In the opening chapter, there is a brief review of the methods used by Kessler, McBride, and others. To the author, it seems that methods for calculating disability that give unit values for pain, incoordination, adaptability, and similar intangibles are of little more value than a good guess and are accurate only in proportion to the experience of the physician who makes them. Dr. Rice states that it is his assumption that, when the useful range of motion is used as a basis for calculating disability, almost all other factors have already been given consideration.

The section dealing with relative values explains the protractor system used by the author in arriving at values for all of the joints of the extremities. He feels that it is the responsibility of the physician to determine the percentage loss of function in the involved member and that the industrial commission should render the award. A plea is made for standardization in the compensation laws, so that anatomic and functional values will be the same in all states.

The author implies that he recognizes the limitations inherent in any system of disability evaluation by stating that he is endeavoring to establish some degree of standardization in a field that almost completely defies it. This book should prove especially useful to those physicians who are called on to make only an occasional estimation of disability.

Dynamic Psychiatry. Edited by Franz Alexander, M.D., and Helen Ross. Cloth. $10. Pp. 578. University of Chicago Press, 58th St., and Ellis Ave., Chicago 37; Cambridge University Press, Bentley House, 200 Euston Rd., London, N.W.1, 1952.

This comprehensive volume consists of 16 articles written by 15 distinguished specialists and practitioners. Part 1 is concerned with theoretical concepts of dynamic psychiatry, such as development of fundamental concepts of psychoanalysis, dreams and rational behavior, history of metapsychological concepts, and personality development. Part 2 is concerned with clinical problems in psychiatry. In this section are articles on neuroses, behavior disorders and perversions, acute neurotic reactions (based on the study of war neuroses), emotional disorders of childhood, psychodynamic approach to the study of the psychoses, and principles of psychiatric treatment. Part 3 is concerned with the influence of psychoanalysis on allied fields. It includes articles on medicine, social anthropology, clinical psychology, animal psychology, and current thought. The book is intended not for the lay public but as a reference book for psychiatrists, psychoanalysts, physicians who are interested in the principles of psychodynamics, psychologists, and social scientists. It is a scholarly and comprehensive presentation, skillfully prepared and edited, which includes a wealth of information that is not contained elsewhere in one volume. The authors have given almost 1,000 references. They have rendered a real service by their respective contributions.

Manic-Depressive Psychosis and Allied Conditions. By Leopold Bellak, M.D., Clinical Assistant Professor of Psychiatry, New York Medical College, New York, with Blaise Pasquarelli, M.D., Research Associate, New York State Psychiatric Institute, New York, Ernest Parkes, M.A., Instructor in Psychology, New York University, New York, Sonya Sorel Bellak, and the collaboration of Sydell Braverman, M.A. Foreword by Winfred Overholser, M.D. Cloth. $9.75. Pp. 306. Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1952.

This book summarizes the literature on manic-depressive psychosis and related disorders, much of which is considered by the authors as not being readily or easily available to psychiatrists. The work was, in part, sponsored by a grant from the National Institute of Mental Health of the United States Public Health Service. The authors mention more than 1,000 studies of affective disorders and provide adequate bibliographic reference to them with the stated purpose of providing an incentive to further work in this field rather than the presentation of a critical review.

The book is divided into two parts. The first part discusses diagnosis, treatment, and prognosis of manic-depressive psychosis, and the second part relates to allied conditions, such as involutional psychosis, reactive depression, depression of old age, and psychotic reactions to pregnancy. Additionally, the author discusses his own multiple-factor psychosomatic theory of manic-depressive psychosis and relates it to a parallel theory of causation in schizophrenia. The summary of the literature will be important to those interested in research, as it cites, in brief form, the statements that have been made by previous investigators of the affective disorders. From this point of view, the volume makes a contribution of considerable value.

The Thoracic Surgical Patient—Preoperative, Anesthetic and Postoperative Care. By Lew A. Hochberg, M.D. Foreword by Frank B. Berry, M.D. Cloth. $8.75. Pp. 364. Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1952.

This book has been written in the main by Hochberg; however, six other specialists have collaborated in writing specific sections of the book. Some of the chapters are repetitive; however, this is no drawback and in some instances accentuates some necessary points and procedures. Some might disagree with certain statements made, but the basic teachings and precepts are well presented and should go far to produce good results. The emphasis upon such ofttimes neglected subjects as anesthesia, nutrition, fluid balance, blood replacement, resuscitation, and rehabilitation makes this work worthwhile. The section dealing with physiology and pathological physiology is particularly well done.

It is regrettable that illustrations are lacking since there are numerous opportunities in this book to have used simple line drawings; these might have emphasized numerous points.

The quality of the paper could have been improved on. The book can be recommended for both undergraduates and postgraduates interested in thoracic surgical patients.

As bases anátomo-patológicas da neuríatria e psiquiatria. Volumes I e II. Por Walter Edgard Maffei, D.M., Livre-docente de anatomia patológica, patologista do Hospital Juquerí (Franco da Rocha, Est. S. Paulo). Cloth. Pp. 491, with 345 illustrations; 494, with 410 illustrations. Imprensa Metodista, Rua da Liberdade 659, São Paulo, 1951.

The author, a distinguished neuropathologist from the University of São Paulo and its associated hospitals, is fully qualified to write on the subject of the pathological changes in the nervous system resulting from injury, disease, and developmental anomalies. His material, based on 16,000 autopsies, is abundant, and his laboratory facilities are excellent. The book covers the usual topics of degenerations, inflammations, disorders of circulation, with long chapters on syphilis, tumors, peripheral nervous disease, meningitis, trauma, and parasitic infections. An alphabetical index is provided, but only a few references to the literature are included as footnotes, without a complete bibliography. Over

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The reviews here published have been prepared by competent authorities and do not represent the opinions of any official bodies unless specifically stated.

800 well-produced illustrations greatly add to the value of the text, particularly for those readers who cannot read Spanish fluently. As the author was trained in France, his background is frequently reflected by references to French literature.

The volumes, well bound and serviceable, are printed with care on good paper. They are a splendid contribution to the subject, of special value to physicians in North America because of the abundant illustrations, the diagrams drawn by the author, the sections on parasitic diseases, and the scholarly text. This monograph should be in all pathology laboratories and medical libraries.

The Welfare State and the National Welfare: A Symposium on Some of the Threatening Tendencies of Our Times. Edited, with introduction, by Sheldon Glueck, Roscoe Pound Professor of Law, Harvard Law School, Cambridge. Cloth. $3.50. Pp. 289. Addison-Wesley Press, Inc., Kendall Sq., Cambridge 42, Mass., 1952.

National social and economic policy has been the subject of contemplation by persons of all nations since the beginning of time. Never before in the history of the United States has it been so important to decide not only which road we will take but how far down that road we will go. A symposium of addresses and articles dealing with some of the more questionable politico-economic trends in the last decade has been compiled and edited, with an introduction by Sheldon Glueck, under the title "The Welfare State and the National Welfare." The contributing authors, statesmen, educators, lawyers, engineers, economists, and journalists, are men from both sides of the political fence who are compelled by their feeling of public responsibility to raise their voices in protest. The book includes articles by Bernard Baruch, Vannevar Bush, Harry F. Byrd, Donald K. David, John Foster Dulles, Dwight D. Eisenhower, Herbert Hoover, Raymond Moley, Edwin G. Nourse, Roscoe Pound, Donald Richberg, Walter E. Spahr, and the editors of *Life*. While each has a different emphasis, all agree that unless some reasonable and clear limits are placed on the conception of what the general welfare is and to what extent our financial future can be mortgaged to carry out this illusion of security we are in grave danger of national bankruptcy. National bankruptcy is just a symptom of the disease that is eating away at the American heritage and will continue to spread if we are willing to legislate away our personal liberties for a feeling of false security and propel ourselves down the road to a welfare state.

Are we losing any liberties? Let us figure it out in dollars and cents. Taxes and federal expenditures took $31.60 from every man, woman, and child in 1929. This amount increased to $268.23 per person in 1948 and in the face of proposed increases could easily amount to nearly $500.00 in 1952. If we add state and local taxes to federal taxes, the annual tax obligation of the American public would be more than 40 cents out of every dollar they earn. Where is the savings or surplus that starts businesses, builds churches, and finances education and retirement. There is none. In the face of these social necessities the welfare state steps in to do it for us, and these are then under the control of the government. The welfare state simply offers to do for us what we can do better, more cheaply, and with greater satisfaction for ourselves. What becomes of our freedom when it is decided for us where, how much, and by whom our money will be spent?

It is a delusion to think that we can buy security and can still retain our liberties simply by paying high taxes. There can be no security for persons whose livelihood depends on political favor and political pressure. In order to have an effective planned or welfare state there must be control of the factors of production by the planners, in this case the government, and this is the very definition of national socialism. As Richberg states, ". . . a comprehensive welfare state must be a police state."

It is the use of so-called democratic methods to achieve inroads to a planned economy that deceives and confuses public opinion. Ambiguous wording in campaigns of special interest groups paint beautiful pictures, but when the paint dries we find we have mortgaged our personal liberties for a controlled state. During this year of national elections, we have a chance to decide if the present trend should continue. We must individually and collectively look beyond special, regional, or personal interests and see where we want to go. America must choose.

Medical Nursing. By Amy Frances Brown, R.N., M.S. in N., Associate Professor of Medical Nursing, State University of Iowa College of Nursing, Iowa City. Second edition. Cloth. $5.50. Pp. 1099, with 387 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; 7 Grape St., Shaftesbury Ave., London, W.C.2, 1952.

This second edition, like the first, is intended to enable the student nurse to give more effective and complete care to medical patients. The author has had wide experience as an educator in the field of medical nursing. In this edition, new chapters have been added on poliomyelitis, viral hepatitis, convalescence, rehabilitation, radiological nursing, and anti-infectious agents. The features that characterized the first edition have been given greater emphasis in this edition. Stress is on nursing care, the consideration of the patient as an individual and his need for total care, the problem of the chronically ill and aged, the teaching of patients, the integration of pharmacology, and the selection of appropriate illustrations. Attention has been given to sociologic, economic, and emotional factors as they affect the health picture of individuals or groups.

The book is divided into 11 units that include diseases of the respiratory system, the cardiovascular-renal system, the digestive system, the skin, the endocrine system and metabolism, and the nervous system. There are also sections on nutritional deficiency disease, the arthritides, allergy, and communicable disease. The illustrations consist of photographs, some in color, and diagrams, charts, and graphs. All chapters are followed by a bibliography.

This is a comprehensive text. Modern diagnosis and treatment are clearly and concisely discussed to a degree that will enable the student to develop an understanding of them as a basis for nursing care. Emphasis is, however, placed on the role of the nurse in patient care. This approach is logical and practical and does much to make this text useful for student nurses.

Attaining Manhood: A Doctor Talks to Boys About Sex. By George W. Corner, M.D., Director, Department of Embryology, Carnegie Institution of Washington, Washington, D. C. Second edition. Cloth. $1.50. Pp. 97, with 16 illustrations. Harper & Brothers, 49 E. 33rd St., New York 16, 1952.

Attaining Womanhood: A Doctor Talks to Girls About Sex. By George W. Corner, M.D., Director, Department of Embryology, Carnegie Institution of Washington, Washington, D. C. Second edition. Cloth. $1.50. Pp. 112, with 15 illustrations. Harper & Brothers, 49 E. 33rd St., New York 16, 1952.

Referred to as companion volumes, these book follow essentially the same pattern of presentation. Each consists of eight chapters, and topics common to both books are the human reproduction system, attainment of maturity, sex attraction, sex conduct, and the scientific study of sex and reproduction. In the volume for boys, the chapter dealing with venereal diseases is called sex disorders, whereas a similar chapter in the book for girls is simply designated as venereal disease. As the author points out, either volume would provide satisfactorily basic information for either sex, but there is good reason for avoiding a composite publication. The books are written in a frank, friendly style without any pompous warnings, and explanations are simple yet entirely adequate. A point on which the author should be commended is his use of correct terms, preferring, as he says, to depend on the intelligence of his readers in discussions in which simpler language would not be accurate. Both volumes are adequately provided with instructive illustrations; there are 15 in the book for girls and 16 in the book for boys. These publications can be recommended to high school students; they would undoubtedly also be of considerable value to many parents whose background of information in this field is hazy or incomplete.

Good Food and Bad Stomachs: 500 Delicious and Nutritional Recipes for Sufferers from Ulcers and Other Digestive Disturbances. By Sara M. Jordan, M.D., and Sheila Hibben. With introduction by Harold W. Ross. Cloth. $2.95. Pp. 255. Doubleday & Company, Inc., Garden City, New York, 1951.

This is a useful and encouraging book of recipes and directions for preparing foods for patients with peptic ulcers and those who have other gastrointestinal disturbances requiring a bland and nonirritating diet. Aside from its thoroughly scientific character and complete reliability, it possesses a virtue by no means common in dietetic cookbooks; namely, it places emphasis on the attractiveness of the diet.

Text-Book of Orthopædic Medicine. Volume II: Treatment by Manipulation and Deep Massage. By James Cyriax, M.D., B.Ch., Physician to Department of Physical Medicine, St. Thomas's Hospital, London. Fourth edition. Cloth. $6. Pp. 335, with 121 illustrations. Paul B. Hoeber, Inc., (medical book department of Harper & Brothers), 49 E. 33rd St., New York 16, 1951.

The text is extremely well illustrated and discusses methods of massage and manipulation and their usefulness in a vast number of conditions pertaining particularly to the musculoskeletal system. Although some techniques of massage and manipulation that are acceptable are discussed, it is difficult to understand why a textbook of orthopedic medicine contains sections on manual vibrations for affecting the pylorus, duodenum, ascending colon, descending colon, and distention of the stomach. Another technique directed toward reproducing "fixed subluxation of the sacroiliac joint" will not be too well received by those who question the existence of such a condition.

The text seems to be overenthusiastic in its indication of the value of massage and manipulation, but, in spite of this, it does clearly present various techniques for the treatment of parts of the musculoskeletal system. The book appears to be written especially for physical therapists.

Map of the World Distribution of Malaria Vectors. By Jacques M. May. Reprinted from Geographical Review. Volume XLI, no. 4, 1951. Paper. Pp. 638-639. American Geographical Society, Broadway at 156th St., New York 32, 1951.

Map of the World Distribution of Cholera. By Jacques M. May. Reprinted from Geographical Review, Volume XLI, no. 2, 1951. Paper. Pp. 272-273. American Geographical Society, Broadway at 156th St., New York 32, 1951.

Map of the World Distribution of Poliomyelitis. By Jacques M. May. Reprinted from Geographical Review. Volume XL, no. 4, 1950. Paper. Pp. 646-648. American Geographical Society, Broadway at 156th St., New York 32, 1950.

These maps form part of a projected "Atlas of Diseases" instituted by the American Geographical Society, a professional organization of geographers that is especially interested in medical geography. The first map, prepared with the assistance of a grant from the National Foundation for Infantile Paralysis, includes data on morbidity rates for poliomyelitis since 1900, regional differences in the age group predominantly attacked, epidemics among military personnel, and percentage of positive reactions to neutralization tests in selected areas. The sources of information are appended. The second map shows the world distribution of the various types of malaria parasites and of the mosquito vectors. Also included are brief notes on the epidemiology of malaria, a résumé on the natural history of the vectors, and a selected bibliography. The third map shows the endemic foci of cholera, the routes of spread of the first four pandemics of cholera, the outbreaks of cholera between 1923 and 1950, monthly occurrences of cholera in India, and the 1947 Egyptian epidemic.

This series of maps should be of great interest to teachers and students of epidemiology and to all others wishing information concerning the incidence of important diseases in various parts of the world.

Fluid Balance: A Clinical Manual. By Carl A. Moyer, M.D., Professor of Surgery, Washington University School of Medicine, St. Louis. Cloth. $3.75. Pp. 191, with 1 illustration. Year Book Publishers, Inc., 200 E. Illinois St., Chicago 11, 1952.

This manual is described by the author as a practical guide to fluid and electrolyte imbalances, with their changes of volume and composition of the body fluids and the symptoms and signs that they produce. The subject is concisely reviewed. Clear-cut descriptions of many problems of electrolyte and water imbalance are discussed in brief form.

The subject is covered by the author from a primarily surgical viewpoint. Emphasis is placed on postoperative complications. Some medical conditions with outstanding problems of fluid balance are mentioned in passing. The changes in extracellular fluid volume and composition are described and illustrated by reports of cases. Some original and useful concepts are included. The coverage of disturbances of ions other than sodium, chloride, and bicarbonate ions is incomplete; an example is the omission of mention of the neuromuscular signs of potassium intoxication. Treatment is clearly outlined and documented by illustrative cases. This should be a useful handbook for the surgeon, but it will be of somewhat less interest to the general physician because of the limited coverage of problems associated with medical disease.

Thomas Jefferson: Scientist. By Edwin T. Martin. Cloth. $4. Pp. 289, with illustrations. Henry Schuman, Inc., Publishers, 20 E. 70th St., New York 21, 1952.

This book is representative of the vast amount of scholarly research on Jefferson that has been going on since his bicentennial in 1943. It provides a fascinating account of a great American president's intellectual curiosity, which brought him into intimate relation with an extraordinary range of scientific fields: botany, geology, medicine, meteorology, agriculture, architecture, astronomy, chemistry, paleontology, ethnology, exploration, invention, and archeology. In these diverse fields Jefferson's insatiable thirst for knowledge was coupled with the desire that the benefits of science be made available to his fellow men so that their lot might be progressively bettered. But perhaps more important than his search for daily application of scientific knowledge to useful social ends was Jefferson's ardent championship of the freedom of scientific inquiry. He maintained that circumscribing free scientific inquiry could only retard "all advances in science as dangerous innovations." Throughout his long life Jefferson expressed a profound belief in "the illimitable freedom of the human mind." This book is highly recommended.

Kinesiology in Nursing: Laboratory Manual. By Bernice Fash, M.A., University of Illinois. Cook County School of Nursing. McGraw-Hill Series in Nursing, Lucile Petry, consulting editor. Paper. Loose-leaf. $2.80. Pp. 142, with 102 illustrations by Paul H. Stone. McGraw-Hill Book Company, Inc., 330 W. 42nd St., New York 18; Aldwych House, Aldwych, London, W.C.2, 1952.

In this book, a detailed application of anatomy and physiology to the problems of posture and exercise for patients is discussed. The author has considered these problems from both viewpoints, namely that of the patient in danger of weakness, deformities, and bedsores and that of the nurse confronted with the equally real dangers of disabling overstrain. The illustrations, which at first strike the reader as unconventional, are soon appreciated as remarkably illuminating; they make the book extremely valuable. It is hoped that in some future edition the author will break further with convention by getting rid of the illogical adjectives "concentric" and "accentric" as applied to certain kinds of muscular activity (page 9). This book should be available in the library of every school of medicine and nursing.

Introduzione alla biometria. Di G. Barbensi, Ars medica, collana diretta dal Prof. Vincenzo Lapiccirella. Cloth. 3000 lire. Pp. 418, with illustrations. Vallecchi editore, Viale dei Mille, 90, Florence, 1952.

The author undertakes to present to biologists an easy exposition of statistical methods useful in their specialty. Interspersed in the smoothly written text are 133 numerical problems from various fields of biology and medicine. Author and publisher have cooperated in producing a remarkably legible book in which one readily finds most of the concepts that have become familiar in current textbooks: probability, graphic representation, measures of central tendency and of dispersion, asymmetry and kurtosis, correlation and contingency, interpolation, regression, and variance and covariance. There are useful tables, a brief bibliography that does justice to foreign publications, an index, and a table of contents.

The book is written more for the observer than for the experimenter, with examples chosen more from anthropometry than from experimental therapeutics. As a result there is little emphasis on the important idea of the contratest, contre-essai, or control, an idea that will require much further emphasizing before it is sufficiently appreciated in clinical research. This deficiency can easily be remedied in future editions, which are to be anticipated in view of the general excellence of this book.

QUERIES AND MINOR NOTES

REMOVAL OF TRACHEOTOMY TUBES

To the Editor:—*In a hospital there is a difference of opinion regarding the proper time to attempt extubation following tracheotomy done for acute nondiphtheritic laryngitis and regarding the procedure to be followed. What is your opinion?*

M.D., Pennsylvania.

This inquiry was referred to two consultants, whose respective replies follow.—Ed.

Answer.—It seems obvious that a tracheotomy tube should be left in place as long as it is required; however, the decision concerning when to remove it usually rests with the operator. In acute conditions, when tracheotomy is indicated, there is generally edema about the glottis. Under such circumstances, the edema subsides with appropriate treatment, as a rule, within a few days, and the tube can be withdrawn at the end of a week or 10 days at the most. A common fallacious practice among residents training in their specialties is to gradually block off the mouth of the tube with adhesive tape or by other means to determine if the patient has a free airway. If the tube happens to fit snugly into the trachea, complete blocking of its protruding end should cause asphyxiation of the patient. If the patient continues to breath it is only because the tube is small enough to permit air to pass along its sides into the lungs. Even under the latter condition the physician is demanding that there be normal breathing with a foreign body (tracheotomy tube) in the respiratory tract. At one time, tracheotomy tubes with a "window" in the shoulder were frequently used. With such a tube, blocking of the external orifice would indicate properly whether there was a free passage of air through the glottis and the tube into the lungs. In some cases of laryngotracheobronchitis and in bulbar poliomyelitis there may be profuse secretions that require frequent aspiration for a considerable time. As a consequence, it may be desirable to leave the tube in place much longer than for most acute infections. Nevertheless, the tube should be removed as soon as this can be done with safety. Cases have been observed in which it was necessary for the patient to use a tracheotomy tube for many years after the acute infection had subsided.

The presence or absence of edema about the glottis can be determined by palpation. It is sometimes advisable to make such an examination before removal of the tube, although inspection with a laryngoscope is generally preferred. After removal of the tube a piece of sterile gauze may be laid over the opening until it becomes apparent that a reinsertion of the tube will not be necessary. The skin surfaces often can be approximated with adhesive tape instead of sutures. The wound heals rapidly.

Answer.—No positive time interval can be established for extubation following tracheotomy for any cause. In acute nondiphtheritic laryngitis, tracheotomy is done to pipe air into the trachea below an obstructed larynx and the tube can be removed only after the obstructing edema and inflammation subsides. This may take 2 or 3 days or a week to 10 days. It is usually more difficult to extubate small infants than older persons, because slight, persisting edema in a small infant causes a high degree of airway obstruction, whereas the same persisting edema in the larynx of an older person will cause a relatively small degree of obstruction.

Several extubation procedures are available. The simplest is to reduce the size of the tracheotomy tube during the daily changes of the tube after the third or fourth day; for example, if the patient was tracheotomized with a no. 2 tube, the tube size should not be changed until toxemia subsides and the temperature and condition of the chest are normal, which, in acute nondiphtheritic laryngitis, usually occur after the third to the seventh day. Then a no. 1 tube should be substituted for the no. 2 and on the next day a no. 0 tube for the no. 1, provided there is no return of obstructive symptoms. Finally, the small tube may be corked with a partial or complete rubber cork or the inner tube may be closed with an adhesive tape cover to encourage resumption of mouth breathing. In the meantime, the fistulous tract for reinsertion of the tube in case the airway is not yet adequate is maintained. If the patient can sleep through the night with the tube plugged, the tube may be removed and the fistula will usually close spontaneously. Close observation of the patient is necessary during this time so that the airway may be reopened, if necessary, by removing the rubber cork or the adhesive tape covering the inner tube. Another technique is to substitute a small diameter blind extubation plug for the tracheotomy tube for a 24 hour test period after the acute process has subsided, thus maintaining the tracheostomy while reestablishing mouth breathing.

MEPHENESIN CONTRAINDICATED IN NERVOUS DRIVERS

To the Editor:—*A professional driving instructor is confronted with a large number of nervous students. Many of these students have acute symptoms, with noticeable tremors and increase in tension, at the time of their examination by the motor vehicle department for a driver's license. Would mephenesin be useful in decreasing the nervous tension and thus improving the driving of these students at the time of examination? What dangers, if any, would be involved in its use?* *John H. Kennard, M.D., Manchester, N. H.*

Answer.—Mephenesin was introduced by Berger and Bradley, in 1946, as a drug capable of producing muscular relaxation without loss of consciousness. Kaada's studies showed the site of action of the drug to be the internuncial neurons of complex polysynaptic circuits. Gammon and Churchill have shown that there is also depression of subcortical and cortical activity. The drug has been used in a variety of clinical syndromes with varying degrees of success. It has been noted to reduce tremors and to produce a sedative effect in certain cases. The drug is not, however, without side-effects; the most frequently observed have been sleepiness and dizziness. Laurence has also reported temporary collapse, fecal incontinence, and vomiting following ingestion of the drug. It would appear, therefore, that while some of the described symptoms might be alleviated by this drug, the side effects would contraindicate its use by anyone driving an automobile.

PERIODIC DIARRHEA

To the Editor:—*A patient has had repeated episodes of diarrhea, which are apparently caused by Endolimax nana infection. The patient has received aureomycin and several courses of diiodohydroxyquinoline (diodoquin®), with no permanent cure resulting. Inevitably, the cysts of the organism reappear in one to three months. I would appreciate suggestions regarding treatment.*

M.D., Illinois.

Answer.—Endolimax nana is not generally considered to be a pathogen, and one should be cautious about attributing gastrointestinal symptoms to this parasite. It is commonly found in routine examination of stool specimens. If thorough gastrointestinal studies, including a complete roentgenographic series, gastric analysis, and proctosigmoidoscopy as well as examination of the stools for fat content and muscle fibers, do not reveal a demonstrable organic cause for the diarrhea, psychogenic factors should be considered in the diagnosis of relapsing or periodic diarrhea.

The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

REDUCING DIET FOR HYPOGLYCEMIC PATIENTS

To the Editor:—*Please outline a reducing diet for a patient with hypoglycemia.* F. H. Corpening, M.D., Horse Shoe, N. C.

Answer.—The diet for an overweight patient with hyperinsulinism (spontaneous hypoglycemia) differs little from the low-calorie (1,250 calories) reducing diets generally prescribed by physicians. It should be low in carbohydrates and high in proteins, with a limited amount of fats. The ingested food should be divided into three small meals a day, an egg for breakfast, fish or other seafood, or cheese and crackers for lunch, and lean meat in liberal quantities for dinner. An abundance of vegetables containing 3 or 5% carbohydrate may be eaten for lunch and dinner. Fruit or fruit juices (orange, apple, or grapefruit) should be taken for breakfast, every two or three hours between meals, at bedtime, and, if awake, during the night.

The following diet has been used successfully in many overweight hyperinsulin patients:

Breakfast.—One fruit: half a grapefruit, an orange, or orange juice, half of a cantaloupe, peaches, or any other fruit in season, without sugar; one egg, cooked any way except fried; one slice of whole wheat bread toast; one pat of butter; and one cup of coffee without sugar if one usually drinks coffee.

Dinner.—Soup: cold or jellied chicken or beef consomme or tomato juice. Green vegetables (one or two varieties): large serving of spinach, turnip greens, mustard greens, tender string beans, cabbage, squash, egg plant, okra, carrots, cauliflower, brussels sprouts, or onions, cooked without grease. Lemon juice may be used freely on vegetables after they have been cooked.

Meat: a large portion of chicken, turkey, mutton, roast beef, tender steak, thinly sliced boiled ham, or fish, cooked without much grease. Trim fats from meats. Bread: one slice of whole wheat bread, dry toast, a small piece of country-ground corn meal bread, or one small corn muffin.

Dessert: peaches, baked apple, or other fruit or berries with milk but no sugar, cantaloupe, a small piece watermelon, or other fruit desserts.

Luncheon or Supper.—Fish, shrimp or other seafood, or any cheese. Fruit or uncooked vegetable: large serving of lettuce, cole slaw, tomato, grapefruit, or waldorf salad. French dressing may be used. Celery and radishes. Bread: one slice of whole wheat bread or toast, and one pat of butter. A glass of buttermilk or skimmed milk. Dessert: an orange, cantaloupe, baked apple without sugar, or other fresh fruit desserts without sugar may be eaten.

Regulating the life of the hyperinsulin patient is important. He should be particularly careful to avoid fatigue and requires rest in bed eight or nine hours at night and from half an hour to an hour after the noon meal. He should drink not more than one cup of coffee a day. It is particularly important to eliminate sweets and cane sugar products of all kinds, since they stimulate the secretion of insulin and are, therefore, harmful. The cola-caffeine containing soft drinks are an important cause of hyperinsulinism and should be eliminated entirely. Beer and all other alcoholic beverages, when used regularly or in excessive amounts, because of their effect on the liver, cause spontaneous hypoglycemia (relative hyperinsulinism). Total abstinence is advisable in all cases of hyperinsulinism.

INFECTIOUS HEPATITIS AND PREGNANCY

To the Editor:—*Is a therapeutic abortion indicated in a woman who is five months pregnant and has severe infectious hepatitis? She is doing well clinically, and her serum bilirubin has steadily declined from 12 mg. per 100 cc. to 2 mg.*

M.D., Tennessee.

Answer.—The only consideration is whether the hepatitis is severe enough to indicate abortion. Since this patient is improving, abortion does not appear indicated. There are apparently no reports of the disease affecting the offspring in utero. In general, if the patient is in good condition and on a high protein diet, there should be no undue strain on the liver. More rest, however, is required, and a considerable length of time is necessary for the patient to regain her strength and tolerance to exercise.

DIFFERENCE OF BLOOD PRESSURE IN THE ARMS

To the Editor:—*A man, aged 28, had been turned down for insurance because of hypertension. He has since been insured, because on succeeding occasions his blood pressure was normal. On the day he was found to have hypertension, the pressure was determined on his left arm, whereas it had always been taken on the right arm previously. The reading of his left arm was 160/105 and of the right arm, 128/80. At subsequent examinations, it has been the right arm that is hypertensive, 170/110, and the left arm has given a reading of 130/80. Is there any known cause for this phenomenon? Could it be a one-sided autonomic nervous system imbalance? Gross tests for the scalenus anticus syndrome are negative.*

Robin Smith, M.D., Neenah, Wis.

This inquiry was referred to two consultants, whose respective replies follow.—Ed.

Answer.—Differences in blood pressure in the two arms are frequent enough to warrant routine determinations on both sides. Angulation of or pressure on the aortic branches and the aortic plaques, or partial occlusion may account for differences between the two sides; also, in some patients the first determination of blood pressure yields higher figures than subsequent ones. It is likely that such a patient has increased vasomotor reactivity. He should be watched for the development of a continuous diastolic hypertension.

Answer.—It is probable that this patient consciously or unconsciously is applying pressure to the artery. It would be interesting to measure arterial pressure with the arms at different angles to the body. The suggestion that the blood pressure differences are due to autonomic imbalance seems unlikely.

PAROTITIS AND RHEUMATOID ARTHRITIS

To the Editor:—*A woman, aged 50, has a mixed type of arthritis. One year ago she had a series of 20 gold treatments. On the date of the 20th treatment, painful swelling of both parotid glands developed. The physician who gave her the gold injections told her she had mumps. The glands were so large that they produced facial disfigurement. The patient also complained of dryness of the mouth. No tenderness of the large and firm parotid glands was elicited. There was no history of infection or other glandular disease. The patient was given weekly x-ray treatments of 100 r each directed to each parotid gland through a 6 by 8 cm. port tangentially. Six treatments, a total of 605 r, were given to the left parotid gland, and seven treatments, a total of 705 r, were given to the right parotid gland. There was complete regression of the swelling in both glands and less dryness of the mouth six weeks after the last treatment. Could the gold injections have caused the bilateral chronic parotitis?*

M. L. Pindell, M.D., Beverly Hills, Calif.

Answer.—This consultant has never known disease of the parotid glands to develop as a consequence to or complication of gold therapy or rheumatoid arthritis. It is highly possible that this patient had inflammation of the parotid glands as one of the infrequent nonarticular disorders seen in patients with rheumatoid arthritis. Such disease of the salivary and lacrimal glands has been described by Sjogren and is sometimes referred to as Sjogren's disease. It is described in a recent article by Reader and co-workers (*Ann. Rheumat. Dis.* **10**:288 [Sept.] 1951).

SEROUS DISCHARGE FROM THE NIPPLES

To the Editor:—*In* The Journal *April 5, 1952 (page 1261), a query concerning a 34-year-old woman who has had at intervals a serous discharge from the nipples makes no mention of whether she has had any pregnancies. Occasionally, women who have had pregnancies show, in later years, a discharge from one or both nipples, which is nonsanguineous and in which no malignant cells can be recognized or other abnormal cytological condition detected.*

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AIRWAY MANAGEMENT IN PATIENTS WITH POLIOMYELITIS

ELECTIVE TRACHEOTOMY AND TRACHEAL INTUBATION

Hamilton S. Davis, M.D.
and
Harold F. Bishop, M.D., Valhalla, N. Y.

There is controversy, at present, concerning the place of early (so-called elective or prophylactic) tracheotomy in the supportive treatment of acute anterior poliomyelitis with severe respiratory involvement. Those who oppose its use, exemplified in the literature by Stimson [1] and Sweet,[2] claim comparable results with conservative treatment, with tracheotomy used as a last resort. Galloway,[3] Weiland,[4] Brown and associates,[5] and West and Bower,[6] on the other hand, favor it as the most effective means of combating obstruction of the airway by secretions and vocal cord palsy. It would appear the trend in recent years has been toward the latter approach, particularly in the larger poliomyelitis centers.

It seems apparent that two phases of the problem exist. Initially, the patient must be protected from early asphyxial death resulting from high respiratory tract obstruction, pulmonary edema, and hypoventilation. Later, lower respiratory tract obstruction with its subsequent atelectasis and pneumonitis, which is the frequent sequel to hypoventilation and inability to cough, must be combated. Practically all who die after surviving the initial phase do so because of pulmonary complications, in spite of antibiotic therapy.

It would seem, therefore, the treatment of choice would be that which coped most effectively with both phases. Unfortunately, reports to date have dealt predominantly with the primary disease process, describing only in a general way the morbidity and mortality arising from pulmonary complications. Such data must be reported carefully, for only by its accumulation can some answer be reached regarding effective therapy. There are so many variables that a controlled study is almost impossible; the only hope seems to lie in the accumulation of enough clinical and pathological data to permit a relatively objective conclusion.

Because of several factors, there was a swing from conservative management, at our hospital, to the use of the early tracheotomy. First, the conservative method had left much to be desired, there having been patients thus treated who, in retrospect, might have fared better with early tracheotomy. Second, we were favorably impressed with the results of tracheotomies in comatose patients, whose respiratory problems simulate those of patients with poliomyelitis. Third, we felt that the writers stressing this method presented rather convincing logic. Also, the virulence of the disease in 1949, with its high incidence of severe bulbar forms, influenced our decisions. To cope with this entire problem, a "poliomyelitis team," of which the anesthesiologist was a member, was formed. The resuscitative features presented in handling these patients, many of whom were children, led to the development of a technique that has not, to our knowledge, been reported elsewhere in the English literature. Certain aspects of this technique are original; however, it must be pointed out that many features in the over-all plan have been borrowed from the works of Galloway,[3] Brown and co-workers,[5] and Leigh and Belton.[7] The purpose of this discussion, then, is twofold: to present a treatment plan based on the use of early tracheotomies in patients with poliomyelitis and to give the results, with emphasis being placed on morbidity due to respiratory involvement as well as mortality.

From the Department of Anesthesiology, Grasslands Hospital.

Read in part before the Annual Meeting of the American Society of Anesthesiologists, Inc., New York, Dec. 10, 1949.

1. Stimson, P. M.: The Diagnosis and Treatment of Bulbar Poliomyelitis, South. M. J. **42**: 415-420 (May) 1949.
2. Sweet, L. K.: Acute Anterior Poliomyelitis: Diagnosis, Differential Diagnosis and Treatment of Bulbar Paralysis and Respiratory Failure, M. Ann. District of Columbia **14**: 501-504 (Nov.) 1945.
3. Galloway, T. C.: Management of Respiratory Complications of Poliomyelitis, Arch. Otolaryng. **46**: 125-136 (Aug.) 1947.
4. Weiland, A. H.; Burbacher, C. R., and Keiser, R. P.: Management of Bulbar Poliomyelitis, South. M. J. **40**: 157-161 (Feb.) 1947.
5. Brown, J. R; Baker, A. B., and McQuarrie, I.: Bulbar Form of Poliomyelitis: Therapeutic Measures Based on Pathologic and Physiologic Findings, J. A. M. A. **135**: 425-428 (Oct. 18) 1947.
6. West, H. E., and Bower, A. G.: Treatment of Poliomyelitis Involving the Respiratory System, Am. J. M. Sc. **217**: 252-255 (March) 1949.
7. Leigh, M. D., and Belton, M. K.: Pediatric Anesthesia, New York, The Macmillan Company, 1948, pp. 142-143.

DEFINITIONS

It is appropriate, at this time, to list certain definitions as we have employed them in our study.

Disease Classification.—Three types of acute poliomyelitis have been recognized in our series of cases: spinal, bulbar, and bulbospinal. "Encephalitic" manifestations have been deliberately excluded because of their questionable clinical differentiation, in most instances, from hypoxic reactions. That there is actual invasion of the cerebral hemispheres by the disease process in certain cases has been demonstrated pathologically by Brown and co-workers[5] and Bodian[8] and in our own material. However, whether such minimal involvement as has been described in these regions is of clinical significance is debatable. There were no so-called pure encephalitic forms in this series.

1. Spinal Type: This type indicated involvement of spinal nerves only, as determined clinically. All patients coming to autopsy revealed involvement of higher centers; the diagnosis of spinal type was, of necessity, a clinical diagnosis only.

2. Bulbar Type: This type indicated involvement of medullary centers and cranial nerves only, as determined clinically or pathologically.

3. Bulbospinal Type: This type indicated involvement of medullary centers, cranial nerves, and spinal nerves, in combinations, as determined clinically or pathologically. This type predominated.

Pulmonary Complication.—The diagnosis of pulmonary complication was made whenever clinical, roentgenographic, or pathological findings indicated significant involvement of the lung parenchyma. Included also was severe tracheobronchial disease; not included were minor degrees of tracheobronchitis associated with the expected trauma of the tracheotomy tube and the frequent tracheal suctioning. For the sake of this study, borderline conditions were considered as pulmonary complications; the figures presented are, therefore, maximum.

TREATMENT

Our treatment schedule may be divided into three periods for the sake of description: the evaluation period, the anesthesia-operative period, and the postoperative period.

Evaluation Period.—When a patient has respiratory dysfunction, whether it be spinal or bulbar in origin, he is observed closely by the poliomyelitis team. This team is composed of appropriate representatives of the medical or pediatric, neurology, otolaryngology, and anesthesiology services. In the evaluation period conservative measures of positioning, suctioning, oxygenation, hydration, administration of antibiotics, and proper nutrition are practiced by special nurses. During this time, the patient is examined frequently for progressing disease; it is emphasized that respiratory failure represents a concrete danger, whereas the "fatigue" secondary to such examinations is largely hypothetical. If the team decides the patient is becoming worse with conservative therapy, a tracheotomy is performed in the operating room. After this, he is placed in the respirator, if indicated. Indications for tracheotomy and use of the respirator are similar to those first outlined in 1943 by Galloway[9] and emphasized by others since. This is, by choice, an elective procedure. The "last-ditch" or "stab" tracheotomy is mentioned only to condemn it, or, at best, to relegate it to emergency conditions in the field in which proper equipment and personnel may not be available.

Anesthesia-Operative Period.—When the team decides that tracheotomy is advisable, a belladonna drug is given to facilitate the subsequent intubation of the trachea. Sedation is not necessary in these cases and may be dangerous. The anesthesiologist decides whether the patient is in immediate need of oxygen only, of a patent airway, or both. If he shows minimal obstruction and is getting along reasonably well with nasal administration of oxygen, he is transported to the operating room with oxygen.

With airway obstruction in a conscious patient, endotracheal intubation is accomplished on the ward with topical anesthesia, 10% cocaine being carefully applied to the pyriform fossa bilaterally through pledgets on curved laryngeal forceps. Only minimal anesthesia is necessary in most cases, because of the diminished gag reflex. It is imperative that a good source of suction be available during this time, because of the hypersecretion present. Tracheal intubation under direct laryngoscopy is preferable to the blind nasal method, as the latter frequently necessitates several attempts, thus increasing the possibilities of trauma and hypoxia. The curved MacIntosh laryngoscope blade has proved the most effective for these intubations. Magill endotracheal forceps for directing the catheter should always be at hand.

In those semiconscious or unconscious patients who need rapid resuscitation, it is neither necessary nor advisable to waste time with cocainization; intubation is carried out as quickly as possible and is not infrequently lifesaving. The nasotracheal route, with direct laryngoscopy, is useful on the ward, as the tube is less likely to become dislodged while the patient is en route to the operating room than if orotracheal intubation is used. The airway thus assured, there is almost always spontaneous improvement in the condition of the patient, often with a return to consciousness. The endotracheal tube is well tolerated without further anesthesia, although "bucking" is always present with suctioning attempts down as deep as the carina. If necessary, respirations are compensated by manual pressure on the breathing bag of a closed to and fro carbon dioxide absorption system connected to a source of oxygen. The danger of asphyxia now past, the patient is taken safely to the operating room on a stretcher equipped with oxygen and capable of being placed in the head-down position, the latter facilitating removal of secretions and vomitus.

Tracheotomies have long been done with local anesthesia with or without a bronchoscope in the trachea. It is felt this is not the safest procedure in patients who, by the nature of their disease, have reduced ventilatory capacity. It is our impression that much of the criticism leveled at the use of tracheotomies in these cases stems from the use of this method. Although local anesthesia has undeniable advantages in the proper situation, it can

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8. Bodian, D.: Histopathologic Basis of Clinical Findings in Poliomyelitis, Am. J. Med. 6: 563-578 (May) 1949.

9. Galloway, T. C.: Tracheotomy in Bulbar Poliomyelitis, J. A. M. A. 123: 1096-1097 (Dec. 25) 1943.

be completely unsatisfactory in terrified, uncooperative patients, especially children. Fear and struggling lead to increased oxygen requirement, trauma with the bronchoscope, and hurried bloody tracheotomies, during which time the circulating epinephrine of the surgeon, as well as the patient, probably reaches considerable levels. For this reason, general anesthesia with the gaseous agents has been adopted in our clinic and has proved satisfactory. Cyclopropane, ethylene, and, on occasion, nitrous oxide are used. These can be applied in such combinations as to provide rapid quiet induction, light maintenance, high oxygen tensions, and almost immediate recovery. They have relatively transient effects on the various organs and are not respiratory tract irritants in the concentrations used.[10] The use of cyclopropane might be objectionable on theoretical grounds in those patients with myocarditis, which is frequently a finding associated with this disease in its severer forms. However, in clinical practice, to date, no ill-effects have been noted, and this gas has been the primary agent in every case. Of interest is the ease of induction and maintenance of anesthesia with minimal amounts of agent in this group of anxious, febrile young patients without premedication other than the use of belladonna drugs. This paradox apparently reflects the altered oxidation processes in the central nervous system in patients with poliomyelitis; it warns against overdosage during the anesthetic management.

In the induction of anesthesia, the breathing bag is filled with oxygen, and then a flow of equal volumes of oxygen and cyclopropane is introduced, usually 500 cc. of each in the adult and smaller volumes in small children, until consciousness is lost. An endotracheal tube is, of course, already in place, having been inserted either on the ward or in the operating room by means of the technique previously described. After induction, a constant flow rate of 5% to 20% cyclopropane with oxygen is maintained, depending on the patient's requirements. A to and fro absorption system has proved to be most adaptable with regard to reduction of dead space and breathing resistance to a minimum, portability features, and ease of sterilization; no doubt a circle absorption method could be adapted satisfactorily. In the subsequent sterilization of the equipment, it might be mentioned here, a dilute formaldehyde solution in alcohol is used for rubber and plastic parts, metal parts being autoclaved.

The surgeon now enters the trachea through a surgical field in which the aim is careful hemostasis. The site of the tracheotomy is, of course, the surgeon's choice. The highest safe tracheotomy, usually through the third tracheal ring, has proved best for subsequent respirator care. At this time the tip of the endotracheal tube is withdrawn just proximal to the tracheal window created. Anesthesia is continued with a high gas flow to compensate for the opening. A mixture of 45% ethylene, 5% cyclopropane, and 50% oxygen is about right, with a total flow of 2 to 5 liters per minute. The trachea is aspirated by the surgeon and the appropriate size of tracheotomy tube inserted. A thin-walled endotracheal catheter, which will fit the tube snugly, is chosen from a previously sterilized set of graduated sizes. The distal end is fitted into the tracheotomy tube by the surgeon; the proximal end is passed through the drape opening to the anesthetist, who attaches it to the canister by means of the usual connectors. Care is exercised to keep dead space minimal. Closed anesthesia is then resumed with a small flow of gasses. One must be careful, at the above stage, not to exert undue positive pressure in the system; mediastinal emphysema could result. It did not occur in this series. The wound is closed in layers and dressed and the flow of anesthetic agent terminated. The patient is awake in a few minutes unless other factors intervene. If he is breathing adequately, he is returned to the ward with oxygen being given through a small catheter in the tracheotomy tube opening. If the respirator is necessary, manually assisted respirations are continued by means of the still attached canister and bag until the patient is placed in the machine.

Postoperative Period.—Survival now depends on two factors: progression of the disease and respiratory complications. At present, we have no control over the former; treatment is directed toward preventing the latter. It has been found that the patients with bulbar disease whose main problem is one of obstructed airway and aspiration, get along well once a free airway is established by means of their tracheotomy. It is felt that with this prophylactic tracheotomy the patient has a greater chance to escape the dangers of aspiration and is offered the best possibilities of vigorous treatment if they do occur. One patient (J. W., table 3) was unquestionably saved from an asphyxial death on two separate occasions during his convalescence, when he vomited and aspirated large quantities of tube feedings. Only prompt evacuation of his tracheobronchial tree by means of a catheter through his tracheotomy and the head-down position saved his life. Equally gratifying was the lack of detectable respiratory complications following these two episodes.

When the respirator enters the picture, the outlook is graver. We have used both cuirass and cylinder type respirators. The former has proved an adjunct to, but not a substitute for, the latter. Its nursing advantages are obvious, but, in our hands, the respiratory exchange has not approached that of the cylinder type. The use of a curved metal brace to push the rubber collar into the machine, leaving the tracheotomy outside and easily accessible, has greatly facilitated care of the airway when the cylinder respirator is used. Such braces are available commercially. In addition, as mentioned previously, a rather high tracheotomy is preferable, so that it is as far outside the collar as possible. Too high tracheotomy is to be avoided, however, because of the danger of subsequent laryngeal stenosis, a formidable complication.

To reduce pulmonary complications in the respirator, it is axiomatic that patent tracheobronchial passages and near normal mucous membranes be maintained. That by-passing the upper respiratory tract disrupts certain important physiological processes cannot be denied. The aim is to lessen this insult so that the advantages outweigh the disadvantages. To this end, the nurses are instructed individually in the essentials of proper care for these patients. This includes the correct method of suctioning the trachea, mouth, and nose; the systematic rotation of the patient through the lateral, Fowler, and Trendelenburg positions; the desired oxygen flow rates and humidification; the appropriate respirator pressures and rates; and the care of the patient's skin. The symptoms and signs of hypoxia are reviewed for them. In short, painstaking attention to detail is taught.

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[10] Adriani, J.: The Pharmacology of Anesthetic Drugs: A Syllabus for Students and Clinicians, ed. 2, Springfield, Ill., Charles C Thomas, Publisher, 1942, pp. 17-24.

A few points on care of the tracheotomy might be mentioned. The otolaryngologist of the team uses a rather long incision, 3 to 4 cm. in adults, closes the wound in layers, and, therefore, does not remove the outer tube initially in less than 72 hours, thus insuring adequate canalization. Subsequently, it is removed, cleaned, and replaced daily. The inner tube is removed and cleaned at least every three hours. Suctioning through the tracheotomy is done by means of low pressures through sterile, terminal orifice, soft rubber catheters. This is carried out rapidly to avoid marked reductions in arterial blood oxygen saturation.

Bronchoscopy is easily performed through the tracheal stoma, even with the patient in the cylinder respirator; the cuirass respirator facilitates this procedure and has proved valuable in this respect. Bronchoscopic aspiration is used without hesitation and, if necessary, repeatedly, when atelectasis occurs. Pounding the chest at intervals is helpful in dislodging thick secretions. Adequate hydration is an absolute necessity to keep secretions thin; to insure this, intravenous polyethylene catheterizations by cutdown have been used almost routinely during the first 48 hours in the respirator, after which tube feeding is attempted. The use of belladonna-like drugs, at this point, is contraindicated. It may be helpful to administer potassium iodide to liquefy secretions.

TABLE 1.—*Child and Adult Tracheotomy-Respirator Cases—1948*

Patient	Age (Yr.), Sex	Type of Disease	Treatment*		Time in Respirator		Pulmonary Complications		Result	
			Tracheotomy	Respirator	Less Than Two Weeks	More Than Two Weeks	Yes	No	Lived	Died*
J. T.	20, F	Bulbospinal	HD 4	HD 1 †	..	x	x	..	x	......
L. F.	4, M	Bulbar	HD 0	HD 1	x	..	..	x	x	......
D. M.	16, M	Spinal	HD 3	HD 3 †	x	..	..	x	x	......
B. M.	24, F	Bulbospinal	HD 2	HD 2	..	x	x	.	x	......
K. O.	14, F	Bulbospinal	HD 2	HD 0 †	x	..	x	..	..	HD 2 ‡
D. H.	34, F	Bulbospinal	HD 1	HD 1 †	x	..	x	..	..	HD 11 ‡
Total	.....	....	6	6	4	2	4	2	4	2

* HD = hospital day; HD 0 = day of admission. † Respirator treatment preceded tracheotomy. ‡ Complete autopsy.

Curare type compounds are occasionally of great value in the treatment of patients who exhibit such intercostal muscle spasm as to reduce tidal exchange significantly. Intravenous administration of small repeated doses, of the order of 3 to 6 mg. (1 to 2 cc.) of *d*-tubocurarine hydrochloride, or similar drugs in comparable doses, usually suffices; larger doses may be given without fear of adverse respiratory effects, if necessary, in a tracheotomized patient in the respirator. However, histamine-like reactions on the vascular system may follow the administration of large doses of *d*-tubocurarine, so that caution is advisable. Sedation may be used, when it is felt necessary, at this stage in the management of the patient. It was seldom needed in our series. It is contraindicated in the early stages of the illness as mentioned previously.

Weaning from the respirator is a complex procedure in some patients, requiring the utmost ingenuity on the part of the physician. The psychic factors involved are by far the most important. The patient rapidly acquires a dependency on the machine, which increases as time passes. In general, it has been the policy to get them out, for gradually increasing time intervals, at the earliest possible moment. This may mean within a few hours after entry in some instances. Tracheotomies are closed when the patient no longer depends on the machine or, in cases of bulbar disease, when protective pharyngeal and laryngeal reflexes have returned.

Antibiotics are used freely. It is a well-known fact, however, that their use will not prevent infection in portions of the lung that are not aerating properly and from which secretions cannot drain because of obstruction in the bronchial tree.

Paralyzed excretory functions are supported by the use of bladder catheterization and enemas until normal function is restored. The value of the use of stimulating drugs for these functions is questionable.

RESULTS

There were 229 cases of diagnosed acute poliomyelitis in our hospital during the seasons of 1948 and 1949 (83 cases in 1948 and 146 in 1949). The ratio of males to females over the two-year period was 53% to 47% and that of children to adults 60% to 40%. The latter figures are based on adulthood placed at 12 years for females and 16 years for males. All the patients were white.

In 1948, there were 18 (22%) patients with bulbur disease, the majority of these also having associated spinal paralysis. In addition, one patient had severe intercostal paralysis without bulbar signs. There were, therefore, 19 (23%) seriously ill patients. Six (7%), including one child and five adults, were placed in the respirator (table 1). All six had tracheotomies, two before being placed in the machine and four afterward, because of obvious respiratory problems. No other tracheotomies were performed in 1948.

In the 1948 series, two patients (2.5%) died. Both had bulbospinal disease with pulmonary complications proved at autopsy; both had been placed in the respirator without preceding tracheotomies. Of the four who survived, two had clinical evidence of pulmonary complications at some time during their illness. Therefore, 66% of respirator patients had respiratory complications. It is of interest that the two patients who were forced to remain in the machine over two weeks both survived and that neither showed evidence of pulmonary complications at any time.

In the outbreak in 1949 there were far more virulent characteristics, in addition to almost double the total number of cases. Thirty-nine (27%) had bulbar involvement, the majority associated with spinal paralysis. Five others had severe intercostal paralysis without bulbar signs, which brings the total to 44 seriously ill, or 30%. Fifteen (10%), including six children and nine adults, were placed in the respirator. (tables 2 and 3). All patients who were placed in respirator had tracheotomies, 12 before being placed in the machine and the remaining 3 later. There were, in addition, three tracheotomies performed on patients who did not require the respirator. The total number of patients subjected to tracheotomy in 1949 was 18.

TABLE 2.—*Child Tracheotomy-Respirator Cases—1949*

Patient	Age (Yr.), Sex	Type of Disease	Treatment*		Time in Respirator		Pulmonary Complications		Result	
			Tracheotomy	Respirator	Less Than Two Weeks	More Than Two Weeks	Yes	No	Lived	Died*
W. B.	8, F	Bulbospinal	HD 0	HD 0	..	x	x	..	..	HD 17 ‡
J. P.	7, F	Bulbospinal	HD 1	......	..	..	x	..	x	......
G. V. B.	9, F	Bulbospinal	HD 0	HD 0	x	..	x	..	..	HD 3 ‡
F. D.	11, F	Spinal	HD 1	HD 0 †	x	..	x	..	x	......
P. R.	10, F	Bulbar	HD 0	HD 0	x	..	..	x	x	......
J. S.	4, F	Bulbospinal	HD 1	HD 1	x	..	x	..	..	HD 4
B. N.	5, M	Bulbospinal	HD 1	HD 1	x	..	x	..	..	HD 1 ‡
Total	.....	....	7	6	5	1	6	1	3	4

* HD = hospital day; HD 0 = day of admission. † Respirator treatment preceded tracheotomy. ‡ Complete autopsy.

TABLE 3.—*Adult Tracheotomy-Respirator Cases—1949*

Patient	Age (Yr.), Sex	Type of Disease	Treatment*		Time in Respirator		Pulmonary Complications		Result	
			Tracheotomy	Respirator	Less Than Two Weeks	More Than Two Weeks	Yes	No	Lived	Died*
M. M.	32, F	Bulbospinal	HD 4	HD 4	x	..	..	x	x	......
A. C.	27, F	Bulbospinal	HD 1	HD 1	x	..	..	x	..	HD 2 ‡
H. J.	29, F	Bulbospinal	HD 2	HD 2 †	..	x	..	x	x	......
M. F.	30, F	Bulbospinal	HD 2	HD 2	x	..	x	..	..	HD 4 ‡
R. W.	18, F	Bulbospinal	HD 0	HD 1	..	x	x	..	x	......
J. W.	33, M	Bulbar	HD 0	......	..	..	..	x	x	......
K. R.	24, F	Bulbospinal	HD 3	HD 3	..	x	x	..	..	HD 41 ‡
D. C.	24, M	Bulbospinal	HD 2	HD 2 †	x	..	x	..	..	HD 7 ‡
J. E.	15, F	Bulbospinal	HD 1	HD 1	..	x	..	x	x	......
J. C.	22, M	Bulbar	HD 0	......	..	..	? §	?	..	HD 0
J. D.	24, F	Bulbospinal	HD 0	HD 0	x	..	..	x	x	......
Total	.....	....	11	9	5	4	4	6	6	5

* HD = hospital day; HD 0 = day of admission. † Respirator treatment preceded tracheotomy. ‡ Complete autopsy. § Patient died a few hours after admission; no autopsy.

In the 1949 series, 11 patients (7.5%) died; all had bulbar involvement, the majority with associated spinal paralysis. Eight of the 15 placed in respirators died; seven of these deaths were associated with pulmonary complications. Of the seven respirator patients who survived, three had similar complications. From these figures it is evident that 10 (66%) of the 15 respirator patients had complicating respiratory infections. Of the 18 patients subjected to tracheotomy, 9 (50%) died; in 10 (55%) the disease was complicated by respiratory infections. There were five patients in whom the machine was required for more than two weeks; two died and three lived. Both patients who died had pulmonary complications at autopsy; of the three survivors, two had no evidence of respiratory infection at any time.

Two facts are apparent in these 229 cases. Of the 24 most critically ill, 18 (75%) were females and 16 (66%) were adults. This in spite of the fact that males and children predominated in the series as a whole. As might be expected with the above ratio of seriously ill, of the 13 mortalities (5.7) over the two years, the majority were females and adults; 10 were females and 8 were adults.

Eleven of the 13 patients who died had complete autopsies. Two of these had evidence of major atelectasis; six others showed, in varying degrees, minute atelectatic foci in the peripheral lung fields, lobular pneumonia, and pulmonary congestion and edema that was frequently hemorrhagic. It was believed that, with one or two exceptions, these deaths were due to central nervous system failure, not pulmonary disease. Many were sudden and unexpected in patients who seemed to be getting along not too badly. The commonest sites of nervous system involvement were the spinal cord anterior horn cells, the floor and roof of the fourth ventricle, and the bulbar reticular formation. Generalized hemorrhagic manifestations were present in some of the patients; however, these may have been agonal. Nine of the 11 showed myocarditis of varying degrees.

SUMMARY

Two hundred twenty-nine patients with acute anterior poliomyelitis were admitted to a county hospital during the seasons of 1948 and 1949. The outbreak of 1949 in

hematologic effects. There were no demonstrable beneficial or harmful effects.

Hematology: The hematologic studies included erythrocyte, leukocyte, differential, platelet, reticulocyte, and eosinophil counts; sedimentation rates and hematocrit determinations; and bleeding and coagulation times. These tests were made routinely before the beginning of the treatments, on the day of each treatment, and at the conclusion of the treatment series. Two patients were checked daily for one week after the first treatment; five patients were checked every other day for two weeks; and several were examined at half-hour intervals for one and a half hours, then hourly for four hours following treatment. In three cases blood was removed for study from the tube leading to the irradiation chamber before exposure and again from the tube leading away from the chamber after irradiation. No significant blood changes were revealed by these studies.

Diagnostic Groups: Twelve diagnostic groups of patients with the following conditions were chosen for treatment: 23 with infectious hepatitis, 8 with stasis ulcers, 7 with arthritis, 2 each with acute rheumatic fever, pneumonitis, thrombophlebitis, and acute leukemia, and 1 each with lymphosarcoma, chronic brucellosis, sickle cell anemia with leg ulcers, atypical hemolytic anemia with leg ulcer, and herpes zoster.

TABLE 1.—*Summary of Data in First Study (Fifty-One Patients)*

	No. of Patients	Maximum No. of Treatments per Patient	Minimum No. of Treatments per Patient	Average No. of Treatments per Patient	Maximum Days from First to Last Treatment	Minimum Days from First to Last Treatment	Average Days from First to Last Treatment
Infectious hepatitis	23	9	1	4.12	42	0	15.7
Stasis ulcers	8	18	1	6.5	147	0	53
Arthritis	7	11	1	5.7	66	0	31
Acute rheumatic fever	2	10	8	9	37	33	35
Pneumonitis	2	2	1	1.5	3	0	..
Thrombophlebitis	2	2	1	1.5	3	0	..
Acute leukemia	2	2	2	2	4	4	4
Lymphosarcoma	1	33	..	....	241	..	..
Chronic brucellosis	1	8	..	....	34	..	..
Sickle cell anemia with leg ulcers	1	7	..	....	29	..	..
Atypical hemolytic anemia with leg ulcer	1	2	..	....	3	..	..
Herpes zoster	1	12	..	....	1	..	..

Infectious hepatitis. In the group with infectious hepatitis frequent liver profile studies as well as physical examinations were made. All patients with hepatitis were at bed rest in the hospital during the treatments and received high protein, high vitamin diets. Three volunteered the information that they felt better after the treatments. None was made worse. The liver profiles were checked periodically, and all, including the controls, showed improvement. The group with hepatitis presented an unusual opportunity in a controlled experiment. The patients were chosen from a large group of young men who presumably had the same source of infection, the disease having been spread in the group by the use of unclean, contaminated needles during a "heroin party." One patient of the group with hepatitis received seven treatments in which all the steps of the irradiation procedure were performed, but the shutter was kept closed to exclude the ultraviolet rays from the blood. There was no demonstrable difference.

Stasis ulcers. In the group with stasis ulcers, one patient was at bed rest in the hospital, whereas the others were ambulatory. Four reported subjective improvement and decrease in pain, and in five the ulcers decreased in size during treatment. In the hospitalized patient, the ulcers almost completely healed but recurred when he increased his activity even though the treatments were continued. Five patients eventually discontinued treatment of their own accord.

Acute rheumatic fever. Subjective improvement occurred in both patients with acute rheumatic fever. The sedimentation rate was unaffected in either. Both were at bed rest in the hospital.

Pneumonitis. Neither of the two patients with pneumonitis was in the hospital long enough for evaluation.

Brucellosis. The patient with brucellosis was not clinically benefited, and the treatments were eventually stopped because of complicating hepatitis.

Lymphosarcoma. The case of lymphosarcoma was most instructive and emphasizes the difficulty of clinically evaluating the efficacy of the hemoirradiation method. The patient had a subjective feeling of well-being after almost all treatments but occasionally reported nervousness. This patient received 33 treatments with 10 control irradiations interspersed among them. During these 10, the entire procedure was performed, but the shutter was kept closed in order to exclude the ultraviolet rays from contact with the blood. The patient was never told that from time to time this would be done and assumed that all treatments were similar. Her subjective response to both control and regular treatments was the same. She felt stronger, "more alive," and invigorated by the procedures and was disturbed when they were discontinued. At no time, however, was there any objective evidence of either clinical or hematologic improvement.

Miscellaneous. The patients with sickle cell anemia and leg ulcers, atypical hemolytic anemia with leg ulcers, and herpes zoster showed no improvement.

Summary of First Study: The number of control experiments was deliberately limited, because only positive results needed control and only subjective responses needed evaluation. A general summary of the number of treatments and the duration of the cause of treatments in the several diagnostic groups is given in table 1.

Evaluation of Second Hemoirradiation Project.—In July, 1950, the blood irradiation project was resumed by the Hematology Department of Cook County Hospital. The second series included a total of 17 patients with diagnoses of rheumatoid arthritis (2 patients), thrombophlebitis (1 patient), rheumatic fever (2 patients—one with chorea), and pelvic inflammatory diseases (12 patients).

The total number of treatments was 62, averaging almost 4 per case. Although the number of cases in each group was small, in no one case was any objective improvement noted. Many patients stated that they felt better, regardless of whether the drawn blood was irradiated (table 2).

TABLE 2.—*Summary of Data in Second Study (Seventeen Patients)*

Patient	Age, Yr.	No. of Treatments	Amount of Blood Irradiated, Cc.	Clinical Diagnosis	Remarks
H. T.	59	8	120-200	Thrombophlebitis with ulcer, chronic, 3 yr., acute 1 week; varicose veins	No improvement objectively; lesions continued to weep
B. M.	49	5	100-200	Arthritis, 6 mo.	No improvement subjectively or objectively
F. E.	41	4	200	Arthritis, 2 yr.	Subjective improvement; at no time was there limitation of movement
L. S.	6	7	105	Acute rheumatic fever;* before treatment, systolic and diastolic mitral murmur; enlarged heart; sedimentation rate *30*; heart block (1 degree)	After first treatment sedimentation rate rose to 44; after third, severe nosebleed requiring packing; after fourth, mitral and aortic regurgitation diagnosed; sedimentation rate 30; after sixth, sedimentation rate 38, heart rate 104; after seventh, heart block (1 degree) persisted
M. McC.	8	8	105	Acute rheumatic fever and chorea, 2 weeks	Apical systolic murmur; x-ray: early mitralization; EKG normal; sedimentation rate after first treatment 15, after third 41, after sixth 23; no significant changes in heart sounds or x-ray
M. L.	38	2	150-200	Pelvic inflammatory disease, 1 mo. duration; fibroid uterus	Cramping pain in right lower abdominal quadrant; cervical discharge; no objective improvement
R. S.	32	3	120-150	Pelvic inflammatory disease 1 week's duration	Subjective improvement after first treatment; objectively no change; pain persisted after third treatment
J. D.	33	3	200	Pelvic inflammatory disease 3 weeks' duration	Lower abdominal pain; received sulfonamides 3 days and penicillin once; chills, fever continued; generalized tenderness; Bartholin abscess; acute bilateral salpingitis; blood not irradiated first two treatments (temperature elevated); blood irradiated third time, temperature down to 98 F and increased later in day; septic temperature persisted; operated on for pelvic abscess
L. F.	38	3	200	Pelvic inflammatory disease	Received no antibiotics; pain in abdomen 1 day; adnexae bilateral diffuse induration; blood not irradiated with first treatment, but subjectively and objectively better; subjectively and objectively improved after third treatment; no further vaginal discharge; no other medication
L. H.	34	2	100	Pelvic inflammatory disease and fibroid uterus	Received penicillin once before irradiation and felt better; painless; no objective improvement
L. B.	23	1	200	Chronic pelvic inflammatory disease, exacerbation 8 days, rebound tenderness all quadrants; vaginal discharge	Signed release before final evaluation possible
N. M.	?	1	160	S[illegible] lower abdominal quadrants; Bartholin abscess	Discharged after first treatment; subjectively improved
V. M. J.	*19*	*3*	*200*	Pelvic inflammatory disease	Abdominal cramps, discharge, chills, fever, one day; tenderness in both lower abdominal quadrants; after first treatment severe abdominal pain; *given penicillin* once; 3 days later process subsided; subjective improvement; objectively slightly improved at completion of treatment
R. R.	?	3	150	Acute pelvic inflammatory dis*ease*	No antibiotics; abdominal pain 2 weeks, *localized to* left lower quadrant day of admission; rebound tenderness bilaterally; subjective improvement after third treatment; objectively, adnexa tender and thickened; mass in left cul de sac; pyosalpinx
H. P.	?	3	100-150	Recurrent *bilateral pelvic in*flammatory disease; lower abdominal pain 28 hours; adnexa bilaterally tender; foul sanguinomucoid discharge	No irradiation; no antibiotics; after first treatment pain lessened; objectively somewhat improved; *marked thickening* of adnexae; some tenderness on movement of cervix; at end of third treatment left adnexa: retort-shaped mass, slightly tender; pyo*salpinx*
H. R.	34	3	200	Pelvic inflammatory disease	Subjective improvement; objectively *pelvic findings* unchanged; mass appeared in right adnexa during treat*ment*
T. C.	21	3	150	Gonococcic infection; acute pelvic inflammatory disease	Blood not irradiated; after treatments pains severe, then subsided *in two days*; no subjective or objective improvement

* Treated also with acetylsalicylic acid.

CONCLUSIONS

We have concluded that none of our patients derived benefit from the irradiation of blood with the Knott hemo-irradiator. These are at best, however, preliminary results. We do not feel that, on the basis of our observations, definitive conclusions can be drawn. It can, however, be categorically stated that, in both series studied by us, the first of 51 cases and the second of 17, no dramatic clinical or reproducible objective changes, which could be attributed to the irradiation treatments rather than the natural variation in the course of the disease or to psychic effects, could be demonstrated. On the other hand, no adverse effects were noted. A longer and more extensive program of study is warranted before in vivo ultraviolet irradiation of blood can be finally either accepted or rejected.

1825 W. Harrison St.

DEVELOPMENT OF PROTEUS AND PSEUDOMONAS INFECTIONS DURING ANTIBIOTIC THERAPY

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Within recent months antimicrobial agents capable of suppressing the multiplication of almost all of the pathogenic bacteria have been given widespread clinical trial. None of these agents has been effective against all strains of the Pseudomonas (Pyocyaneus)[1] and Proteus group of organisms. As the result of the suppression of other more pathogenic bacteria in the prophylactic or therapeutic use of the antibiotics, these relatively nonpathogenic organisms have frequently been allowed to multiply and have played an active part in the pathogenesis of infectious processes.

It is the purpose of this paper to present examples of infections due to Proteus or Pseudomonas that developed during or following therapy with the commonly used antimicrobial agents. During the past two years 56 patients have been observed with such infections. Their diagnoses are presented in the table and eight illustrative cases are described. The pathogenesis, diagnosis, and treatment of these infections are also discussed.

REPORT OF CASES

Case 1.—A 53-year-old chronic alcoholic was admitted to the hospital because of chills, fever, cough, and chest pain. The symptoms began four days before admission and progressed in spite of penicillin therapy. On physical examination there was evidence of consolidation of the lower lobe of the left lung. The liver was enlarged. On admission the white cell count was 5,250, with a predominance of polymorphonuclear leukocytes showing toxic granulations. Sputum and throat cultures revealed Klebsiella pneumoniae. X-ray studies confirmed the clinical findings of pneumonia of the base of the left lung posteriorly.

The patient was treated (fig. 1) with penicillin, dihydrostreptomycin, aureomycin, and chloramphenicol (chloromycetin®). He gradually improved, and by the fourth day of therapy K. pneumoniae could not be cultured from the sputum, the leukocyte count had risen to 18,650, and his temperature had gradually decreased. On the seventh day the patient appeared somewhat worse. The following day his condition appeared markedly worse, and he became disoriented. At this time, x-ray and physical examination revealed evidence of a spread of the pneumonia in the lower lobe of the left lung as well as involvement of the lower lobe of the right lung. Cultures of the sputum revealed Pseudomonas aeruginosa in pure culture, except for an occasional colony of Candida albicans (Monilia). The patient's condition became progressively worse in spite of an increase in the dose of dihydrostreptomycin.

In vitro sensitivity tests indicated that the strain of Pseudomonas was resistant to penicillin, streptomycin, chloramphenicol, and aureomycin, but inhibited by 1.9 μg per milliliter of polymyxin B. The antibiotics being administered were discontinued as the patient was given polymyxin B intramuscularly in doses of 50 mg. every six hours. Within 48 hours the patient began to improve, and there was gradual subsequent resolution of the pneumonia, decrease of the fever, and reestablishment of the normal flora of the sputum. On the eighth day of polymyxin therapy the dose was reduced to 25 mg. every six hours. The drug was continued for a total of 15 days, during which results of daily examinations of the urine and blood and determinations of blood urea nitrogen and urinary output were normal except for occasional traces of albumin. The patient made an uneventful recovery and has remained well.

Case 2.—A 42-year-old woman who worked as an office supervisor had had diabetes since 1927, but had otherwise been well until December, 1949. During the Christmas holidays of that year she had an acute upper respiratory tract infection complicated by a purulent sinusitis. She was treated with penicillin and irrigations intermittently for a month. On the first of February she developed chills and fever, with urinary frequency and burning. She was found to have pyuria and was treated for two days with aureomycin. She improved and returned to work, but the following day there was a recurrence of symptoms. She responded to aureomycin therapy again but in a few days relapsed. During the next month there were eight episodes of urinary infection, which responded to aureomycin but recurred when it was discontinued. The ninth course of aureomycin failed to control elevation of temperature and urinary symptoms, and the patient was referred to a urologist. She was treated with penicillin and streptomycin with improvement. Three weeks later there was a return of the symptoms, and she was admitted to the hospital.

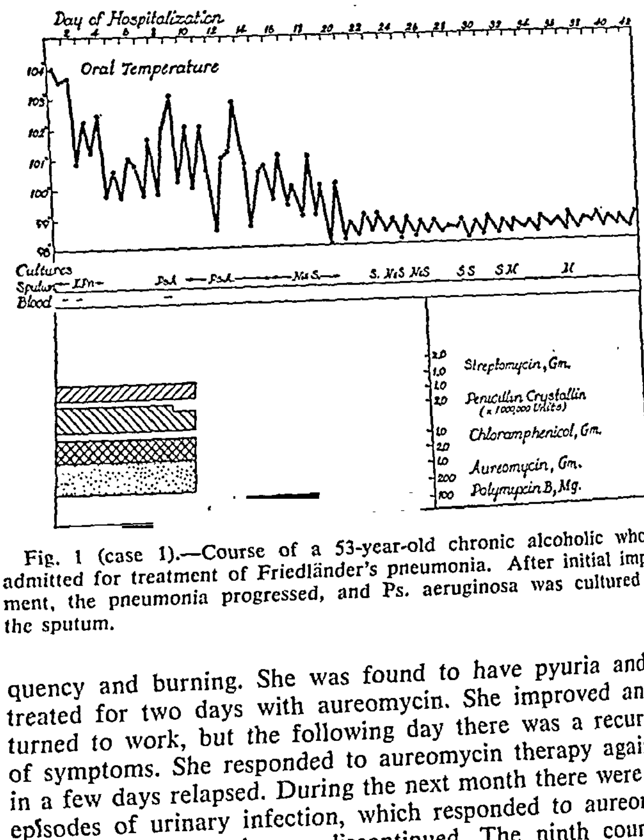

Fig. 1 (case 1).—Course of a 53-year-old chronic alcoholic who was admitted for treatment of Friedländer's pneumonia. After initial improvement, the pneumonia progressed, and Ps. aeruginosa was cultured from the sputum.

Physical examination at this time revealed a chronically and severely ill patient. The liver was slightly enlarged, and there was bilateral costovertebral angle tenderness. Laboratory examinations revealed a moderate leukocytosis and a slight hypochromic anemia. The urine contained albumin (2+), sugar (4+), many white blood cells, and gram-negative bacilli. The blood urea nitrogen level was 107 mg. per 100 cc. Ps. aeruginosa was repeatedly isolated from the urine and blood. The patient was given sulfonamides, methenamine mandelate (mandelamine®), streptomycin, and chloramphenicol without improvement (fig. 2).

Beginning the first of July the patient received polymyxin B intramuscularly in doses of 50 mg. every four hours for 24 hours, followed by 25 mg. every four hours. The blood culture

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This article has been shortened by omission of figures 2, 3, and 7. They will be included in the author's reprints.

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[1] Stanley, M. M.: Bacillus Pyocyaneus Infections: Review Report of Cases and Discussion of Newer Therapy Including Streptomycin, Am. J. Med. 2:253 (March) 1947; 2:347 (April) 1947. Hayes, E. R., and Yow, E. M.: Meningitis Due to Pseudomonas Aeruginosa Treated with Polymyxin B, Am. J. M. Sc. 220:633 (Dec.) 1950. Schaffer, A. J., and Oppenheimer, E. H.: Pseudomonas (Pyocyaneus) Infection of the Gastro-Intestinal Tract in Infants and Children, South. M. J. 41:460 (May) 1948.

[2] Deleted on proof.

on the first day after beginning polymyxin B injections was negative and a decrease in the number of bacteria in the urine was recorded; however, cultures of the urine remained positive. Administration of polymyxin B was continued for 10 days, during which the patient's temperature returned to normal and she improved generally. She complained of numbness and tingling of her hands and face and pain at the site of the injections. These symptoms disappeared after discontinuing the drug. At the beginning of polymyxin therapy the urine contained albumin (3+). The amount of albumin in the urine decreased during treatment. At the termination of polymyxin B therapy the urine was not entirely free of pus cells, and the cultures were not sterile. On the second day after discontinuing the drug the fever returned and the abnormalities in the urine progressed. The patient was subsequently explored surgically, and multiple kidney abscesses were found and aspirated. She was then given terramycin with gradual but complete recovery.

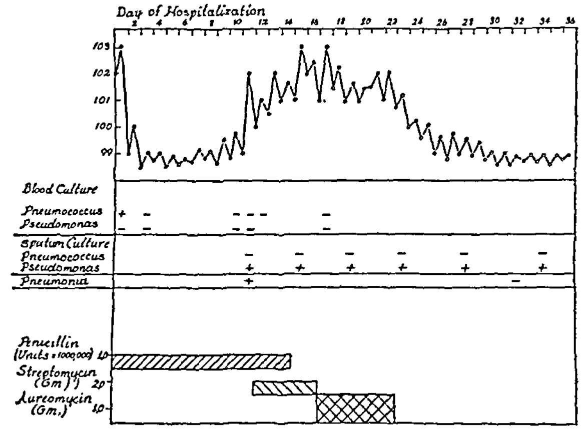

Fig. 4 (case 4).—Course of a 27-year-old woman with Pseudomonas pneumonia following pneumococcal bacteremia.

Case 3.—A 78-year-old man when first seen was comatose. An acquaintance stated that he had apparently been well until about a month previously, when urinary frequency had developed. He was given antibiotics with initial improvement, but while he was still receiving these drugs chills and fever developed. About a week before admission a productive cough developed. Examination revealed an emaciated and chronically ill man with a fever of 103 F, pulse rate of 128, and respiratory rate of 24. There were rales in both lung bases, but the remainder of the physical examination was noncontributory. The white cell count was 4,300, with 50% polymorphonuclear leukocytes; urinalysis revealed a slight trace of albumin, with 5 to 8 white blood cells per high power field. The blood urea nitrogen level was 51 mg. per 100 cc. Administration of penicillin was begun shortly after admission without improvement (fig. 3). He was subsequently given aureomycin and later streptomycin. Proteus vulgaris was isolated repeatedly from the blood and urine. This organism was found to be inhibited in vitro by 31.2 μg of chloramphenicol per milliliter, but resistant to other antibiotics tested. He was subsequently given 0.5 gram of chloramphenicol in acetyl dimethylamine, intramuscularly, every four hours. Blood levels varying between 20 and 50 μg. per milliliter were reached with temporary improvement in the patient's condition. While he was taking the drug, however, the bacteremia returned and he died on the 13th hospital day.

Case 4.—A 27-year-old Negro woman who was a chronic alcoholic was admitted to the hospital because of chills and fever. Physical examination was noncontributory. Her temperature was 103 F. The white blood count was 12,800, with 84% polymorphonuclear leukocytes. Pneumococci were isolated from the blood stream. The patient was treated with penicillin, with a prompt control of the fever and bacteremia (fig. 4). On the ninth day of penicillin therapy the fever returned, and there was clinical and x-ray evidence of pneumonia of the base of the right lung. Beginning with the 11th hospital day, the predominant organism repeatedly isolated from the sputum was Ps. aeruginosa. The penicillin was discontinued, and streptomycin and aureomycin were substituted. There was no definite evidence of the beneficial effect of antibiotic therapy. The patient improved gradually and was afebrile by the 30th hospital day. There was slight residual evidence of pneumonia at discharge.

Case 5.—A 61-year-old salesman had been found to have an adenocarcinoma of the lung and was admitted to the hospital for pneumonectomy. Prior to and following the operation, the patient was given penicillin (fig. 5). Following the operation he developed a bronchopleural fistula and empyema. Cultures of the empyema fluid contained a mixed flora, including Streptococcus hemolyticus, Micrococcus pyogenes, and species of Hemophilus and Neisseria. There was a gradual rise in temperature in spite of the administration of penicillin. On the 10th postoperative day administration of streptomycin was begun and two days later aureomycin was added. There was a prompt improvement. The fever returned, however, and Ps. aeruginosa was isolated from the pleural fluid. The previous therapy was discontinued, and on the 20th postoperative day polymyxin B was given intramuscularly (50 mg. every six hours) and intrapleurally (25 mg. daily). The patient improved slightly, but he continued to have a low grade fever and the chest continued to drain. The number of Pseudomonas colonies cultured from the chest fluid decreased with polymyxin B therapy, but the organism was not entirely eliminated. During the second week of therapy the cultures of the chest fluid revealed staphylococci and hemolytic streptococci. The patient's condition gradually deteriorated, and he died on the 47th hospital day. During the administration of polymyxin B there was no evidence of nephrotoxicity except for occasional mild albuminuria. There was, however, a terminal rise in the blood urea nitrogen to 76 mg. per 100 cc., which was considered to be prerenal and not related to the drug therapy. An autopsy was not performed.

Case 6.—An 18-year-old youth was admitted to the hospital because of hydrocephalus that had developed following a skull injury four years previously. A diagnostic pneumoencephalogram was complicated by purulent meningitis. This infection responded promptly to sulfadiazine, penicillin, and streptomycin. An operation was subsequently performed, anastomosing the subarachnoid space with the left ureter. In spite of prophylactic administration of penicillin and sulfisoxazole (gantrisin®), a spiking fever and purulent spinal fluid developed three days following the operation (fig. 6). Cultures of the spinal fluid revealed P. vulgaris; Esch. coli, P. vulgaris and Pseudomonas aeruginosa were isolated from the urine. The doses of the penicillin and sulfisoxazole were increased, and, when there was no improvement, streptomycin and chloramphenicol were added. There was no response to either of the antibiotics, and on the 23rd postoperative day polymyxin B was given intramuscularly (50 mg. every six hours) and intrathecally (2 mg. daily). The patient improved promptly and on the fourth day th
intrathecal administration of polymyxin B
The intramuscular dose of polymyxin B was
every six hours the following disc

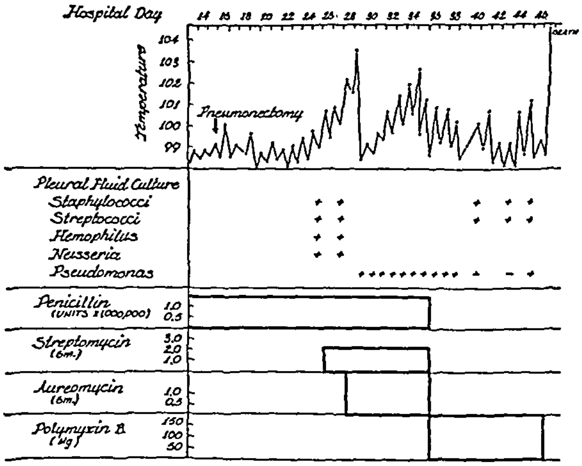

Fig. 5 (case 5).—Course of a 61-year-old man with postoperative empyema.

days of therapy. The patient made an uneventful recovery. During the polymyxin B therapy the patient had no complaints, and results of daily urinalyses were normal except for slight albuminuria and pyuria that had been present prior to therapy. At the beginning of polymyxin therapy the blood urea nitrogen level was 23 mg. per 100 cc., but during therapy it returned to normal. This patient was discharged on the 60th postoperative day without evidence of recurrence of infection.

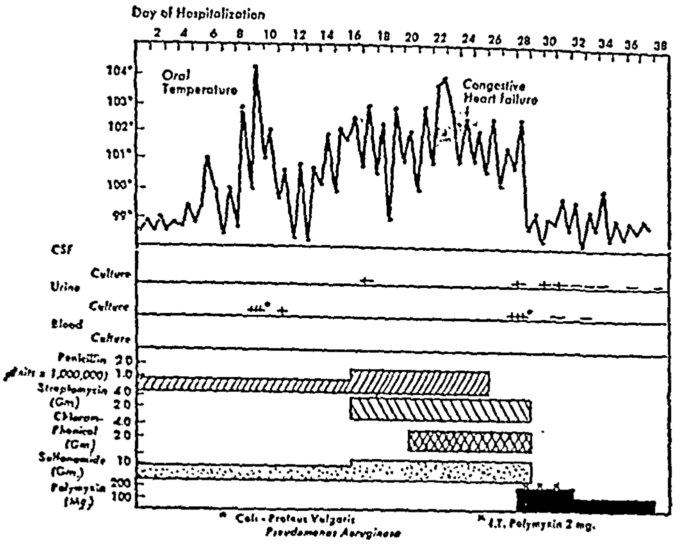

Fig. 6 (case 6).—Course of an 18-year-old youth with postoperative meningitis due to P. vulgaris.

Case 7.—A 54-year-old Mexican was admitted to the hospital because of acute urinary retention. He had been in the hospital previously for an abdominoperineal resection for adenocarcinoma of the colon. At this time he was given prophylactic phthalylsulfathiazole (sulfathalidine®), penicillin, and streptomycin before and following the operation. On admission he had a low grade fever and was given penicillin and streptomycin (fig. 7). In spite of this treatment the temperature rapidly rose to 105 F by the sixth hospital day. Urine cultures revealed a mixed infection, including Aerobacter aerogenes, Esch. coli, Streptococcus hemolyticus, P. vulgaris and Ps. aeruginosa. Blood cultures were sterile. Penicillin and streptomycin were continued and on the eighth hospital day, terramycin was added to the regime. There was rapid clinical improvement, and a transurethral resection was performed on the 22nd hospital day. The following day there was a sharp rise in temperature and P. vulgaris was isolated from the blood stream. In addition to continuing administration of penicillin and streptomycin, administration of terramycin was again begun on the 23rd hospital day. There was no clinical improvement, and blood cultures were consistently positive for Proteus vulgaris. The penicillin dosage was increased and sulfithoxazole was added. On the 28th day the patient developed cutaneous petechiae and had two subsequent pulmonary infarctions. In vitro sensitivity tests indicated that this strain of Proteus was resistant to penicillin, streptomycin, and terramycin, but relatively sensitive to chloramphenicol (inhibited by 31.2 µg per milliliter). He was consequently given chloramphenicol but had congestive heart failure and died two days later. Autopsy revealed bilateral pyelonephritis, generalized carcinomatosis, and pulmonary congestion and infarctions.

Case 8.—A 61-year-old publisher was admitted to the hospital with chills and fever, urinary frequency, and burning. He had had previous episodes of acute urinary infections that had responded to either sulfonamides, penicillin, streptomycin, or aureomycin. In addition, he had arteriosclerotic heart disease with a recent myocardial infarction and congestive heart failure. Urinalysis on admission revealed albumin (4+), many white blood cells, and occasional red blood cells. The white blood cell count was 26,000 per cubic millimeter. The blood urea nitrogen level was 12 mg. per 100 cc. Ps. aeruginosa was cultured from the urine and blood. He was treated with sulfithoxazole and aureomycin without improvement (fig. 8). On the fourth hospital day intramuscular injections of polymyxin B were begun (50 mg. every six hours). Ps. aeruginosa was not cultured from the urine after the fifth hospital day, and blood cultures became negative. On the fourth day of therapy the temperature had returned to normal, and the dose was reduced to 25 mg. every six hours. The following day a transurethral prostatic resection was performed. Administration of polymyxin B was continued for a total of 11 days, during which time the pyuria disappeared, the albuminuria varied from 1+ to 4+, and the urinary output and blood urea nitrogen remained within normal limits. No evidence of recurrent infection occurred during this hospitalization. The patient was discharged on the 18th hospital day.

COMMENT

The patients discussed illustrate what is believed to be an increase in relative importance of Proteus and Pseudomonas in the production of infectious processes that may arise during antibiotic treatment of other infectious diseases. Clinical experience suggests that there might also be an actual increase in frequency of infections due to these organisms since the introduction of the agents with the broadest antimicrobial spectra. In an analysis (fig. 9) of the routine cultures received in the bacteriology laboratory of a large general hospital, the incidence of Proteus in the specimens received rose from 8% of the gram-negative bacilli recovered in June, 1948, to 32% in January, 1951. During the same time the incidence of Pseudomonas recovered increased from 15% to 30%. These data also reflected the clinical experience reported by others of the increase in the frequency of infections due to Candida (Monilia).[3] During the same period of two and one-half years there was a relative decrease in the isolation of certain other gram-negative bacilli, particularly Esch. coli. One cannot conclude from these figures that there has been an actual increase in clinical infections due to Proteus and Pseudomonas, since they are based on laboratory reports and not individual patients. The results of this study indicate, however, that these organisms are oftener present in body fluids or exudates and may serve as potential sources of infections.

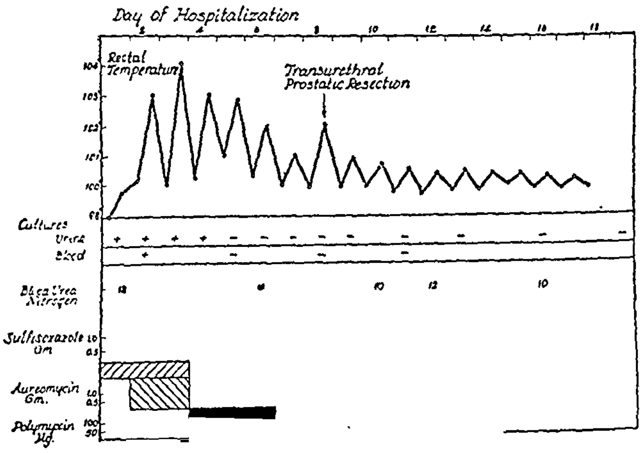

Fig. 8 (case 8).—Course of a 61-year-old man with an acute urinary infection previously treated with antibiotics. Ps. aeruginosa was cultured from the urine.

The isolation of Proteus or Pseudomonas from a patient does not necessarily mean that either of these organisms is playing a significant role in the production of an infectious process, since they are both normal inhabitants of the skin and the mucous membranes of the respiratory and gastrointestinal tracts. If the organisms are isolated in pure culture repeatedly from body fluids that are normally sterile, such as the blood stream, spinal fluid, or urine, in the presence of clinical evidence of infection, there can be little doubt as to their significance. When they are obtained in mixed infections or from skin secretions or mucous membrane surfaces, it may be difficult to evaluate their importance. However, if there is clinical and laboratory evidence of progressive infection and either of these two organisms is isolated in pure culture or as a predominant organism the importance of their role in pathogenesis must be strongly considered and their control undertaken.

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[3] Woods, J. W.; Manning, I. H., Jr., and Patterson, C. N.: Monilial Infections Complicating the Therapeutic Use of Antibiotics, J. A. M. A. 145:207 (Jan. 27) 1951.

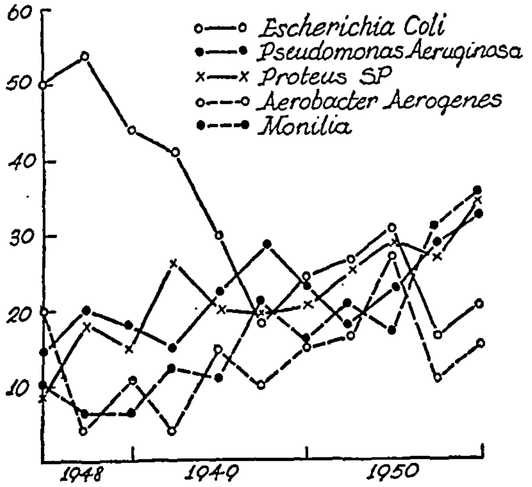

Fig. 9.—Analysis of results of routine cultures in a large general hospital illustrating the relative frequency of some of the gram-negative bacilli.

The relationship of the infections to the antimicrobial therapy was thought in most patients to be simply a matter of the elimination of sensitive species of bacteria, permitting the multiplication of resistant species. Since Proteus and Pseudomonas are usually the only bacteria surviving the action of the most recently introduced antibiotics, they are frequently the only remaining etiological possibilities. There was, moreover, a disturbance of some fundamental defense mechanism against infection in many of the patients. In some of the patients the infections resulted from anatomical defects prevented the adequate drainage of contaminated material. Urinary infections associated with prostatic or ureteral obstructions were frequently observed. Bronchiectasis and undrained abscesses are other examples. In other patients infected material was introduced into a closed body space, such as by puncture wounds of the bowel or chest either from trauma or operation, or by the contamination of the cerebrospinal fluid.

The onset of the signs and symptoms of the Proteus and Pseudomonas infections frequently occurred after a period of improvement following suppression of the initial etiological agent. This is illustrated by the patient (case 1) with cirrhosis of the liver in whom a Friedländer's pneumonia developed. This improved with the institution of massive antibiotic therapy, but later the pneumonia spread in spite of continued therapy. In case 4 pneumonia developed due to Pseudomonas while the patient was still receiving penicillin that had controlled pneumococcal bacteremia.

The course of the Proteus or Pseudomonas infections was quite variable. The correction of the underlying anatomical defect and surgical drainage was frequently adequate to control the infection. Other patients were able to develop sufficient immunity to overcome the infection when the antibiotic therapy was discontinued and the normal bacterial flora reestablished. The administration of specific antibacterial agents sometimes played a significant role in shortening the course of the infection. In four patients no therapy was effective and the infection contributed to a fatal outcome.

An important factor in controlling the infections studied was establishment of adequate surgical drainage and removal of nonvital tissue. In urinary tract infections, particularly, antibiotic therapy usually produced only transient benefit if adequate urinary drainage was not achieved. The extent of the surgery necessary varied considerably. Needle aspiration was sufficient drainage in a patient with septic arthritis. In postoperative empyema extensive surgical drainage in conjunction with streptokinase and streptodornase administration was necessary in some cases.

When a particular strain of Proteus or Pseudomonas was found to be sensitive in vitro to one of the antibiotics, the administration of this agent was found to play a valuable part of the management of the infection. Because of the wide variation in sensitivity of Proteus and Pseudomonas to antibiotics, in vitro sensitivity tests were routinely performed as an aid in selecting the agent of choice. The results of such in vitro tests using the tube dilution method against 50 strains of Pseudomonas are illustrated graphically in figure 10. These findings illustrate generally the resistance of Pseudomonas to most of the antibiotics as contrasted to the high degree of sensitivity to polymyxin B. The chart does not, however, present an entirely accurate picture, since some of the strains tested were isolated from patients who had already received one or more of the antibiotics, thus eliminating some of the more sensitive strains from the survey. Furthermore, the arbitrary test level of 15.6 μg per milliliter indicating sensitivity in this table may not entirely parallel the results in patients, since this drug level may not be attained in the infected tissues and body fluids. The study indicates, however, that the largest number of strains of Pseudomonas were sensitive in vitro to polymyxin B. Many were also sensitive to terramycin and neomycin.

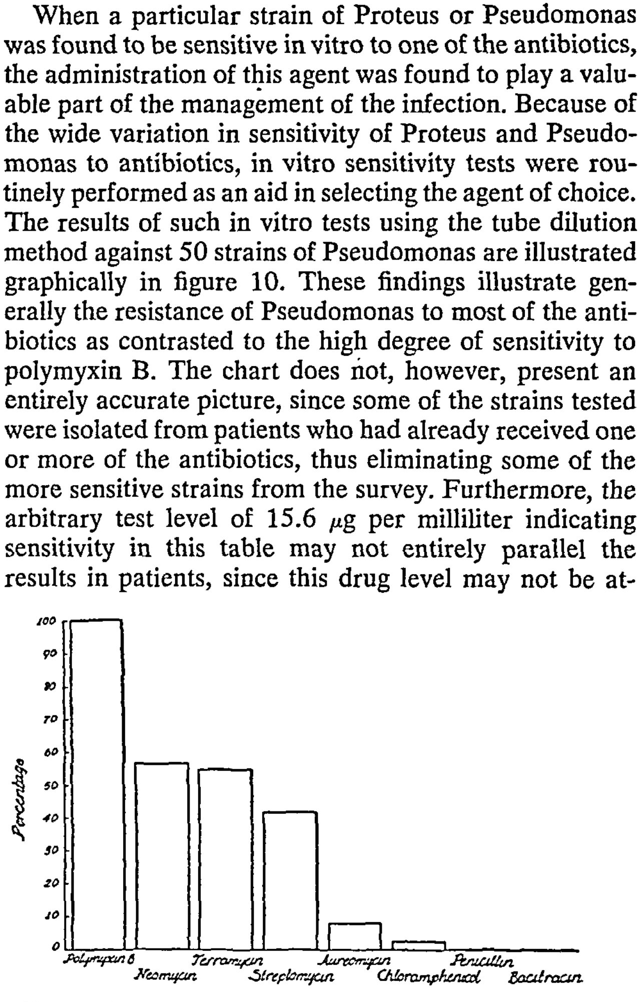

Fig. 10.—The in vitro sensitivity of 50 strains of Pseudomonas to various antibiotics at a concentration of 15.6 μg per milliliter.

A similar study was performed using 20 strains of Proteus. Many of these organisms were partially inhibited by the antibiotics against which they were tested, but only an occasional strain was completely inhibited by concentrations of the drugs obtained in the body fluids by the usual dosage schedules.

A summary of the results of the antibiotic therapy in the 56 cases reported in this series reveals that polymyxin B was the most effective agent in the treatment of Pseudomonas infections. In the nine patients in whom it was used, the infections were satisfactorily controlled in seven, or 78%. In contrast to reports following the use of less pure preparations, there was no evidence of significant toxicity noted from the use of this antibiotic. Terramycin and streptomycin exerted a favorable influence on the course of the infection in an occasional patient. Neomycin was not evaluated clinically because of other reports of toxicity following parenteral administration. Antibiotics played a less significant role in the treatment of the Proteus infections. Two patients with polymyxin-sensitive strains responded to the use of this drug. Streptomycin, given with large doses of one of the sulfonamides, is probably still the best treatment for infections due to this organism.

Number of Patients in Whom Pseudomonas or Proteus Infection Developed During or Following Antibiotic Therapy

Infection	No. of Cases	
	Pseudomonas	Proteus
Bacteremia	4	2
Pulmonary infections	5	0
Empyema	4	0
Meningitis	3	1
Peritonitis	0	1
Arthritis	1	0
Urinary infections	19	15
Prostatitis	1	0

SUMMARY

The suppression of the other bacteria by the widespread use of antimicrobial agents has increased the relative importance of Proteus and Pseudomonas as causative agents in infectious processes. Cases of infections due to these organisms that developed during or following repeated courses of antibiotic therapy are reported. The relationship of the infection to the antibiotic therapy was believed in most patients to be a matter of change in bacterial flora owing to elimination of sensitive bacteria and multiplication of resistant species. The importance of being aware of the possibility of Proteus or Pseudomonas infections in patients who originally respond to antibiotic therapy but who present evidence of renewed infection while still receiving therapy is illustrated by the patients described. The possible seriousness of giving the broad spectrum antibiotics prophylactically must be considered; since if an infection does occur with a nonsensitive organism during such therapy, it is almost certain to constitute a difficult therapeutic problem.

The problem of treatment of Proteus and Pseudomonas infections is considered. Streptomycin in combination with large doses of sulfonamides was the most effective therapy in Proteus infections. Polymyxin B was most consistently effective in Pseudomonas infections, though some strains were sensitive in vitro to terramycin. The correction of any underlying anatomical disorder was considered a most important part of management. The discontinuance of ineffective antibiotics was sufficient in some patients to control the infection by allowing the reestablishment of normal bacterial flora.

1200 M. D. Anderson Blvd.

THERAPEUTIC VALUE OF PROBENECID (BENEMID®) IN GOUT

Luke R. Pascale, M.D., Alvin Dubin, M.S.
and
William S. Hoffman, M.D., Chicago

Probenecid (benemid®) was first introduced into clinical medicine by Boger[1] as an adjunct to penicillin and paraaminosalicylic acid therapy. Probenecid has been found to be a powerful agent that reversibly inhibits renal tubular transfer, suppressing tubular secretion of penicillin, paraaminosalicylic acid, paraaminohippuric acid, and phenolsulfonphthalein.[2] In this respect it is similar to carinamide; but while the latter is required in doses of 18 to 24 gm. per day for its tubular effect, probenecid accomplishes the same results in doses of 2 gm. daily. That probenecid, like carinamide, is also capable of blocking tubular reabsorption of glomerular constituents was demonstrated by Schneider and Corcoran,[3] who were able to increase the excretion of phosphate by administration of the drug. Gutman, acting on the report by Wolfson and associates[4] that carinamide was a uricosuric agent, demonstrated that probenecid had a similar effect, presumably by blocking the partial reabsorption of filtered urate. A preliminary report by Gutman on the efficacy of probenecid in the treatment of gout has recently appeared.[5] Bishop and associates[6] also reported a uricosuric activity of probenecid but with no clinical studies.

The similarity of action of probenecid to that of carinamide led us independently to study the potential value of this new drug in the treatment of gout. Our studies were begun without benefit of the experiences of Gutman and Bishop. In these studies, after a preliminary demonstration of the uricosuric action of probenecid, an effort was made to determine an adequate dose schedule in the treatment of gout, the extent of improvement in patients with gouty arthritis, and the possible toxicity from prolonged administration.[7]

Mrs. Anne Whittier made the chemical analyses for this study.

Presented in part before the Central Society for Clinical Research, Chicago, Nov. 2, 1951.

From the Hektoen Institute for Medical Research of the Cook County Hospital, the Department of Medicine, Stritch School of Medicine, Loyola University (Dr. Pascale), and the Department of Physiology, University of Illinois College of Medicine (Dr. Hoffman).

[1] Boger, W. P.; Beatty, J. O.; Pitts, F. W., and Flippin, H. F.: The Influence of a New Benzoic Acid Derivative on the Metabolism of Para-Aminosalicylic Acid (PAS) and Penicillin, Ann. Int. Med. 33:18-31 (July) 1950. Boger, W. P.; Pitts, F. W., and Gallagher, M. E.: Benemid and Carinamide: Comparison of Effect on Para-Aminosalicylic Acid (PAS) Plasma Concentration, J. Lab. & Clin. Med. 36:276-282 (Aug.) 1950.

METHODS AND MATERIALS

The 20 male subjects for this study were ambulant or bed patients of the Cook County Hospital. The diagnosis of gout was established in all these patients by the history or observation of typical acute gouty attacks and by the finding of elevated serum uric acid concentration. In seven patients there was the additional evidence of tophi and/or diagnostic x-ray changes. Limitation of joint motion was seen in these seven as well as in four other subjects. In four subjects the limitation was severe, in five it was moderate, and in the remaining two it was mild. Ten of the patients were under observation for periods of five to seven and a half months. Six more were followed for two to five months. Four patients were dropped from the study after the preliminary survey.

The demonstration of the uricosuric effect of probenecid therapy was carried out in the first five patients studied. The subjects were placed on a standard low purine diet. After a control period, probenecid was prescribed in doses of 0.5 gm. (one tablet) every six hours. The 24-hour urine collection and the serum were analyzed for uric acid by a photoelectric modification [8] of the Folin method. (Since the values were comparative, no effort was made to eliminate the nonspecific chromogen by the use of uricase. Wolfson and associates [9] have shown that true urate of serum and urine in gouty patients consistently represents 85% to 90% of the apparent urate. The range of serum concentrations in normal persons by this method was 3.0 to 5.9 mg. per 100 cc.) With increased excretion of urate and a fall in the serum urate level, probenecid therapy was discontinued to allow a return to pretreatment conditions. Then the experiment was repeated.

Several regimens were tried out in most of the patients. These included administration of 2 gm. of probenecid daily, 2 gm. every other day, and 2 gm. for only two successive days out of each week. In three patients the effect of acetylsalicylic acid simultaneously administered with probenecid for four days was compared with that of probenecid therapy alone. During the later part of the study five patients were not given probenecid and treated with 5.2 gm. of acetylsalicylic acid and 8 gm. of sodium bicarbonate daily for three weeks. The administration of 2 gm. of probenecid daily was then resumed.

A standard low purine diet was used only for the patients hospitalized for the urine studies, chiefly to assure a constant purine intake. During the period of the various maintenance regimens, the patients were allowed a more or less general diet.

The possible toxicity of probenecid was studied by weekly physical examinations and frequent blood chemical analyses. The latter included the determination of nonprotein nitrogen, albumin, globulin, calcium, phosphorus, alkaline phosphatase, chloride, sodium, potassium, carbon dioxide capacity, cholesterol, cephalin cholesterol flocculation, and thymol turbidity. Also, at frequent intervals complete blood cell counts were made and routine urine examinations performed.

Uric Acid Clearances Before and During Probenecid Therapy in Gouty Patients

	Before Probenecid Therapy				During Probenecid Therapy			
Patient	Serum Uric Acid, Mg./ 100 Cc.	Urine Uric Acid, Mg./ 100 Cc.*	Volume of Urine, Cc./ Min.*	Uric Acid Clearance, Cc./ Min.	Serum Uric Acid, Mg./ 100 Cc.†	Urine Uric Acid, Mg./ 100 Cc.*	Volume of Urine, Cc./ Min.	Uric Acid Clearance, Cc./ Min.†
G. R.	8.5	18.9	1.4	3.1	4.8	23.5	1.5	7.3
H. S.	9.0	28.8	1.8	5.8	4.8	24.5	1.7	8.7
O. B.	11.2	50.1	0.9	4.0	7.8	65.3	1.2	10.5
F. C.	14.1	34.2	1.2	2.9	6.7	60.0	1.4	12.5
H. Sp.	9.3	23.0	2.1	5.2	7.4	27.9	2.3	8.7

* Based on 24 hour output.

† Uric acid clearances were determined on the third or fourth day, after stabilization of serum uric acid levels had occurred.

RESULTS

Effect of Probenecid on Urinary Urate Excretion and on Serum Urate Level.—An increase in urinary excretion of urate occurred promptly in 17 of the 20 patients studied. In the five subjects of whom protracted quantitative studies were made, the increase was equivalent to 35% to 200% of the control excretion. In some instances the 24-hour urine contained 1.5 to 2.4 gm. of urate. Much of it was already precipitated in the collection bottle. The initial high excretion was not maintained. Gradually it fell, corresponding to the lowering of the serum uric acid concentration (fig. 1). The pattern of excretion was similar to that reported by Gutman [5] and by Bishop and associates.[6] The roughly estimated 24-hour urate clearances were increased two to fourfold by the administration of probenecid (see accompanying table). This increased clearance was still effective during the diminished excretion, the latter merely reflecting the fall in serum uric acid concentration. Though continuous quantitative collections of the urine were carried out only in the first phase of the study, occasional 24-hour analyses gave us the impression that the daily excretion of urate during administration of probenecid was always higher than that of the control period, at least in the patients with tophaceous gout, just as was found by Gutman.

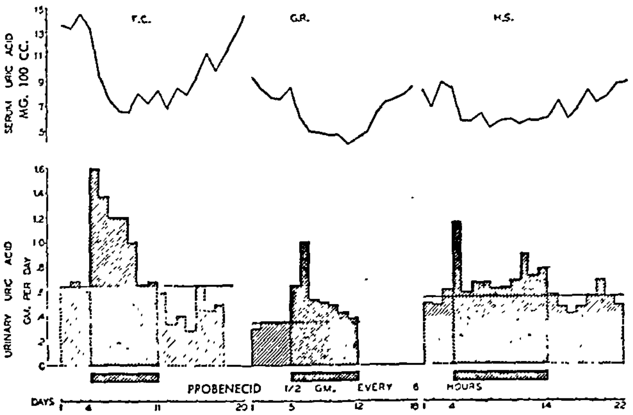

Fig. 1.—Effect of probenecid on urinary uric acid excretion and serum uric acid levels.

A prompt precipitous fall in the serum urate concentration occurred in 14 subjects, as illustrated in the cases shown in figure 1. Three patients showed a more gradual fall in serum levels, the nadir not being reached until 96 or 120 hours. In three other subjects no decrease in the serum uric acid level occurred, even though 2 gm. of probenecid was administered daily for one to three weeks. The failure was apparently associated with the renal

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2. Beyer, K. H.: Functional Characteristics of Renal Transport Mechanisms, J. Pharmacol. & Exper. Therap. 99: 227-280 (Aug.) 1950.
3. Schneider, R. W., and Corcoran, A. C.: Familial Nephrogenic Osteopathy Due to Excessive Tubular Reabsorption of Inorganic Phosphate: A New Syndrome and a Novel Mode of Relief, Proceedings of the Central Society for Clinical Research, vol. 23, 1950.
4. Wolfson, W. Q.; Cohn, C.; Levine, R., and Huddlestun, B.: Transport and Excretion of Uric Acid in Man, Physiologic Significance of the Uricosuric Effect of Carinamide, abstracted in Am. J. Med. 4: 744 (May) 1948.
5. Gutman, A. B.: Some Recent Advances in the Study of Uric Acid Metabolism and Gout, Bull. New York Acad. Med. 27: 144-164 (March) 1951.
6. Bishop, C.; Rand, R., and Talbott, J. H.: The Effect of Benemid (p-[di-n-propylsulfamyl]-benzoic acid) on Uric Acid Metabolism in One Normal and One Gouty Subject, J. Clin. Investigation 30: 879 (Aug.) 1951.
7. Sharpe & Dohme, Inc., supplied the probenecid used in this study.
8. Hoffman, W. S.: Photelometric Clinical Chemistry, New York, William Morrow & Co., 1941.
9. Wolfson, W. Q.; Huddlestun, B., and Levine, R.: The Transport and Excretion of Uric Acid in Man: The Endogenous Uric Acid-Like Chromogen of Biological Fluids, J. Clin. Investigation 26: 995-1001 (Sept.) 1947.

insufficiency discovered in these subjects. Two had urea clearances of 26% and 27% of normal, respectively, and the third had chronic azotemia with nonprotein nitrogen levels ranging from 68 to 85 mg. per 100 cc. These three unresponsive patients were not studied beyond the three-week period.

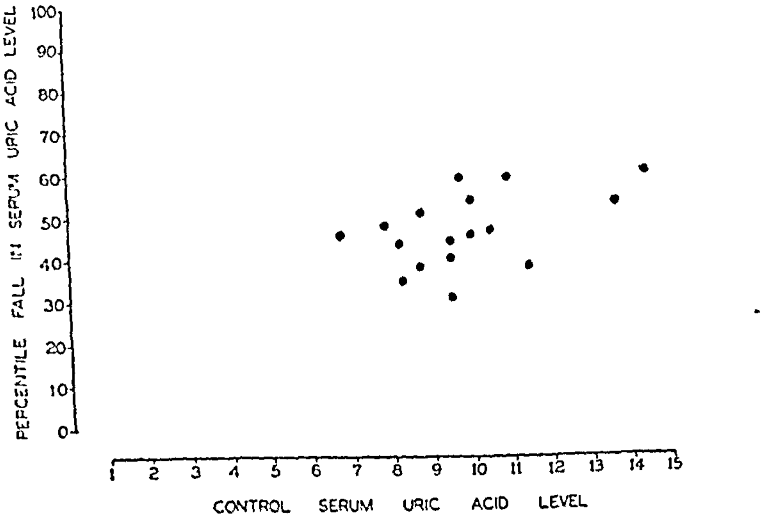

Fig. 2.—Relation of percentile fall in serum uric acid levels after 72 hours of probenecid therapy to the control (pretreatment) serum uric acid levels, in milligrams per 100 cc.

The percentile fall in the serum urate level at the end of the 72 hours in the 17 subjects who responded to probenecid therapy ranged from 30% to 58%, the average fall being 45%. In some instances a lower serum level was encountered several days later, but for the most part the serum concentration rose slightly with continued probenecid therapy and was maintained at a level slightly higher than that of the nadir. There was no significant correlation between the percentile fall and the height of the control serum urate concentration (fig. 2).

In the five patients who were given 2 gm. of probenecid daily for only the first two days of each week, the initially lowered serum uric acid level was not maintained; instead, there was a rapid rise during the next five days to the pretreatment levels. With each new two-day period of probenecid administration, the serum uric acid level fell as promptly as before. Though in several instances the extent of the fall with each new treatment period appeared to be slightly less than previously, there were no instances of developed refractoriness to probenecid therapy. Figure 3 is illustrative of the curves obtained.

During the course of the study 16 subjects were maintained on a regimen of 2 gm. of probenecid given every other day for periods ranging between 4 and 15 weeks. The response to this dosage varied unpredictably. Four patients, within the first two weeks, had a slow but steady rise in serum uric acid concentration above the initially obtained minimal level. In four more, a rise eventually occurred but was delayed for 6 to 10 weeks. In the remaining eight patients, though an initial rise might temporarily have occurred, the depressed serum uric acid level was on the whole well maintained during the 4 to 15 weeks of observation. However, all the patients who were again given 2 gm. of probenecid daily showed a further depression of the serum uric acid concentrations. There was no doubt that, insofar as serum levels were concerned, the most satisfactory regimen was a dosage of 2 gm. of probenecid daily given continuously. For greater convenience the medication schedule was 1 gm. given twice a day instead of 0.5 gm. given every six hours, without any appreciable change in the serum levels. With this continuous therapy the uric acid concentration could be maintained indefinitely at levels ranging from 4 to 8 mg. per 100 cc., with occassional temporary rises, especially at times of an acute attack.

One of the 20 patients in this study died suddenly of coronary occlusion after preliminary daily treatment with probenecid for four weeks. He was 76 years old. Autopsy revealed no anatomic defects that could be related to the administration of probenecid.

Effect of Probenecid on the Symptoms of Gout.—In figure 4 are summarized the pertinent clinical data of the 16 patients who were treated with probenecid for an extended period. The eight patients who displayed limitation of joint motion all showed progressive improvement during probenecid therapy. G. R., a barber with gout for five years, had not been able to work more than three days a week, if at all, during the past three years because of stiffness and pain in the joints of the extremities. After six weeks of probenecid therapy he became completely relieved of pain and joint dysfunction and is now working at his trade six full days per week, with continued medication. O. B. had been completely bedridden for a period of one year because of gouty arthritis. This lesion was superimposed on a residual paralysis of the right lower extremity and weakness of the left foot resulting from poliomyelitis. After five months of probenecid therapy, he was able to walk with the aid of his brace and crutches as well as he did before the onset of gout. J. B. had been bedridden two months with continued pain and severe limitation of joint movement. At the end of three months of probenecid therapy he was able to walk unaided and to drive his automobile. Five other patients showed comparable, if less dramatic, improvement. Of this group, H. P. and P. K., both of whom had large tophaceous deposits in the hands, showed the least benefit from treatment, but even in these two there was enough improvement, both objective and subjective, to make them enthusiastic over the treatment and desirous of its continuation.

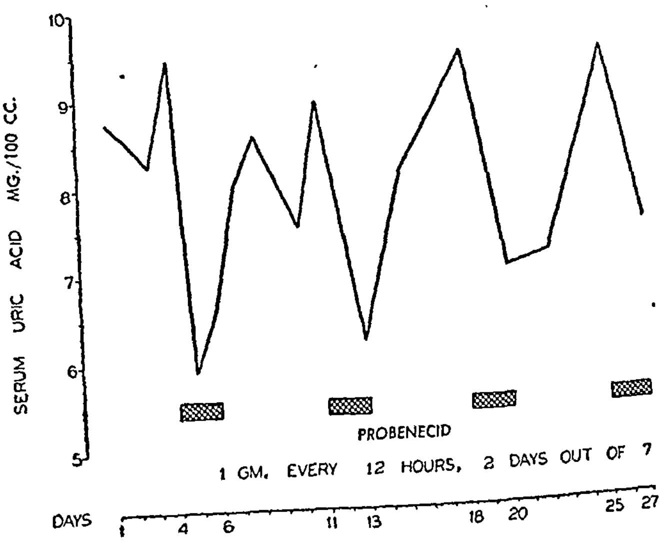

Fig. 3.—Serum uric acid levels in patient receiving intermittent probenecid therapy.

In the eight patients with acute recurrent gout without symptoms in the intercritical periods, the results of therapy were, of course, more difficult to evaluate. The serum uric acid was maintained in these persons at levels only slightly higher than the upper limits of normal without any restrictions of diet. This was an important benefit in itself. The patients felt well and had no complaints, but patients with recurrent gouty attacks usually acted that way even without medication. Three of these patients had acute attacks during the period of medication. J. D. had a moderately severe attack after six days of probenecid therapy, which lasted four days; then he had a mild attack four weeks later, which lasted two days. P. M. had one attack during the three and a half months of therapy, and F. M. had one mild attack after three months of therapy. The remaining five patients were free of acute manifestations of gout during the therapeutic study; but, since they normally had only two or three attacks a year, a much more prolonged study will be required to ascertain the effect of probenecid therapy on the incidence of acute joint manifestations.

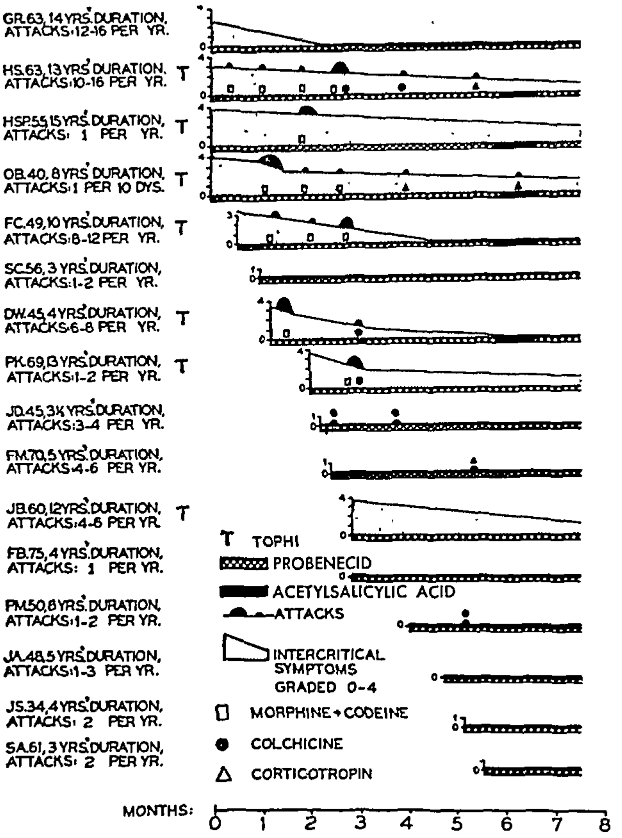

Fig. 4.—Clinical course of 16 patients with gout during probenecid therapy.

The fact that one or more attacks occurred in nine patients during probenecid therapy is an important physiological as well as therapeutic problem. Gutman [5] has reported the same phenomenon, and he has attributed the attacks to the action of probenecid. This may be the case, but it is far from certain. The attacks occurred principally in those patients who had had them frequently before probenecid therapy and who had chronic gouty arthritis with multiple urate deposits. In the first attacks we were courageous enough to continue probenecid therapy and to avoid the use of colchicine or corticotropin (ACTH). Only codeine or morphine were utilized for the pain. As a consequence, the attacks were rather prolonged, some lasting 12 days. It was obvious that probenecid had no colchicine type of action. It neither relieved the acute pain nor shortened the length of the attack. With the subsidence of the inflammation in the acutely involved joint, there was usually less limitation of motion in that joint than before the attack. D. W., for example, before the first attack in his left knee during probenecid therapy, had had only 80 degree flexion of the knee joint. After the attack subsided, he was able to bend the knee 135 degrees, or practically normally. Succeeding attacks in the same patient usually migrated to other joints.

The frequency and intensity of acute attacks in the patients with chronic gouty arthritis appeared to diminish with continued probenecid therapy, as is seen in figure 4. When it was finally recognized that probenecid therapy was of no value for the acute attacks, subsequent episodes were managed with colchicine or corticotropin [10] therapy. The former was given in doses of 0.65 mg. (1/100 grain) every four hours until diarrhea set in. Corticotropin was used in four attacks in doses of 10 mg. every six hours; two to four injections were required. Whether colchicine or corticotropin was used, the attacks usually subsided completely in 24 to 72 hours.

Effect of Salicylate on the Uricosuric Action of Probenecid.—It is well known that sodium salicylate or acetylsalicylic acid given in large doses has a uricosuric effect and that with prolonged administration a lowering of the serum urate level is produced in gouty subjects. It was interesting, therefore, to determine the influence of acetylsalicylic acid administered simultaneously with probenecid. To our surprise, acetylsalicylic acid given in doses of 1.3 gm. every six hours completely suppressed the effect of probenecid. The blood urate remained at least as high as without any medication. When acetylsalicylic acid therapy was stopped after four days, while probenecid therapy was continued, the fall of the serum uric acid was just as prompt and as precipitous as in

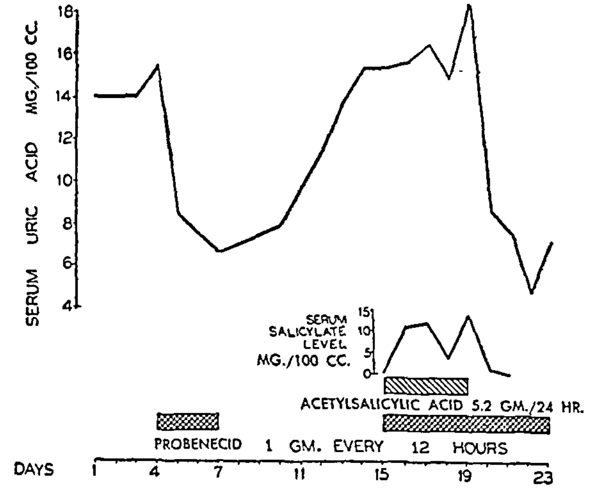

Fig. 5.—The influence of acetylsalicylic acid therapy on the ability of probenecid therapy to lower the serum uric acid concentration.

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[10] The corticotropin used was furnished by the Wilson Laboratories, Chicago.

previous probenecid therapy. Identical results were seen in the three patients in whom this effect was studied. One experiment is shown in figure 5.

The fixation of the serum uric acid level when acetylsalicylic acid was given along with probenecid was almost certainly due to a blocking of the uricosuric action of probenecid. It could not be due to falsely high serum uric acid levels, because of the presence of salicylates or its metabolites in the blood. We have made innumerable determinations of uric acid in gouty and nongouty patients during salicylate therapy and have never found any false elevation of serum uric acid. However, to prove conclusively that salicylates exert their influence by preventing the uricosuric effect of probenecid and to determine at what threshold serum salicylate level this inhibition occurs, it is necessary to carry out urine urate analyses. One must take special precautions in analyzing urine for urate during salicylate therapy, for it has been shown [11] that gentisic acid, a metabolite of salicylate, may on occasion raise the apparent urine urate concentration by as much as 25% even when the uricase method of analysis is utilized. Such studies are now in progress.

Some of the five patients were maintained for a period of three weeks on a regimen of 1.3 gm. of acetylsalicylic acid and 2 gm. of sodium bicarbonate given four times a day for five out of each seven days. The serum uric acid concentration rose in all these patients from the low level of the previous probenecid regimen to the high control levels. Slowly the concentration diminished in two of these patients, but it never fell to the level found in the probenecid therapy period.

Toxic Effects of Probenecid Therapy.—On the whole the patients remained remarkably free of side-effects of probenecid therapy. Only three patients had changes that could be attributed to toxicity due to probeneicd. H. S. had a rash, which disappeared without recurrence in spite of continued probenecid therapy. During his only gouty attack while receiving treatment, proxysmal auricular fibrillation developed. This arrhythmia was converted to normal by digitalization. The rhythm has since then remained of sinus origin. J. A. had epigastric distress, which subsided after two days despite continued therapy.

The one potentially serious complication occurred in O. B. This was the development of urate crystalluria and hematuria early in the course of treatment with probenecid. Treatment with the drug was temporarily stopped, and the complications disappeared within two days. After eight days, probenecid therapy was resumed and was supplemented with administration of sodium bicarbonate and an increased water intake. There was no recurrence of the urinary findings.

Blood chemical analyses showed no significant deviations from normal that might indicate renal or hepatic injury or disturbances of electrolyte metabolism. Serum phosphate levels were at low normal. Since the data of the chemical analyses were not enlightening, they have been omitted. Complete blood cell counts made serially gave no evidence of inhibition of normal erythropoiesis and granulopoiesis or of any other abnormality. There were no alterations found in the urine on repeated routine examinations, except for heavy urate precipitations when the specimens were allowed to stand.

COMMENT

Gout is still a mysterious disease, the etiology and pathogenesis of which have not been unraveled; yet modern investigations point to three fundamental features of the gouty patient that must be understood if the potentialities of probenecid therapy are to be appreciated:

1. Gout occurs in patients with familial hyperuricemia.[12] The cause of the elevated serum uric acid level has not been determined, but it may be (*a*) overproduction of uric acid, (*b*) diminished destruction of uric acid, or (*c*) diminished renal excretion of uric acid. In the last analysis, it is absolute or relative renal inefficiency that is responsible for the hyperuricemia, for the normal urate clearance is only 5% to 10% of the glomerular filtration rate, a large portion of the filtered urate being reabsorbed by the tubules by active enzymatic processes.[13] The system of the gouty patient, instead of adjusting excretion by diminishing tubular reabsorption, attempts to do so by raising the level of serum uric acid.

2. Hyperuricemia is attended by actual or potential supersaturation of urate in the interstitial fluids, at least in special sites, such as around joints, in bursae, and in cartilage. Thus hyperuricemia ultimately defeats its own purpose. With precipitation of urates in the tissues, the serum level should fall. However, a renewed high level is soon established to maintain excretion, which level allows further precipitation. Repeated waves of positive balance of uric acid are thereby produced by the very hyperuricemia designed to avoid this eventuality. From the work of Benedict and his associates [14] with isotopic uric acid, it appears that the freshly precipitated urate is in the form of fine crystals that are easily redissolvable and are part of the miscible pool of uric acid. The older deposits, however, particularly the material in the depth of tophi, are chalky and much less soluble. This portion is not in equilibrium with the soluble or dissolved urate.

3. The gouty patient has a unique exaggerated inflammatory reaction to precipitation of urates in the region of the joints, associated with severe pain, swelling, and limitation of motion. This reaction is promptly reversible by the administration of colchicine or corticotropin, without relation to any change in the excretion of urate.[5] The fact that gouty patients even during intercritical periods have low excretions of 17-ketosteroids,[15] the fact that the administration of small quantities of corticotropin is effective in reversing such attacks, and the fact that gouty attacks are induced by all the stresses that are known to call for an increased production of adrenocortical hormones and by administration of corticotropin followed by its withdrawal,[16] all point to a disturbance in adrenal function in gouty patients.

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[11] Yu, T. F., and Gutman, A. B.: Interference of Gentisic Acid in Determination of Urinary Uric Acid After Administration of Salicylates, Federation Proc. **8**:267, 1949.

[12] Smyth, C. J.; Cotterman, C. W., and Freyberg, K. H.: The Genetics of Gout and Hyperuricemia: An Analysis of 19 Families, J. Clin. Investigation **27**:749-759 (Nov.) 1948.

[13] Berliner, R. W.; Hilton, J. G.; Yu, T. F., and Kennedy, T. J.: The Renal Mechanism for Urate Excretion in Man, J. Clin. Investigation **29**:396-401 (April) 1950.

[14] Benedict, J. C., and others: The Effect of Salicylates and Adrenocorticotropic Hormone upon the Miscible Pool of Uric Acid in Gout, J. Clin. Investigation **29**:1104-1111 (Aug.) 1950.

[15] Wolfson, W. Q., and others: An Endocrine Finding Apparently Characteristic of Gout: Very Low Urinary 17-Ketosteroids with Clinically Normal Androgenic Function, J. Clin. Endocrinol. **9**:497-513 (June) 1949.

[16] Hellman, L.: Production of Acute Gouty Arthritis by Adrenocorticotropin, Science **109**:280-281 (March 18) 1949.

Probenecid therapy attacks the problem of gout at the level of renal excretion. It does nothing for the specific gouty response to urate deposition; it merely removes the hyperuricemia. Probenecid is apparently the most effective and most harmless uricosuric agent so far discovered. It is far more effective than neocinchophen or acetylsalicylic acid, without the attendant dangers of these drugs. It produces a large negative balance of uric acid when first administered at a time of high serum uric acid levels, and a continued small negative balance when the serum level has been lowered to near the upper limit of normal. Gutman has estimated an extra excretion of about 75 gm. in five months. Though we have been unable to carry out prolonged urine collections to determine this excretion, extrapolations from our limited data would tend to confirm Gutman's estimation.

The therapeutic value of the negative uric acid balance produced by probenecid was unequivocal in the patients with chronic tophaceous gout. The disability was diminished in all cases and almost completely removed in several. Apparently the urate that is first removed is in the form of easily soluble crystals loosely deposited around the joints and on the surface of old tophi. Benedict and associates [14] have demonstrated that as much as 29 gm. of such miscible urate deposits can be removed in three months by the uricosuric effect of acetylsalicylic acid. The rate of removal with probenecid therapy is undoubtedly greater.

Benedict and his associates have called attention to the fact that the removal of urate deposits by acetylsalicylic acid in their case was accomplished without a significant lowering of the serum uric acid level. However, it has been our experience both in the several weeks of acetylsalicylic acid therapy in the present series of cases and in long-term studies in other cases that the serum uric acid level does slowly fall with continued acetylsalicylic acid therapy. Nevertheless, it is undoubtedly true that administration of acetylsalicylic acid does not produce the rapid fall in serum level accomplished with probenecid therapy. Apparently the uricosuric effect of probenecid therapy is so great that excretion is always ahead of resorption of deposited uric acid, whereas in the case of acetylsalicylic acid therapy the excretion is slow enough to allow solution to keep pace with it, as long as an appreciable store of easily miscible urate is available. Eventually, as the miscible pool shrinks, the excretion becomes slightly faster than solution, and the serum uric acid level slowly declines.

It was impossible in the short period of the present study to evaluate the effective probenecid therapy on recurrent nontophaceous gout. Nevertheless, it is fair to prophesy that, with continued therapy and with the maintenance of the serum levels near the range of normal, tophi will be prevented from forming and that eventually acute gouty episodes will seldom occur. Of course, such an eventuality will depend on the innocuousness of continued probenecid therapy over a long period.

It is possible, though far from certain, from our data and from those of Gutman that acute attacks are precipitated shortly after the beginning of probenecid therapy, at least in patients with chronic tophaceous gout. The mechanism of this development in the presence of a much lowered serum uric acid level is difficult to visualize. Perhaps solution of urates takes place in one region while reprecipitation takes place in another region, where local factors (which might be characteristic of gout) are conducive to disturbances of the supersaturated solution. At any rate it is our clinical impression that these attacks are not likely to occur in the same joint again with continued probenecid therapy and that the rate and intensity of recurrence ultimately diminishes. Certainly, with the recognized efficiency of colchicine and/or corticotropin therapy in alleviating the acute episodes, this circumstance should prove no contraindication to probenecid therapy.

The blocking effect of acetylsalicylic acid on the uricosuric action of probenecid was unexpected. If salicylates act, as Levine and associates maintain,[17] by freeing serum urate from its combination with serum protein, this annulling effect should not occur, whether probenecid owes its uricosuric effect to a similar liberation of urate from protein or whether it acts by interfering with tubular reabsorption of urate. Indeed, one should expect a synergistic effect. In consideration of the recognized action of probenecid in preventing tubular excretion of penicillin and paraaminosalicylic acid, it appears unreasonable to look elsewhere than in the tubules for the uricosuric effect of probenecid.

Salicylate probably exerts its interference with the action of probenecid directly in the tubules. It has been demonstrated [18] that, unless the urine is strongly alkaline, 50% to 80% of ingested salicylate or acetylsalicylic acid is excreted as salicyluric acid or salicyl glycuronide. Almost all of this conjugation takes place in the renal tubules. If we assume that probenecid produces its blocking action on urate reabsorption only after it is conjugated in the tubular cell, then it is possible to visualize the negating effect of salicylate as being due to its competition with probenecid in the tubule for the conjugating enzymes. This hypothesis is not out of accord with Beyer's theory of tubular transport mechanism.[2]

Obviously we have not been able to explore all possible dosages of probenecid. On the basis of previous experience with probenecid in penicillin and paraaminosalicylic acid therapy, we have kept the daily dosage at 2 gm. per day, but have varied the days of treatment, hoping to find a less rigorous management than daily administration satisfactory. Though we could see no clinical difference between the dosage of 2 gm. of probenecid given every day and that of 2 gm. given every other day, it was clear that in only a few patients—usually those without tophi—could the serum urate level be kept relatively low without continuous therapy.

The possibility of the development of hematuria from uric acid crystals or calculi must be seriously considered. We have recognized the phenomenon in only one patient, but we may have overlooked transient episodes in other patients. When the initial uric acid level is very high—12 to 15 mg. per 100 cc.—the daily excretion may be of the order of 2 gm. This quantity of urate, even in urine volumes of 2,000 cc., is far too great to be held in solution at the usual pH of urine.[20] Therefore, we have recommended a large fluid intake and the ingestion of about 8 gm. of sodium bicarbonate daily during probenecid therapy. When the initial serum uric acid level is very high, it is probably safer to begin treatment at 1 gm. of probenecid per day. However, we have had no experience with that dosage.

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[17] Levine, R.: Personal communication to the authors.

[18] Smith, P. K.; Gleason, H. L.; Stoll, C. G., and Ogorzalek, S.: Studies on the Pharmacology of Salicylates, J. Pharmacol. & Exper. Therap. 87:237-255 (July) 1946. Hoffman, W. S., and Nobe, C.: The Influence of Urinary pH on the Renal Excretion of Salicyl Derivatives During Aspirin Therapy, J. Lab. & Clin. Med. 35:237-248 (Feb.) 1950.

The failure of probenecid therapy to lower the serum urate level in three patients with diminished renal function must be further explored in both gouty and nongouty patients. It is important to determine whether renal insufficiency was actually the cause of failure of probenecid action in these cases and, if so, what the nature and degree of the renal defect must be to cause this failure. Such experiments are now being carried out.

SUMMARY

The therapeutic potentialities of probenecid in the treatment of recurrent gout and chronic gouty arthritis were studied in 20 patients. Ten were followed for periods of five to seven and a half months. With a dose of 2 gm. daily, serum uric acid fell within 72 hours to an average of 55% of the control level. This was associated with an initial increase in urate excretion of 35% to 200%. Urate clearances remained increased two to fourfold with continued probenecid administration.

In three patients with decreased renal function, probenecid therapy produced no fall in the serum uric acid level. When acetylsalicylic acid was given simultaneously with probenecid, the action of the latter in lowering the serum uric acid level was completely annulled but was restored when acetylsalicylic acid therapy was discontinued.

Probenecid therapy proved of greatest benefit in chronic gouty arthritis. Joint movement improved progressively in all patients; pain and swelling subsided. Acute attacks of gout developed in some patients soon after treatment was started, but their incidence diminished with continued therapy. No significant or consistent changes occurred in the serum electrolyte concentrations during prolonged therapy. No abnormalities in blood cell distribution were found. Results of hepatic and renal function tests remained normal. In one patient mild hematuria occurred from urate crystals during the first stage of treatment.

629 Wood St. (Dr. Hoffman).

19. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry Interpretation, ed. 2, Baltimore, Williams & Wilkins Company, 1946.

BLUEPRINT FOR IMPROVED PHYSICIAN-PATIENT RELATIONS

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The public is becoming impatient with the physician who is merely a good technician. Years ago, the patient had a beloved family physician to whom he could take all of his problems, medical and personal. Today, advances in medical and surgical methods assure the patient of improved physical treatment, but he often finds that he is no longer treated as an individual with a disease but as a "case" of a certain disease. Worse still, if the patient suffers from one of the many conditions that cause bothersome, disabling aches and pains that cannot be analyzed medically nor classified definitely into one category or another, the illness is too often considered to be psychosomatic.

Every patient who seeks medical help for the relief of painful symptoms is entitled to an unhurried and sympathetic hearing, whether his problems be primarily physiological, primarily psychological, or a combination of the two. Patients with organic problems are often in need of a strong dependable relationship, a framework within which reassurance can be offered to offset or alleviate the anxiety aroused by the patient's fears. On the other hand, patients whose symptoms are mildly or markedly excessive in view of the physical findings often experience a great deal of relief in sharing emotional problems with their physicians.

Beginnings of the relationship between the physician and the patient are laid even before the initial contact with the physician. The impress of the physician's personality can be felt the moment the patient steps into the reception room. Favorable first impressions are most important.

OFFICE MIRRORS THE PHYSICIAN'S ATTITUDE

The patient should be regarded as a guest, not as an interruption of the receptionist's work. The receptionist, like all persons employed by the physician, will take her cue from her employer: if he apparently welcomes the patient's visit, the receptionist will do so; if the physician is annoyed at the interruption, that attitude is passed on by the girl at the front desk.

Someone should be on hand to greet each patient. The newcomer should be made to feel welcome; he should be smiled at and spoken to cordially, by name if he has been in before. If the patient's name is foreign or difficult to pronounce, the receptionist should record the correct pronunciation of the name on the folder or card for the physician's use and for future reference. Use of the patient's name shows recognition and appreciation.

The patient should be given some idea of the probable wait before he can see the physician. If the physician has been called out, the patient should be informed of this, and the receptionist might suggest that the patient use the time for having coffee or shopping. If, for any reason, the physician will be unable to see the patient at the appointed time, every effort should be made to reach the patient at the earliest possible moment by telephone or even by telegram. The physician's staff should show that they and the physician appreciate the fact that the patient's time, too, is valuable.

The physician himself can do much to make the patients awaiting their turns in the reception room feel his friendly interest. A smile or nod in recognition and welcome as he catches sight of an old patient will accomplish wonders. A high degree of intelligence is needed to increase the patient's feeling of his own individuality and to convince him that his best interests are being respected. The physician should insist that the office nurse usher in patients in their turn, rich and poor alike. Patients should not be turned away because they have neglected to make appointments if they can be fitted into the appointment schedule.

Special provision should be made for the handling of exceptional patients, such as the patient with facial deformity who shuns contact with others, the patient who coughs and sneezes, the drunk or belligerent patient, and the talkative, forgetful, and argumentative elderly patient who needs kindly and tactful attention.

Patients expect a sense of reserve on the part of the physician and his employees. If the receptionist or office nurse gossips and discusses the idiosyncrasies of the man or woman who has just left the office, the patient receives the impression that the physician, too, is probably too talkative about his patient's concerns. If charts are left out in the open where they can be studied by all comers, and, if names are used loosely in telephone reports of laboratory or physical findings, patients are uneasy. Closed doors add to feelings of privacy. The waiting room serves as the physician's showcase, and the secretary or receptionist should be considered a mirror of the doctor himself.

CONCEPT OF THE "PERSONAL PHYSICIAN"

Men in medical practice today fall into one of two categories: the specialist and the general practitioner. The specialist publicly announces his intention to limit his practice to some specialty, possibly to the study of one organ or one disease process. The general practitioner, on the other hand, purports to fill the place of the beloved old-time family physician—a family physician, however, with modern techniques and knowledge.

The public is confused by the multiplicity of medical specialities, yet it has come to think of the general practitioner as a medical drudge who can handle small, routine medical problems adequately but is hardly to be trusted with serious medical disease. The public has become very specialist minded.

Ernest Dichter, in his report to the Committee on Medical Economics of the California Medical Association, has suggested that the term "general practitioner," to which such disapprobation has attached, should be replaced by the concept of the "personal physician," a specialist in medical management whose job it is to administer, secure, and supervise the best available care for all medical and surgical problems. While the specialist limits his study to one disease or one organ, the personal physician gives his attention to the disease process in the whole person. Moreover, he sees the whole person operating in a specific family set-up and within a definite socioeconomic framework.

For example, the specialist, focusing his attention on the effects of tension and overwork on a single organ or group of organs might say, "Stop worrying. Go to a beach resort for a period of at least three months, and rest up." This advice, while practicable for patient A or B, may be wholly unrealizable when applied to patient C, who is barely able to support herself and her bedridden mother. The personal physician, knowing the circumstances, that a three month holiday is out of the question and that the suggestion would only add to the patient's distress and give her additional cause for worry, might suggest a more practical alternative in the light of the patient's situation, such as a rearrangement of work hours, frequent rest periods, week-ends in bed, and the like. Knowing the patient's background, the personal physician is able to sift his observations with shrewdness and judgment not always given to the specialist.

SYMPATHETIC HANDLING IMPORTANT

In the physician's inner sanctum, the physician-patient relationship is defined as the physician listens with calm, unhurried, and sympathetic attitude to the patient's version of the problem. The physician will often learn much in this way that he might miss in the more direct question-and-answer type of interview. Kindness and tactfulness are most important. It is not easy to be pleasant as one nears the end of a long day, but every patient needs good humor, sympathy and a quiet manner, no matter how weary or upset the physician may be personally. At times, high tension in the patient may be relaxed by a pleasant bit of conversation or a momentary diversion.

The physician should concentrate his entire attention on the patient during the consultation. His desk should be free from distracting material. If possible, he should refrain from answering the telephone while seeing a patient. Patients should receive special handling with reference to age and position. The older man expands in an atmosphere of respect and deference; the aging woman objects to any act that may emphasize her frailty. Adolescent youngsters object to being "treated like children."

Careful and efficient physical examination and performance of indicated laboratory procedures impart to the patient the feeling that a sympathetic and capable friend is in control of the situation. If the examination is made with authority and assurance, the procedure itself has psychotherapeutic value and the medications prescribed often acquire psychological meaning that increases their efficiency beyond their pharmacologic value.

A common criticism of the medical profession is that the physician does not explain the patient's illness to him. Explanations need not be lengthy or complicated. Simple explanations couched in terms the patient can understand and possibly accompanied by simple line diagrams, are sufficient. Explanations should include the following: (1) what the condition involves; (2) the chances of recovery; (3) the duration of hospitalization; (4) the length of convalescence; (5) the probable cost; and (6) how soon normal activities can be resumed. The patient should be invited to become an active participant in the whole treatment.

False hopes should not be aroused. Patients usually react to bad news more favorably than members of the family. If the patient's fears are unfounded, the physician should not laugh at the fears; he should explain them away instead. Patients whose symptoms cannot be explained by physical findings or who report symptoms out of all proportion to observable pathological conditions often make speedy recoveries after the nature of their illness is explained to them. Physicians should be slow to dismiss an illness as psychosomatic. Apparently psychotains viable organisms (dormant) even though nutrients are absent. Controls in table 3 clearly illustrate that the methods of sanitization employed by hospitals are often inadequate and are therefore conducive to postoperative infections that are usually attributed to other causes.

Particular significance was placed upon the transmission of tuberculosis bacilli, and with this in mind studies were initiated to depict this possibility. Acid-fast Mycobacterium tuberculosis var. hominis and Myco. tuberculosis var. bovis were never shown to be present in apparatus examined; however, patients who were known to harbor tuberculosis bacilli were not anesthetized in any of the cases. Although tuberculosis bacilli were absent, several pathogenic species of microbes were isolated and identified. Pyogenic Micrococcus, Streptococcus, and Pseudomonas aeruginosa were frequently present. These organisms are likely to become secondary invaders and in addition produce serious primary infections in their own right.

Saprophytic micrococci and bacilli were constantly isolated, and even though they are nonpathogenic per se they are occasionally incriminated as secondary invaders complicating more acute diseases. Species of organisms of the genera Bacillus (B. subtilis) and Micrococcus (M. citreus) depict secondary infective agents commonly observed. Viruses were not included in this report although they are certainly transmitted by anesthetizing apparatus: Virus infections would appear to be the most frequently transmitted agents owing to their submicroscopic size and distribution.

Several species of unidentified hemolytic microbes of the genera Micrococcus, Streptococcus, and Bacillus were represented, but their relation or etiology to disease is dubious. These organisms occur with greater frequency in the ether-air tubes than in the rebreathing bags. Aspergillus organisms and one other unidentified organism were obtained in tremendous numbers from the rebreathing bags of some of the apparatus tested. This may be due to the collection of debris in the rebreathing bags, which serves as an excellent pabulum, or to the resistance of the fungal spore. Table 4 will elucidate more emphatically the type of genera and species that have been isolated and identified.

Table 4.—*Species of Microorganisms Identified from Anesthetizing Apparatus*

- Bacillus species
  - B. subtilis
  - B. mycoides
- Micrococcus species
  - M. citreus
  - M. pyogenes var. aureus
  - M. pyogenes var. albus
- Streptococcus species
  - Str. pyogenes
- Gram-negative bacilli
- Aspergillus species
- Alternaria species
- One unidentified genus
- Pseudomonas species
  - Ps. aeruginosa
- Flavobacterium species
- Sarcina species
  - S. lutea
- Candida albicans

Apparatus removed from the patient and immediately sanitized according to routine methods employed (rinsing with tap water) were found to contain viable and dangerous organisms; however, sanitization with water and pHisoderm® with 3% hexachlorophene proved to be an appropriate procedure. This method is suggested for sanitizing anesthetizing tubes and rebreathing bags prior to or immediately after use. Table 5 illustrates the effect of cleansing methods upon the resulting degree of sanitization.

SUMMARY

The passage of microorganisms from the patient to the rebreathing bag of the apparatus used for administering gaseous anesthetics occurs with unrecognized frequency. If the apparatus is not properly sanitized, pathogens may be disseminated to subsequent patients. One must not come to rely upon the removal of microbes by the soda lime tower of the apparatus. These studies demonstrate beyond doubt the passage of microbes from the patient through the tower to the rebreathing bag.

Table 5.—*Effect of pHisoderm® Containing 3% Hexachlorophene on the Degree of Sanitization*

Apparatus Tested	Total Bacterial Counts	
	Tube	Rebreathing Bag
D-1	200	200
D-2	1,300	800
D-3	200	100
D-4	500	600

From a preliminary survey of the procedure for administering gaseous anesthetics and the sanitization of used apparatus, it is apparent that more appropriate methods are required. Merely rinsing with water is not sufficient and is not advocated; however, a substantial reduction in the microbial count is obtained by cleansing with pHisoderm® containing 3% hexachlorophene. The pHisoderm® can then be removed by rinsing with water. Frequent cleansing of the rebreathing bag prevents the accumulation of debris, which serves as an excellent medium for the growth of microorganisms. Cognizance should be taken of the data presented in this report, and thus another possible source of disease transmission can be eliminated.

Sir Charles Scott Sherrington (1859-1952).—Running throughout all of Sherrington's work is a broad philosophy to which he seldom referred except by indirection. But it led Leon Asher, when the University of Berne conferred an honorary degree upon Sherrington in 1931, to refer to him as "the philosopher of the nervous system." This brief citation brought a thundering ovation from the huge audience of neurologists, representing virtually every nation of the world, who had gathered at Berne for the first International Neurological Congress. . . . Through the years of his remarkable scientific productivity Sir Charles had continued his interest in poetry, art, and the cultural backgrounds of medicine. It was not widely known that he was an accomplished classical scholar, but perusal of his more recent books, such as "*Man on His Nature*" and more particularly his "Endeavour of Jean Fernel," will clearly reveal his familiarity with classical texts in which he had always found so much pleasure and relaxation. In preparing for the Fernel, Sir Charles had immersed himself in Galen and the contemporary literature of sixteenth century France. The result is a work of ripe scholarship that would be the envy of any scholar of the period who might have devoted an entire life to the subject. . . . It has been said of him that his genius lay in his extraordinary capacity for translating the facts of morphology into physiological language. This he had done with notable clarity at the spinal and medullary levels; he also elucidated the organization and somatic functions of the cerebral cortex, but he disclaimed any credit for advancing knowledge of mental functions. When discussing the brain as an organ of the mind, Sir Charles insisted, as recently as 1947, that we are no farther along today in accounting for mental processes in physiological terms than was Aristotle two thousand years ago.—*J. F. Fulton, M.D., Sir Charles Scott Sherrington, O.M. (1857-1952), Journal of Neurophysiology*, May, 1952.

FROSTBITE: AN ANALYSIS OF SEVENTY-ONE CASES

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Though frostbite is a well-known and serious malady, knowledge of its pathogenesis and of the optimal methods of its management is far from complete. This is due in large part to the paucity of cases coming under the scrutiny of any single group of investigators. The war in Korea in the winter of 1950-1951 resulted in a high incidence of frostbite among our troops. Seventy-one of these patients have been cared for at the Murphy Army Hospital and form the basis for this report. All these soldiers suffered from true freezing of the hands or feet, with resultant necrosis involving at least small areas of deep tissues, and with gangrene of the digits and forepart of the feet in several patients. No cases of cold injury short of this extent are included.

The climatic environment and the combat situation combined to form the epidemiological background of the disease. Most of the patients were frostbitten in north central Korea in the week between Nov. 27 and Dec. 3, 1950. According to information supplied by the Air Force, the temperature in that locality and time reached minimal levels of 12 F. The wind attained a maximal velocity of 20 miles per hour. Our patients were mainly infantrymen. Their protection against the weather was adequate until they were attacked by overwhelming numbers and forced to retreat. The effect of a more stabilized battle line and a more southerly position, which obtained after Dec. 3, served sharply to reduce the incidence of frostbite, though temperatures later dropped as low as -13 F.

The chronology of evacuation is pertinent to the timing of observation and treatment. The time of removal from the field varied from 1 to 11 days after the first onset of symptoms. An aid station was reached in several hours to two days, where the patients usually remained for two to seven days. Further evacuation was by air, the patients arriving at an overseas hospital within a few hours and remaining for about 20 days. Most patients reached the Murphy Hospital three to four weeks after freezing, exceptional cases falling in a range of 17 to 40 days.

We shall describe the clinical picture of the disease, dividing it into acute, subacute, and chronic stages. The acute stage corresponds to the first three or four weeks spent in the overseas medical installations and was characterized by freezing, thawing, exudation, and the initiation of necrosis. Data on this stage were obtained from the patient's history and his medical record. The subacute stage was the one during which the patient was observed at the Murphy Hospital. It was marked by a resolution of the necrosis and the commencement of vasospasm and cutaneous acrosclerosis. From former experience we may expect a chronic stage, consisting of a continuation of the vasospasm and sclerosis, as well as occasional recurrent ulceration.

Data will also be presented bearing on certain unsettled aspects of the frostbite problem, namely, predisposing factors, factors determining severity, the effectiveness of anticoagulant therapy, the recognition of the presence and extent of gangrene, and the late effects of freezing. In 27 patients with severe lesions, special attention was paid to evidence of preexisting cold sensitivity, including examination of the blood for cold agglutinins. The status of the blood vessels was determined, and the Pachon oscillometer was used as an objective check on the examination. The presence and degree of sweating were recorded in several patients through the aid of photography after the ferric chloride and tannic acid reaction.[1] A biopsy of the skin of the extremities was performed in eight patients. Roentgenograms were made of all the involved extremities.

CLINICAL DESCRIPTION

General Data.—The patients numbered 71 and were all men. The majority were 20 to 23 years of age, the range being 18 to 40. In 67 the feet, and in 3 the hands, were the only parts seriously affected. One patient had severe frostbite of both the hands and feet. No instances of freezing of other portions of the body came to our attention. At least superficial necrosis had resulted in all the patients, and gangrene of entire digits in several. Major amputations, comprising those of fingers or of toes and metatarsal heads, were performed in five patients and will be necessary in two more, a total of 10% of all patients. Six more had surgical removal of portions of one or more toes. Finally, in all of the remaining patients, there were at least localized areas of deep necrosis that did not require formal débridement.

At the time of writing of this report 64 patients have recovered, 63 having been sent to duty and 1 discharged for neurosis. Six patients spent 44 to 60 days in the hospital; 50 patients, 61 to 118 days; and 8 patients, 120 to 165 days. Seven patients are still hospitalized.

Predisposition.—The precipitation of the disease in individual cases appeared related either to an exceptional aggravation of the environmental influence or to some predisposition in the soldier himself. Examples of environmental factors noted included removal of gloves or shoes after wounding, accidental wetting of the feet, and exceptional immobility. Three illustrative cases may be cited.

CASE 1.—A 20-year-old infantryman, on the line for several weeks, suffered no ill effects till he received multiple shell fragment wounds of his left forearm and right hand on Nov. 28, 1950. He could now no longer wear his gloves. In the next few days, continuing a retreat on foot, he felt numbness and throb-

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[1] Silverman, J. J., and Powell, V. E.: A Simple Technic for Outlining the Sweat Pattern, War Med. 7: 178, 1945.

bing in the hands. On Dec. 2 he suffered a perforating gunshot wound of the middle of the left foot. He crawled through the snow on hands and knees for a few hundred yards, with the hands and left foot exposed. At a first aid station, one-half hour after the second wounding, the hands and feet were observed to be cyanotic, blistered, and swollen. On Dec. 5 he arrived at a hospital, where stellate and lumbar sympathetic blocks were performed; but, despite this, mummification of all the fingers, the left great toe, the ball of the left foot, and the tips of the second, third, and fourth left toes ensued. Only bleb formation and superficial necrosis occurred in the right foot.

Amputation of the fingers and distal part of the thumb of both hands and of the left great toe and metatarsal head was performed on Jan. 25, 1951. Both hands now show residual loss of ulnar blood flow, this being more marked in the left. The pedal pulses are normal, but the feet are cold, sweaty, and cyanotic, and the skin is somewhat rigid.

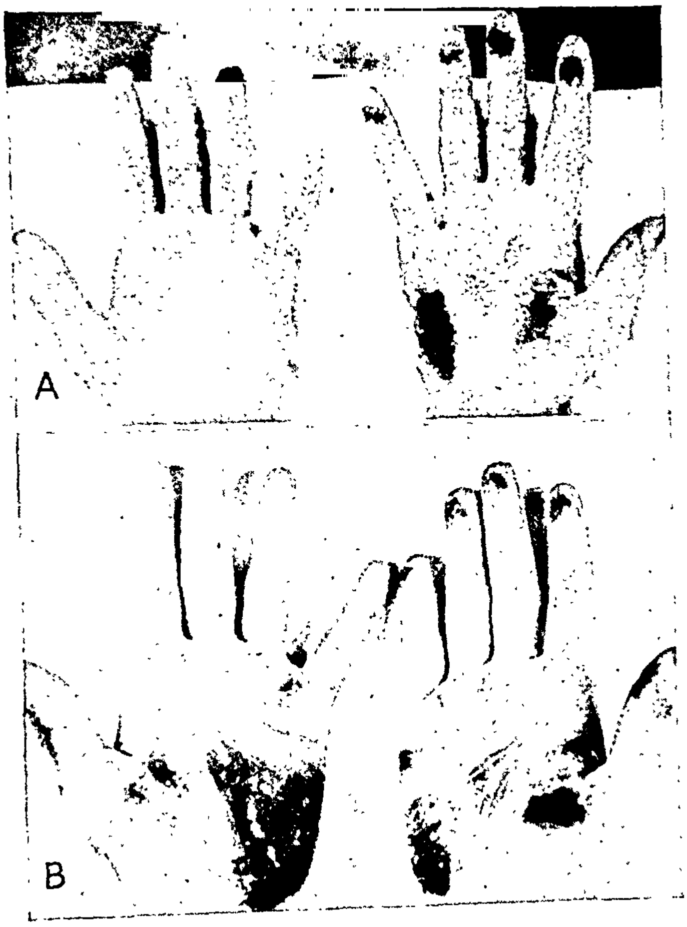

Fig. 1.—The sweat pattern in the hyperemic and in the vasospastic stages, outlined by ferric chloride and tannic acid. The hands shown are those of the patient in case 3, *A*, 5 weeks, and *B*, 14 weeks, after freezing. In the hyperemic stage the left hand (*A*), more severely frozen, shows none of the sweat reaction. The sweating of the right hand is increased. The left hand has entered on the cold hyperhidrotic stage (*B*), with excessive sweating proximally, but with failure of sweating in the distal, most seriously injured parts. The tapering, shiny appearance of the left fingers is characteristic of the acrosclerosis. Sweating, perhaps in increased amount, is expected to return later in the fingers of the left hand.

Case 2.—This soldier, surrounded by the enemy, hid in caves from Nov. 2 to Nov. 13, 1950. He was well clad but had no gloves. On Nov. 4 he wet his feet in a river. He had no fire for food or warming and did not change his socks. Numbness and pain of the feet resulted. When rescued on Nov. 13, he was found to have redness of the feet, without swelling, and black, dry toes. He reached a hospital Dec. 1, where he received 100 mg. 2-benzyl-2-imidazoline hydrochloride (priscoline®) daily. Bishydroxycoumarin (dicumarol®) was also given from Dec. 6 to Dec. 11. Gangrene of all the toes necessitated bilateral transmetarsal amputations on Dec. 27.

No history of previous sensitivity to cold was elicited. Also, the patient was certain he had not frozen his hands; nevertheless, the hands were cold and cyanotic, and there was moderate pterygium of the fingernails. The feet were similarly cold and cyanotic, to above the ankle on the left and to the middle of the leg on the right. The skin of the feet was rigid and smooth. The peripheral pulses were normal. The patient was comfortable, and it was decided to give him a trial of activity.

Case 3.—An 18-year-old infantryman found himself surrounded by the enemy on Jan. 1, 1951. He was in a foxhole, with his rifle at the ready for 12 continuous hours. In this position the left hand rested on the snow, while the right hand was at the trigger close to the body and lower in the foxhole. In spite of the protection of ski mittens, the left hand became numb and stiff. The next day he reported at a first aid station, where the left hand was found to be white and cold. On the third day it was blistered. On his admission to a hospital Jan. 7 he was given 15 intravenous injections of procaine and nicotinic acid. The blisters formed thimble-like eschars, and the nails blackened. Very slight vesiculation and superficial eschars were found only at the very tips of the fingers of the right hand. The feet were slightly uncomfortable but showed no objective changes.

On examination Feb. 2, it was seen that eschars of both hands had separated. The nails of the left hand, now black, were being shed, and the left hand itself had the characteristics of a "trench foot," being extremely hot, red, swollen, and dry. The right hand was in vasospasm, with marked sweating. The peripheral pulses were normal. Nine weeks later the left hand had become cooler, marked sclerodactyly had developed, and new nails were growing. Sweating had returned everywhere except to the fingertips (fig. 1).

Twenty-seven patients, including those with the most extensive gangrene, were studied with special reference to personal factors that might have predisposed to cold injury. Physical signs taken to indicate the presence of vasospastic disease were coldness, cyanosis, sweating, pterygium of the fingernails (widened cuticles),[2] and rigidity and smoothness of the skin over the digits and the dorsa of the hands and feet. When patients were observed early, and when they could remember the chronic presence of one or more of the signs, a diagnosis was made of the existence of vasospastic disease prior to the frostbite. When signs developed several weeks after freezing, they were interpreted as effects of the cold injury. A history of previous frostbite or trench foot, with residual sensitivity to cold and excessive sweating, was obtained in three patients.

Case 4.—A 25-year-old soldier on Jan. 14, 1951, suffered frostbite of moderate severity in the left foot only. Significant in his history was frostbite of the same foot in January, 1950, while he was a ski trooper with the Army in Alaska. Freezing had been attributed to excessive tightness of a leather strap across the ankle, and he had been hospitalized till July, 1950. Since then the left foot had been cold and sweaty, and painful in cold weather. The patient was exceptional in that he smoked but rarely.

Examination showed an eschar of the left foot and toes that was superficial everywhere except at the very tips of the first and second toes. The foot was cold and sweaty. The right foot and both hands were slightly cool. The pulses were all intact. A left lumbar sympathectomy was performed on Feb. 9, 1951. Progress was satisfactory, and the foot was healed and comfortable by March 30.

Nine patients gave a prior history of coldness, either constant or episodic, and two also had hyperhydrosis. In two additional patients, hyperhydrosis was the only symptom. Cold isoagglutinins were found in only one patient. Whether the presence of isoagglutinins played any part in the frostbite in this person could not be determined. No examination was made for cryoglobulins. No patient gave any history suggestive of cold allergy.

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[2] Edwards, E. A.: Nail Changes in Functional and Organic Arterial Disease, New England J. Med. 239: 362, 1948.

The group of 27 patients was questioned as to their use of tobacco. Twenty were steady smokers of 10 to 30 cigarettes a day. Of the three patients who smoked infrequently, never using more than five cigarettes a day, two had other predispositions to freezing (previous frostbite in one and cold agglutinins in the other), while a third had suffered only minor effects from the frostbite.

Wounding by gunshot or shell fragment seemed clearly to precipitate freezing in six patients. Freezing had either not occurred until the injury or was greatly intensified by it. It must be noted, however, that two of the six patients had suffered from chronic vasospasm and sweating and that a third had suffered from prior frostbite.

Case 5.—For seven days, ending Dec. 1, 1950, this 23-year-old man was in continuous combat, with no shelter or hot food. The feet were sweaty and soon became numb. On Dec. 1 he received two gunshot wounds, one bullet passing through the superficial tissues about the left knee, the other perforating the right side of the chest, with hemoptysis and subcutaneous emphysema. He was in shock and unconscious during evacuation by truck. At the aid station, on Dec. 2, the feet were found to be "dead white," swollen, and blistered. On Dec. 4, when he reached a hospital, the toes were black and the feet sweaty and cyanotic. His wounds were débrided and the chest closed. Whiskey was given daily.

When seen Jan. 5, 1951, he was observed to have gangrene of the tips of the toes and the distal third of the left great toe. The nails of the right second, third, fourth, and fifth toes had been shed, and the others were dark. The right dorsalis pedis and posterior tibial pulses were of good quality. On the left side the dorsalis pedis could not be felt. There was extreme sweating and coldness of the feet, but the toes did not sweat. A roentgenogram revealed extensive demineralization of the metatarsal heads of the left foot. The hands showed pronounced sweating, coldness, rigidity of skin, and pterygium formation. The patient stated that the coldness, sweating, and enlarged cuticles had been known to him since youth. Paravertebral blocks and intravenous injections of procaine failed to hasten healing or diminish the pain in the left great toe. A left lumbar sympathectomy Jan. 28 resulted in good vasodilatation. After the left great toe was amputated, healing was prompt.

Clinical Course and Management.—1. Acute stage: Data on the initial period of the disease were obtained from the patients and their medical records. The universal immediate symptom was numbness of the affected parts, sometimes preceded by a prickling sensation. Before thawing, the parts were white; after thawing, however, redness, swelling, moderate heat, and a burning sensation were noted. These symptoms were apparently much milder and more transient than those seen in trench foot and usually required no sedation. Blistering, with the formation of large bullae, was also seen within a day or two of thawing. The blisters were at first filled with clear yellow fluid, but this became hemorrhagic within three to five days. A similar process apparently took place between the nail plates and their beds, for hemorrhage occurred beneath many of the nails, giving them a black appearance. Some of these nails were shed within several days, others after a few weeks, as they were pushed forward by a regenerating nail plate. The parts were described as turning black about the fifth day. The blistering and discoloration were distributed over wide zones of the digits and of the hands and feet. The extent of this change bore no relation to the later demarcation of deep necrosis or gangrene (fig. 2).

Thawing of the extremities was ordinarily not possible until the patient arrived at the aid station, because continued freezing often occurred during evacuation from the field. Once the patient was in the aid station, thawing was usually allowed to continue in the room atmosphere. Other treatment consisted only of dressings, tetanus toxoid, and penicillin. Treatment at the overseas hospitals almost invariably included the use of vasodilator substances. About two-thirds of the patients received procaine hydrochloride intravenously in a dose of 1 gm. in 1,000 cc. of isotonic sodium chloride solution, which was repeated daily up to 15 doses. Nicotinic acid, 300 mg. a day, was given alone or with the procaine to about the same number of patients, while priscoline® and whiskey were each used sporadically. Sympathetic nerve block was performed in only four patients. Anticoagulants were used in only two patients, one receiving heparin during the first four days, another bishydroxycoumarin for five days one month after freezing. Both suffered extensive gangrene.

2. Subacute stage: Necrosis was the presenting problem on admission of the patients to the Murphy Hospital in about the third week of the disease. Pain was minimal but was increased when infection was present. A black eschar was present over variable portions of the toes and feet, and in three instances over the fingers and hands. In two patients necrosis involved the upper heel, penetrating the insertion of the Achilles tendon. Mummification of gangrenous digits was beginning. Infection was usually minor. The extent of true gangrene could usually not be accurately determined till about the sixth week, since even the superficial portions of the eschar required about four weeks for their separation.

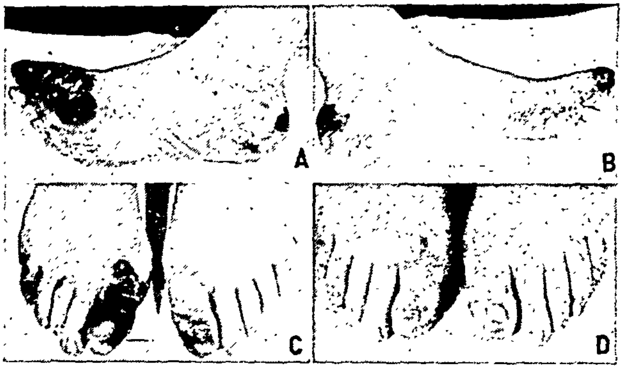

Fig. 2.—Necrosis, resolution, and initiation of acrosclerosis. *A, B,* and *C,* three weeks after freezing, show the extent of black eschar. In *D,* 12 weeks after freezing, the slough has separated, disclosing the necrosis to have been superficial. The great toes are, however, shrunken, with tight, rigid skin and regenerating stubby nails. The sclerosis is less pronounced over the other toes and on the dorsa of the feet.

The duration of freezing after the initial symptoms were noted was probably the most significant single factor in determining the severity of tissue damage. When freezing lasted less than one day, the deep necrosis was usually minor in extent; when it lasted two days or more, at least parts of the digits were lost. Patients who were predisposed to freezing in general suffered severer effects than the others. Wounding in the limb or elsewhere was the most significant factor in this regard.

Roentgen studies of the bones proved of limited value. The bones of gangrenous digits revealed no change at all, evidently because of the lack of metabolic exchange (fig. 3). More localized areas of soft tissue necrosis were occasionally associated with absorption in the underlying bone, with an irregular line of the cortex and a disorganization of the trabeculae, which appeared reticular in arrangement (fig. 4). The typical gross disruption of osteomyelitis was seen beneath badly infected lesions. The atrophy of disuse was seen late, usually about the eighth week of the disease, and it was manifested by a glassy appearance of the metatarsal heads and of the bases of the proximal phalanges (fig. 3).

The occlusion of a peripheral artery was probably indicated by the absence of the dorsalis pedis pulsation on one side, which was noted in 3 patients out of the specially examined group of 27. The lack of pulsation was accompanied by an asymmetry of the oscillometric reading. Only one patient of the three had suffered from previous cold injury.

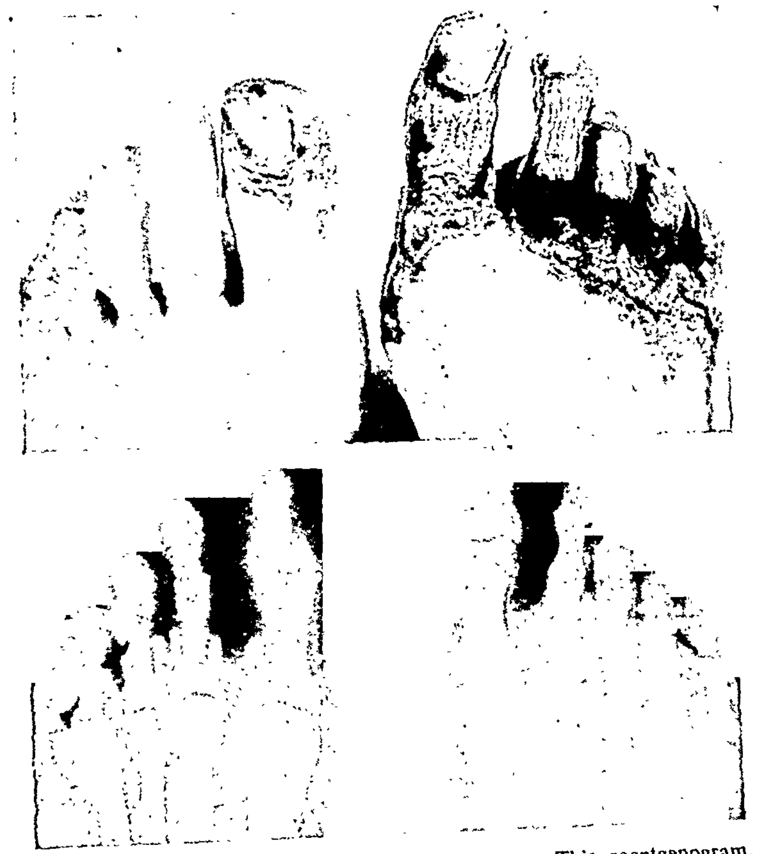

Fig. 3.—Gangrene and the roentgen appearance. This roentgenogram, made eight weeks after freezing, reveals no change either in the gangrenous toes or in the toes showing superficial necrosis. On both sides there is absorption of the heads of the metatarsals and of the bases of the proximal phalanges.

Sympathetic paralysis with hyperemia and dryness of the extremities was seen in a few hands and feet in which the freezing had been only moderate (fig. 1). In some patients with extensive necrosis, a brief hyperemic phase had been noted in the acute stage. Sweating was absent in cold areas from which a fairly deep eschar had separated. In these areas atrophy of the sweat glands may have been important. The absence of sweating, whether or not associated with hyperemia, was often replaced by excessive perspiration as vasospasm supervened.

Vasospasm has already been described, and the point was made that this change often existed in some patients prior to the freezing, while in others the vasospasm was evidently the result of the frostbite, becoming manifest in several weeks. When only one limb had been frozen, the presence of the vessel change in that limb alone supported the contention of its causation by the freezing.

Vasospasm was a common effect in the subacute stage, although it was not extreme in degree unless the patient had suffered from prior chronic vasospasm.

Neurological changes consisting of sensory loss were evident in all cases. The loss of sensation was never absolute except over gangrenous parts. It was "glove" or "stocking" in extent, the hypesthesia being most pronounced distally, with normal sensation present at the ankle or wrist. The sensations most affected were light touch and fine temperature discrimination. A light pinprick was often not felt in the most distal parts of the limb, but a stronger pinprick was discernible and pain could be elicited on strong pressure. Deep pressure and vibration sense were not impaired. The neurological findings were consistent with the lack of pain as a complaint during the early weeks of the subacute stage. Pain was felt, however, when active infection was present, and pain was a frequent complaint after the eighth week of the disease, at a time when sensory acuity had returned. The evidence for sympathetic paralysis, and the vascular changes and sweating indicative of sympathetic irritation, have already been discussed.

Cutaneous atrophy and sclerosis were evident over areas where superficial necrosis had been present and to a lesser extent proximally, to the line of the ankle and wrist. The signs of this acrosclerosis became apparent about the sixth week of the disease but certainly progressed somewhat for an additional four weeks. They consisted of shortening of the digits, with thinning of the digital pulp pads; irregularity, thickening, and shortening of the nails; loss of normal wrinkling and surface friction of the skin on the dorsa of the digits, hands, and feet; and rigidity of the subcutaneous tissue (fig. 1 and 2). The changes were those ordinarily described as acrosclerosis. Vasospasm was apparently constantly associated with these changes, but the two processes were not proportional in degree.

A biopsy of the skin was performed in eight patients, specimens being taken from toes or dorsa of the feet, and in two patients of the hands as well. Since the site of biopsy was considerably proximal to the gangrene, the histological examination was more significant as showing the changes of acrosclerosis than as revealing the lesions leading to necrosis. The epidermis was thin, with flattening of the basal papillae. Immediately beneath the epidermis the collagen appeared light, but in the deeper cutis and in the subcutaneous tissue the collagen was heavier than usual. The elastic tissue was fragmented. The sweat glands appeared atrophied in some areas. Small blood vessels showed swelling of the endothelium, and here and there intimal proliferation could be seen. In one patient this process had obliterated the lumen of a small artery. Some nerve fibers showed degeneration with fibroblastic replacement. In general, inflammatory response and fibroblastic proliferation were notably minimal.

Treatment was directed toward the following aspects of the subacute state: (1) prevention and treatment of infection, (2) release of vasospasm, (3) débridement of necrosis, and (4) amputation of gangrenous parts. The affected parts were kept covered with sterile dressings, which were moist if infection was prominent and dry otherwise. Penicillin was used at least initially in all patients. Vasodilating agents (priscoline® or beta-pyridilcarbinoltartrate [roniacol®]) were given to those showing much vasospasm. There was no clear-cut evidence that the drugs hastened healing. Lumbar sympathectomy was performed in six patients in whom preexisting vasospasm had been, and still was, severe. This included the three persons in whom one dorsalis pedis pulse was missing. Sympathectomy was distinctly helpful in speeding healing as well as in giving a warm, comfortable limb.

Removal of the superficial eschar was aided by cutting away loosened portions in order to allow an earlier delineation of the extent of the deep necrosis. Deep necrotic tissue was likewise removed from time to time as demarcation became evident, exposed portions of bone being rongeured back. Amputations, when necessary, were performed close to the line of demarcation, with satisfactory healing.

3. Chronic stage: The changes we know to be characteristic of the chronic stage of frostbite could already be observed in most of our patients during their hospital stay. The sclerodactyly noted several weeks after freezing often increased as healing took place. The affected digits thus became shortened, with atrophy of the digital pad, with an irregular nail plate, and with the smooth, rigid skin of the digit and hand or foot already described (fig. 1 and 2). Vasospasm and hyperhidrosis gradually disappeared in some limbs but remained prominent in others, especially where cold sensitivity had existed prior to freezing. Pain was now a more frequent complaint, not only in the scars resulting from the necrosis but also in the more proximal parts.

COMMENT

Etiology.—Stated in simplest terms, an extremity is frozen when its loss of heat is greater than can be offset by local heat production or by local warming from the blood perfusing the limb. As regards heat loss, both the temperature of the ambient air and the wind velocity are important. Humidity is so low at freezing temperatures as to be of negligible importance. Wetting of the foot gear, either by perspiration or by accidental immersion, greatly increases the tendency to freezing, since water transfers heat 23 times more readily than air.

The effective blood perfusion of the limbs is diminished via peripheral vasoconstriction from a variety of causes incident to adverse combat conditions. Even with so-called adequate clothing, a man exposed to severe winter weather often suffers considerable chilling and peripheral vasoconstriction. Under ordinary circumstances this chilling is not allowed to continued uninterrupted for many hours, being relieved by the general warming and vasodilatation occasioned by eating, especially when the food is hot, and by intermittent shelter and warming, especially during sleep. When this relief from chilling is denied, vasoconstriction is unrelieved. Hunger, exhaustion, and anxiety all increase the vasoconstriction. The vasoconstrictive effects of tobacco smoking may aid in this process and help to induce freezing. Wounding appears to be an effective precipitant of freezing, adding the vasoconstriction of shock and hemorrhage, loss of blood volume, wetting of the part with blood, and bodily immobility. Blair states that 35% of all cold injury patients seen in the army had been wounded prior to the development of the frostbite.[3]

It is widely known, in a general way, that persons vary in their response to cold. We have noted more specifically that many of our patients had suffered from chronic vasospasm and that a few had suffered previous freezing or trench foot. It would seem that soldiers showing stigmas of cold sensitivity, or with a history of previous cold injury, are poor subjects for duty in freezing climates. Further search seems indicated for evidence, in cold injury patients, of the presence of isoagglutinins (cold agglutinins) and cryoglobulins. The role of a low basal metabolism might also be investigated with profit, since it is known that the temperature of the extremities in patients with a low metabolic rate drops lower under adverse conditions, such as tobacco smoking, than does that of patients with a higher rate.[4]

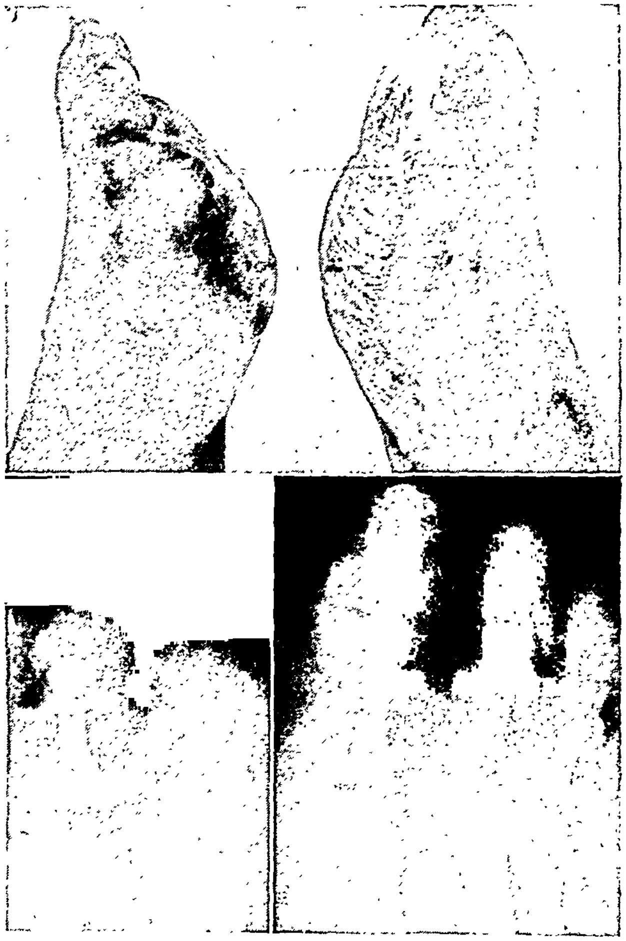

Fig. 4.—Roentgen findings associated with superficial necrosis. The film, made nine weeks after freezing, shows on the right, spotty absorption with disruption of the trabeculae in the head of the metatarsal and in the head of the first phalanx of the great toe (corresponding to the area of maximal injury seen in the photograph). The necrosis of the stump of the previously amputated left great toe was not accompanied by any roentgen change.

Pathogenesis and Treatment.—A brief statement of the mechanisms responsible for the effects of frostbite will suggest the rationale of treatment. Two excellent reviews [5] may be consulted for the present status of our knowledge in this field. It appears that necrosis may be caused by one or more of the following factors: (1) direct injury from the freezing itself, with evidence most striking in the case of muscle[8] and nerve tissue,[6] (2) edema of the hyperemic phase, which ensues soon after thawing,[7] and (3) vascular occlusion by "conglutination" of the blood cells in the vessels in the early hours after thawing, or by larger thrombi in which some fibrin is seen and which develop some days after thawing.[8]

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[3] Vascular Surgery: Cold Injuries, National Research Council, 64-126, 1951.

[4] Roth, G. M.: Tobacco and the Cardiovascular System, Springfield, Charles C Thomas, Publisher, 1951.

[5] (a) Footnote 3. (b) Kreyberg, L.: Development of Acute Tissue Damage due to Cold, Physiol. Rev. 29: 156, 1949.

Anoxia due to lack of dissociation of oxygen from hemoglobin at low temperatures is probably unimportant in the actually frozen tissue, since its metabolism is adequately depressed. Changes in the blood vessels themselves, consisting of early swelling and vacuolization of the intima, with later changes of proliferation, are known to occur early and to continue into the chronic stage. These changes are probably not responsible for the early necrosis but may cause some of the chronic effects.[9] The duration of exposure, once freezing has commenced, seems of extreme importance in the extent of the subsequent gangrene of the entire part. This was true in the experience of Brahdy,[10] as well as in our present study. Rapid thawing is thought by many to significantly reduce the effects of frostbite.[3] Rapid thawing, as defined by Schumacker and Lempke,[3] consists of immersion of the frozen parts in water at 42 C. They agree with others that temperatures as high as 50 C may be harmful and urge also that, once thawed, the part should be allowed to remain at the cooler level of room temperature. Rosenfeld and his co-workers[11] present the contrary view that rapid thawing increases the necrosis, at least after experimental freezing in the dog. Attempts to diminish the effect of the edema by the use of pressure bandages or casts have not altered the course of the disease. Experimentally edema is increased by early sympathectomy or post-thawing heating, with increase in pain and perhaps in necrosis.[5]

The importance of blood cell stasis and thrombosis in producing ischemia and subsequent necrosis was emphasized by students of the frostbite problem in World War II, notably Greene[8b] and Friedman.[8c] Lange and Boyd[12] utilized visualization by fluorescein to demonstrate that maximal hyperemia of the frozen tissues at 4 hours after thawing is followed by a lack of circulatory exchange at 48 hours. These authors suggested the use of heparin to prevent the red blood cell conglutination responsible for the circulatory stasis. Kreyberg[5b] sums up the considerable evidence that heparin does not prevent the early conglutination. Friedman, Lange, and Weiner[13] have come to agree with this view, but nevertheless hold that heparin will prevent the formation of secondary agglutinative thrombi and is therefore important in preventing necrosis. This group emphasizes that the heparin must be given early and in adequate dosage to prolong the coagulation time uninterruptedly for at least five days after freezing.[14] The available reports on treatment of human cases with anticoagulants seem inconclusive. A further, well-controlled clinical trial with this mode of therapy would appear highly desirable.

Our data support the view that the release of sympathetic tone by medical or surgical means is not useful in the acute stage of frostbite. Indeed, experimental work previously available[15] had suggested that vasodilatation may be harmful at that time. It is in the subacute and chronic stages of the disease, when vasoconstriction is prominent, that release of sympathetic tone would seem effective. Surgical sympathectomy in the subacute stage is particularly desirable when it appears reasonably certain that chronic sequelae would otherwise result. At the present we are unable to predict accurately the future course of the disease in all patients. As a working rule we propose that loss of a major peripheral pulse, or severe chronic vasospasm existing prior to the frostbite, be considered indications for the operation. Indications in the chronic stage include recurrent ulceration or pronounced vasospasm and discomfort that have failed to disappear after the freezing. Judging by experience with veterans of World War II, it would seem that a period of about two years might be considered adequate time for such symptoms to clear spontaneously.

SUMMARY AND CONCLUSIONS

The clinical course of 71 cases of frostbite of the extremities, all showing necrosis, is described. The patients were all soldiers, and the precipitating factor was the adverse combat situation. Personal factors of importance in the causation of the freezing were the existence of chronic vasospasm or hyperhidrosis, previous cold injury, wounding, and possibly tobacco smoking.

The disease is described in three stages: (1) the acute, characterized by the freezing, thawing, blistering, and beginning of necrosis; (2) the subacute, during which the necrosis is resolved and vasospasm and acrosclerosis start; and (3) the chronic, when the vasospasm and acrosclerosis continue and pain and recurrent ulceration may occur. Treatment during the acute stage consisted mainly of administration of antibiotics and sympathicolytic drugs. Anticoagulants were used in only two patients. In the subacute stage use of antibiotics and sympathicolytic drugs was continued, and the eschar was gradually removed. Sympathectomy was done in six patients in whom preexisting vasospasm had been pronounced.

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7. Fuhrman, F. A., and Crismon, J. M.: Studies on Gangrene Following Cold Injury: VI. Capillary Blood Flow After Cold Injury, the Effects of Rapid Warming, and Sympathetic Block, J. Clin. Investigation **26**: 468, 1947.

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11. Rosenfeld, L.; Langohr, J. L.; Owen, C. R., and Cope, O.: Circulation of the Blood and Lymph in Frostbite and Influence of Therapeutic Cold and Warmth: Experimental Study, Arch. Surg. **59**: 1045, 1949.

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The superficial necrosis was always more extensive than any underlying gangrene, and observation over several weeks was necessary before amputation was performed. Amputations of fingers or of toes at metatarsal levels were necessary in seven patients, while amputations of portions of toes were necessary in six additional patients.

The duration of freezing after initial symptoms was the most important single factor in the extent of necrosis. Patients showing personal predisposition to freezing, especially those wounded, also suffered most severely. The use of sympathicolytic drugs in the acute stage seemed to have no effect on the outcome. Anticoagulants would appear to deserve a more thorough trial, and more data may help in the question of rapid versus slow thawing.

Sympathectomy appeared to hasten healing in the patients operated on in the subacute stage. It would seem to be indicated when severe vasospasm or other vascular disease has existed prior to the freezing or when the frostbite has caused loss of a peripheral pulse. It is not possible to predict accurately which patients will show long-lasting chronic effects. It is suggested that one wait no longer than two years for spontaneous disappearance of symptoms before performing sympathectomy.

330 Dartmouth St. (Dr. Edwards).

METASTASIS OF RECTAL CARCINOID TO THE BROAD LIGAMENT

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and
E. B. Hanan, M.D., Springfield, Mo.

It is our purpose in reporting this case to call attention to an extremely uncommon tumor of the broad ligament. The tumor in our case proved very confusing as to identity until it was found to be metastatic from a small inconspicuous primary lesion of the rectum.

The adnexae and parametria have been the site of many unusual tumors. Many of these tumors have been difficult to identify as evidenced by the numerous suggested classifications of ovarian tumors. The development of these structures gives rise to innumerable possibilities for different types of tumors and for their appearance in unusual locations. Reports of carcinoids in this region, however, are rare. Stewart and Taylor[1] described a patient with primary carcinoid of the appendix with two metastatic nodules implanted on the pelvic peritoneum; the patient was well 10 years after the removal of the appendix and the two nodules from the cul de sac. Siburg[2] reported a case of carcinoid of the rectum with extensive metastases to sacral and aortic lymph nodes, vertebra, liver, and chest with a rapidly fatal course. Stewart, Willis, and de Saram[3] described two patients with dermoid cysts of the ovary with areas of argentaffin carcinoma, one of whom was well three and one-half years after surgery. We have been unable to find any reported case of carcinoid that resembled an intraligamentary tumor or any case of carcinoid of the rectum with metastasis to this region. We believe that ours is the first reported case of carcinoid of the rectum metastasizing to the broad ligament and also the first reported case of carcinoid of the rectum with known metastasis of four years' duration. Our case is also unusual in that the metastatic tumor was found before the primary tumor in the rectum was discovered, which gave rise to considerable speculation and discussion as to its classification and origin.

As early as 1888 Lubarsch[4] reported two cases of multiple small submucosal cancers of the small bowel composed of nests of uniform cells not resembling cells of the intestinal mucosa, and in 1901 Whipham[5] reported unusual yellow carcinomas of the ileocecal region and referred to 18 cases reported by Kelly, one by Rolleston, and one by Hurdons. Whipham's description of the histological changes was remarkably similar to descriptions by present day authorities, but it was not until 1907 that Oberndorfer,[6] who believed them to originate from the mucosa and to be benign, suggested the term "carcinoid." Saltykow[7] probably reported the first carcinoid of the rectum in 1912. Numerous investigators have objected strenuously to the term carcinoid and have proposed such terms as "argentaffinomas,"[7a] "Kultschitsky cell tumor," and "carcinoma grade 1 (Broders[7b])," but the term carcinoid still prevails. Since Horn and other authorities have demonstrated that not all rectal carcinoids contain argentaffin granules and that some rectal carcinoids contain mucin, it seems that the term carcinoid is probably as good as any, particularly since the malignant potentiality is well recognized but extremely variable.

There are excellent reviews of the literature pertaining to carcinoids of the rectum by Horn,[8] Stout,[9] Pearson

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From the Departments of Surgery, Obstetrics, and Pathology, Burge Hospital.

[1] Stewart, M. J., and Taylor, A. L.: Carcinoid Tumor of the Appendix with Large Pelvic Deposits, J. Path. & Bact. **29**: 136-139, 1926.

[2] Siburg, F.: Über einen Fall von sogenanntem Karzinoid des Rektums mit ausgedehnter Metastasenbildung, Ztschr. f. Path. **37**: 254-269, 1929.

[3] Stewart, M. J.; Willis, R. A., and de Saram, G. S. W.: Argentaffine Carcinoma (Carcinoid Tumour) Arising in Ovarian Teratomas, J. Path. & Bact. **49**: 207-212, 1939.

[4] Lubarsch, O.: Ueber den primären Krebs des Ileum nebst Bemerkungen über das gleichzeitige Vorkommen von Krebs und Tuberculose, Arch. f. path. Anat. **111**: 280-317, 1888.

[5] Whipham, T R. C.: A Case of Primary Carcinoma of the Vermiform Appendix, Lancet **1**: 319-321, 1901.

[6] Oberndorfer, S.: Karzinoide Tumoren des Dünndarms, Ztschr. f. Path. **1**: 426-432, 1907.

[7] Saltykow, S.: Ueber die Genese der "karzinoiden Tumoren," sowie der "Adenomyome" des Darmes, Beitr. z. path. Anat. u. z. allg. Path. **54**: 559-594, 1912.

[7a] Willis, R. A.: Argentaffin Carcinomata ("Carcinoids") of the Small Intestine, M. J. Australia **2**: 400-403, 1940.

[7b] Dockerty, M. B., and Ashburn, F. S.: Carcinoid Tumors (So-Called) of the Ileum: Report of Thirteen Cases in Which There Was Metastasis, Arch. Surg. **47**: 221-246 (Sept.) 1943.

[8] Horn, R. C., Jr.: Carcinoid Tumors of The Colon and Rectum, Cancer **2**: 819-837, 1949.

[9] Stout, A. P.: Carcinoid Tumors of the Rectum Derived from Erspamer's Pre-enterochrome Cells, Am. J. Path. **18**: 993-1009, 1942.

and Fitzgerald,[10] and Pautler and Scotti.[11] Horn's report probably is the most recent and comprehensive; he reports 56 cases, including six of his own. Of the 56 lesions, five were malignant. Since Horn's report, Shepard and co-workers[12] reported two cases of carcinoid of the rectum, both of which were locally invasive but without metastasis. Wilson[13] reported another small carcinoid in the rectum, but in his case there was one metastatic gland at the upper end of the rectum in the mesentery. Early in 1951 Hines and Riddler[14] reported three cases of rectal carcinoids with no mention of metastases, and Mayo and McKee[15] reported two cases of polypoid carcinoids with no known metastases. Seven other cases were mentioned in a paper by Pearson and Fitzgerald,[10b] which were observed at the Memorial Hospital in New York, but no details were given as to location, age, or malignancy. The addition of our case makes a total of 72 cases of rectal carcinoids, in seven of which the lesion was definitely malignant. Some evidence as to the rarity of rectal carcinoids is gained by the fact that Stout[9] found only two cases in 13,809 routine autopsies and Hines and Riddler[14] found only three cases in 30,000 routine proctological examinations.

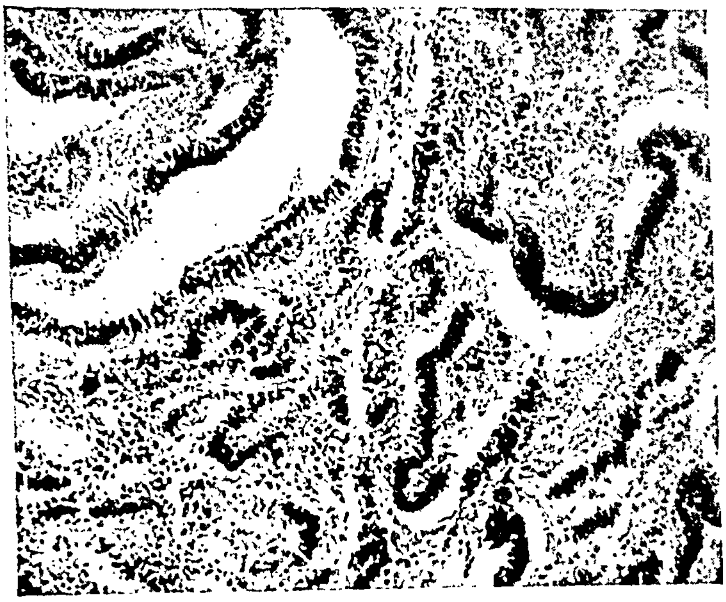

Fig. 1.—Metastatic tumor of broad ligament (X 170).

In spite of the malignant tendency of some carcinoid tumors, several reports show that metastases may lie dormant for several years. Terplan[16] reported a carcinoid of the cecum with metastases in mesenteric glands and liver, in which a biopsy and palliative ileocolostomy was done. The patient died by drowning three and a half years later, and the autopsy showed no progression of the tumor or metastases. Merke[17] reported a case in which a palliative ileocolostomy was done; the patient died of pneumonia and erysipelas five years later, and the autopsy showed no progression of the tumor. Decker[18] reported another case in which carcinoid tumor was found in mesenteric glands, but at autopsy, two and a half years later, no tumor could be found. Mallory[19] reported a most unusual case in which a segment of ileum was resected for carcinoid, with known mesenteric metastases left behind. The patient died 20 years later of pneumonia, and the autopsy showed no further extension of the tumor. Horn's[8] patient with carcinoid of the rectum with extensive metastases lived 17 months, but in this case death was due to metastatic disease.

Another unusual feature we observed in studying the literature was the relatively high incidence of these tumors on the anterior rectal wall, 5 to 10 cm. from the anal margin. In our case the lesion was typically situated on the anterior wall, about 6 to 8 cm. from the anal margin. Of the 72 cases reported in the literature, 22 were of lesions located on the rectal wall. In 14 (an incidence of 64% if the same ratio holds for all of the other cases) the primary tumor was located on the anterior rectal wall, in 3 on the posterior wall, and in 2 on the lateral wall. In two cases the lesion was anular and in one case it was diffuse. In the remaining 50 cases the site was not specified.

REPORT OF A CASE

Mrs. M. T., a 26-year-old white woman, was first admitted to Burge Hospital Oct. 18, 1950, complaining of dyspareunia and a tumor of the left ovary. She stated that the tumor was first found on postpartum examination, in September, 1946, after the birth of her first baby, and that no treatment was advised. She was first seen by one of us (R. N. W.) in October, 1947; she had a normal pregnancy, but the tumor of the pelvis was not noted at the time. She had a normal delivery in April, 1948. On June 23, pelvic examination revealed what was thought to be a prolapsed left ovary. She was examined fairly frequently after this, and in October, 1948, the ovary was thought to be larger. In September, 1949, she again returned, complaining of

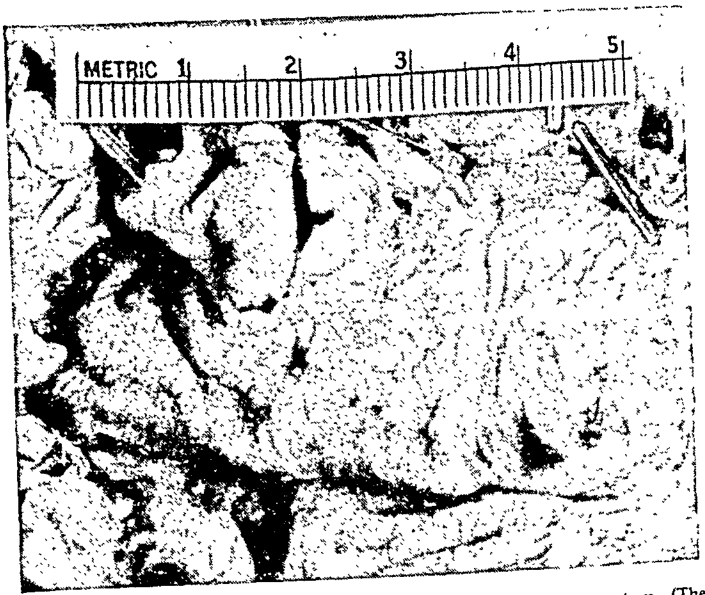

Fig. 2.—Gross Appearance of the primary tumor of the rectum. (The location in the rectum is distorted because the specimen was fixed before it was photographed.)

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10. (a) Pearson, C. M., and Fitzgerald, P. J.: Carcinoid Tumors of the
Rectum: Report of Three Cases, Two with Metastases, Ann. Surg. **128:**
128-143, 1948; (b) Carcinoid Tumors—Re-emphasis of Their Malignant
Nature: Review of 140 Cases, Cancer **2:** 1005-1026, 1949.
Carcinoid in Meckels' Diverti-
11. Pautler, E. C., and Scotti, T. M.: ... Including Usual
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culum: Report of a Case and Review of the Literature Including Usual
and Unusual Sites of Carcinoids, Gastroenterology **17:** 88-95, 1951.

12. Shepard, R. M., Jr.; Strug, L. H., and DiLeo, J. H.: Carcinoid Tumors of the Rectum, Surgery **29:** 205-209, 1951.

13. Wilson, H.: Carcinoid of the Rectum, Arch. Path. **48:** 187-189 (Aug.) 1949.

14. Hines, M. O., and Riddler, J. G.: Unusual Lesions of the Rectum and Anus, Surgery **29:** 496-499, 1951.

15. Mayo, H. W., Jr., and McKee, E. E.: Carcinoid of the Rectum, A. M. A. Arch. Surg. **62:** 506-513 (April) 1951.

16. Terplan, K.; Weintraub, D., and Wolf, N. J.: Stationary Metastasizing Carcinoid of the Ileocecal Valve, Arch. Path. **30:** 1155 (Nov.) 1940.

17. Merke, F.: Das Karzinoid der untersten Ileumschlinge, Schweiz. Med. Wchnschr. **67:** 639-641, 1937.

18. Decker, P.: Contribution a l'étude du carcinoïde, Rev. méd. de la Suisse Rom. **48:** 145-146, 1928.

19. Case Records of the Massachusetts General Hospital: Case 26162 [Carcinoid of the Ileum], New England J. Med. **222:** 684-687, 1940.

low backache and rectal bleeding. An examination at this time revealed the same firm mass in the region of the left ovary, but at this time it was definitely thought to be a tumor in the broad ligament. She was seen in consultation by one of us (C. H. E.); and the diagnosis of an intraligamentary solid tumor was confirmed. A sigmoidoscopic examination disclosed nothing essentially abnormal except for bleeding hemorrhoids. The patient was advised to have the tumor removed at this time, but she declined to do so and she was not seen again until September, 1950, when she returned complaining of dyspareunia, low backache, and rectal bleeding and requested surgery.

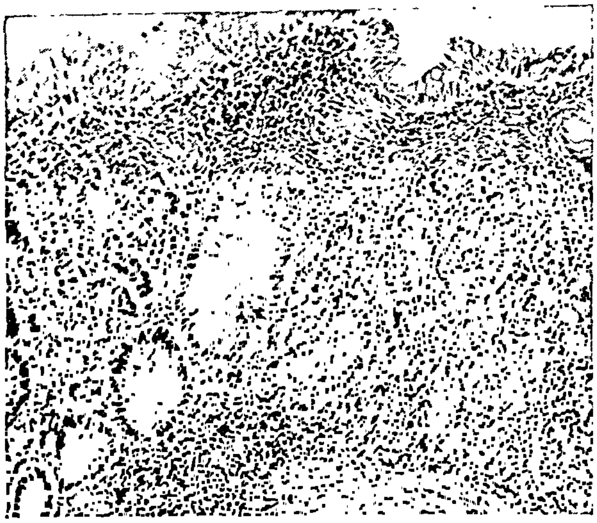
Fig. 3.—Primary rectal carcinoid (× 95).

Her physical examination disclosed nothing abnormal except in the rectum and pelvis. Pelvic examination revealed a parous introitus with fairly good support. The cervix was lacerated bilaterally and mildly eroded, and the uterus was anterior and normal. The right adnexa was normal; but on the left a firm and tender mass, the size of an English walnut, was found deep in the broad ligament just to the left of the cervix. The ovary was palpable anterior to this mass. Rectal examination revealed moderate internal and external hemorrhoids, which bled from the trauma of the anoscope. Laboratory findings were all within normal limits, and the Kahn reaction was negative.

On Oct. 19, pelvic laparotomy was performed. A tumor, approximately 4 by 4 by 5 cm., was found at base of left broad ligament. The peritoneum over the tumor was incised, and the nodule was shelled out easily. Hemostasis was secured and the peritoneum closed. The remainder of the abdomen was explored and found to be entirely normal. The appendix was removed, and the abdomen was closed in the usual manner. The postoperative course was uneventful.

The tumor nodule was studied by one of us (E. B. H.) and thought to be of unusual origin and probably of low-grade malignancy. Dr. Lauren Ackerman (Washington University School of Medicine, St. Louis), who studied the microscopic preparations, felt the tumor was of low-grade malignancy and probably metastatic from the large bowel. Sections of the tumor were sent to several other national authorities, most of whom agreed that it was of low-grade malignancy. Because of its location in the broad ligament, one authority felt it arose from remnants of the wolffian duct; another suggested its origin from the müllerian duct; and another thought it to be a granulosa cell tumor of the ovary. Still another thought it to be a benign tumor arising from remnants of the müllerian duct. On Dr. Ackerman's suggestion a thorough examination was made, which revealed a small tumor on the anterior rectal wall 6 by 8 cm. above the anal margin. A biopsy of this nodule was interpreted as adenocarcinoma.

On Dec. 2, an abdominoperineal resection was done; a portion of the left hypogastric vessels and some of the remaining scar tissue, where the metastasis had previously been removed, were excised. The patient had an uneventful convalescence following this last procedure. The pathologist found the primary tumor to be essentially submucosal, with some muscle infiltration but confined to the bowel wall. In the adjacent lymph nodes and tissue removed from the pelvic wall he found no tumor cells. The final diagnosis was carcinoid of the rectum with retroperitoneal metastasis. Because tumor tissue might have been left on the left pelvic wall, the patient was given a course of roentgen therapy; she was discharged in good condition. During the period Jan. 3 to Feb. 1, 1951, a total of 2,200 r (air) was delivered to each of four anterior and posterior pelvic ports, measuring 10 by 12 cm. each, with the following factors: 250 kv., Thoraeus II filter, and half-value layer of 2.32 mm. copper.

Pathological study of the metastatic tumor revealed it to be firm, 4 by 4 by 5 cm., and well circumscribed, with a slightly bosselated surface. The cut surface revealed a slightly bulging homogenous pale yellowish pink surface with small focal hemorrhages. Microscopic sections showed it to be composed of numerous strands and cords of regular low columnar and cuboidal cells with uniform even-staining nuclei. In most areas the strands were made up of double rows of cells with characteristic festooning as described by Stout (fig. 1).

The second specimen consisted of sigmoid and rectum 30 cm. in length and several small pieces of fibrous tissue. The surface of the colon, rectum, and anal skin all appeared normal, and a careful search of the mesentery failed to reveal any enlarged lymph nodes. When the specimen was opened, the mucous membrane appeared normal except for a 1 by 1 by 1.5 cm. firm elevated nodule on the anterior wall of the rectum 8 cm. above the anal orifice, with no gross evidence of ulceration. The cut surface showed the same firm homogenous white tissue as noted in the metastatic tissue and apparently was limited to the submucosa. The gross appearance is shown in figure 2. Microscopically the tumor was situated in the submucosal layer, pushing the epithelium upward and thinning it out in its center and in small areas producing ulceration. The base of the tumor invaded the muscle superficially but did not extend through the wall. The same cell pattern and architecture as observed in the metastatic tumor were noted. Sections of the rectal tumor are shown in figures 3 and 4.

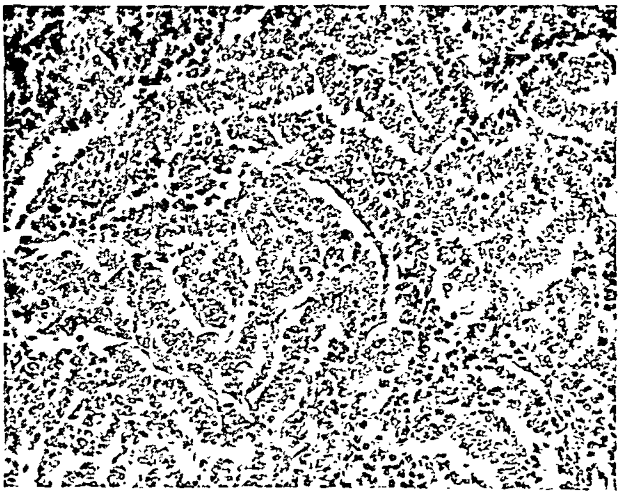
Fig. 4.—Primary rectal carcinoid (× 125).

COMMENT

The pathological changes of rectal carcinoids have been thoroughly studied and adequately described. The carcinoids are characteristically small, firm, hemispherical submucosal nodules situated frequently on the anterior rectal wall 3 to 10 cm. above the anus (table 1). Although Rigdon[20] reported a case in which the entire rectum was covered with small nodules, Pearson and

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[20] Rigdon, R. H., and Fletcher, D. E.: Multiple Argentaffin Tumors (Carcinoids) of the Rectum, Am. J. Surg. 71:822-824, 1946.

Fitzgerald[10] reported a large annular lesion, and Koch[21] reported a similar constricting lesion, the primary tumor is usually small and single. The benignity of metastases in some instances is exemplified in Horn's patient, with 17 months' survival in the face of extensive spread, and in our own patient, with metastasis of four years' duration and no evidence of spread to other organs or nodes.

All authorities are agreed that these tumors arise from the Kultschitzky (basigranular) cells at the base of the crypts of Lieberkühn. Stout refined this further by suggesting their origin from the pre-enterochrome cells of Erspamer, thereby explaining the absence of argentaffin granules in most of these tumors. Erspamer divided the basigranular (Kultschitzky) cells into four groups, of which the pre-enterochrome cells represent an earlier stage of development and do not show an affinity for

TABLE 1.—*Previously Reported Cases of Carcinoid of the Rectum*

Author	No. of Cases	Date	Age of Patient	Sex	Location in Rectum and Size (When Recorded)	Metastases	Treatment and/or Remarks
Saltykow[7]	1	1912	28	F	Anterior wall; 5 cm. above anus	None	Puerperal death; incidental autopsy finding
Staemmler *	1	1924	35	F	10 cm. above anus	None	
Martin and Dechaume †	1	1924	56	M	....	None	Incidental findings with surgical tissue
Siburg[2]	1	1929	71	M	Anterior wall, 4 cm. above anus	Lymph nodes (sacral, aortic), vertebra, liver	Exploratory laparotomy; autopsy later
Brunschwig[23b]	1	1933	63	M	Anterior rectal wall, 8 cm. above rectum	None	Local excision
Feyrter ‡	5	1934	14	M			
			54	F			
			56	F			
			59	F			
			67	F			
Koch[21]	1	1940	64	M	2-3 cm. above anus; infiltrative lesion constricting 10 cm. of rectum to pencil size	Lymph nodes, liver, bladder, ureters	Autopsy finding
Humphreys §	1	1934	28	M	....	Apparently none	Autopsy finding
Mallory[19]	1	1940	?	?			
Ashworth and Wallace ‖	1	1941	31	F	10 cm. within rectum; wall not mentioned	None	Abdominal perineal resection
Stout[9]	6	1942	19	F	Anterior wall; 2 cm. mass	....	Local removal
			41	F	Anterior wall; 5 cm. mass	....	Local removal
			34	F	Anterior wall; 1 cm. mass	None	Local removal
			34	F	Anterior wall; 5 cm. mass	....	Local removal
			42	F	Anterior wall; 7 cm. mass	....	Autopsy finding (mediastinitis)
			43	M	Just above anus	....	Autopsy finding (stomach operation)
Helwig[23c]	1	1943	56	M	?	None	Local (?) excision
Yaker[23e]	1	1944	29	M	Anterior wall; ½ in. tumor 4 in. within rectum	None	Local excision
Rigdon and Fletcher[20]	1	1946	60	M	15 cm. of rectum covered with *argentaffin* nodules	None	Ruptured aneurysm Incidental autopsy findings
Severance ¶	1	1946	33	F	?	None	Routine examination for vaginal bleeding
Dukes[23d]	9	1946	(5 no age given)			Apparently none in any of 9	Incidental findings in 3 in surgical specimen; no follow-up
			40	F	Lower ⅕ of wall?		Local excision
			34	F	Same		Local excision
			36	F	?		Local excision; well 9 yr.
			36	M	?		Abdominal perineal resection
Wilson[13]	1	1949	57	F	....	One node at upper end of resection	Abdominal perineal resection
Shepard[12]	2	1950	61	F	Anterolateral wall	None	Abdominal perineal resection
			51	M	Posterior wall, 6 cm. up	None	
Jackman ‡‡	4	1947	?	?	?	None apparently	Apparently local excision in all
Ehrlich and Hunter **	10	1947	?	?	?	None	All excised locally
Pearson and Fitzgerald[10a]	3	1948	51	M	3 tumors in posterior and anterior wall	Liver, kidney	Exploratory laparotomy; autopsy later
			45	F	Annular lesion	Liver, aortic nodes	Exploratory laparotomy; palliative colostomy
			69	M	Left anterior wall at rectosigmoid junction	None	Local excision
Horn[8]	6	1949	29	F	Right lateral wall, 1 cm. above anus	Adrenals, pancreas, liver, lungs, spleen	Autopsy (death from liver disease)
			42	F	?	None	Local excision
			56	F	?	None	Local excision
			25	F	?	None	Local excision
			..	M	?	None	Local excision
			62	M	?	None	Local excision
Hines and Riddler[14]	3	1951	49	..	Anterior wall, 4 cm. up; 3 cm. nodule	....	Local excision
Mayo and McKee[15]	2	1951	24	F	Polyp, 1 cm., at 6 cm. level	Right lateral wall	
			29	M	Polyp, 1 cm., at 5 cm. level	Right posterior lateral wall	

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* Staemmler, M.: Das sogenannte Karzinoid des Darmes, Neue deut. Chir. 33a: 230-244, 1924.
† Martin, J. F., and Dechaume, J.: Double adénome endocrinien du rectum, Lyon méd. 133: 686-688, 1924.
‡ Feyrter, F.: Carcinoid und Carcinom, Ergebn. d. allg. Path. u. path. Anat. 29: 305-489, 1934.
§ Humphreys, E. M.: Carcinoid Tumors of the Small Intestine: A Report of Three Cases with Metastases, Am. J. Cancer 22: 765-775, 1934.
‖ Ashworth, C. T., and Wallace, S. A.: Unusual Locations of Carcinoid Tumors, Arch. Path. 32: 272-276 (Aug.) 1941.
¶ In Stout, A. P.: Tumor Seminar: Carcinoid Tumor of the Rectum (Case 1), Texas State J. Med. 41: 564, 1946.
‡‡ Jackman, R. J.: Submucosal Nodules of the Rectum: Diagnostic Significance, Proc. Staff Meet., Mayo Clin. 22: 502-504, 1947.
** Ehrlich, J. C., and Hunter, O. B., Jr.: Tumors of the Gastrointestinal Tract: A Survey of 813 in Persons of Military Age During World War II, Surg., Gynec. & Obst. 85: 98-106, 1947.

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21. Koch, F.: Maligne Carcinoide, Chirurg 12: 270-275, 1940.

silver stains. All of the reported cases of rectal carcinoids are shown in table 1. Table 2 shows the relative frequency of carcinoids of the rectum as compared to carcinoids occurring in other unusual locations. Most of the patients described were older than our patient; all the patients with malignant carcinoids were older.

Most authors [22] consider rectal carcinoids, as well as carcinoids in other portions of the bowel, as definitely malignant but slow growing and slow to metastasize. Ackerman [22b] believes the infrequency of metastases in carcinoids of the appendix and rectum is based on the fact that they give rise to symptoms early. In the case of appendiceal carcinoids, the symptoms are obstructive and usually appendectomy is performed before metastases take place. In rectal cases bleeding, tenesmus, diarrhea, and backache are early symptoms. The rectum can be easily and satisfactorily examined, which results in a relatively early diagnosis.

TREATMENT

Most writers have advised conservative management of these tumors.[23] Recently, however, Shepard and coworkers [12] advised a more radical treatment and gave several sound reasons for this. The experience of Stout,[9] Helwig,[23c] and Dukes [23d] would seem to obviate radical surgery if no metastasis can be found and if the local lesions are not too large to be removed easily. It is our opinion, after reviewing the literature and reviewing our case, that these tumors should be suspected by their gross appearance and should be treated conservatively if at all feasible. Ariel [24] and Klemperer [25] suggested that radiation may be effective, and Ariel [21] reported one case of carcinoid of the small bowel treated by surgical excision; however, the patient returned two months later with an abdominal mass at the operative site. This mass was irradiated; it responded well, and the patient was well two and a half years later. Ariel suggested, on the basis of this case, that carcinoids may be radiosensitive. Since some patients have lived for years with stationary metastases, it is difficult to evaluate a reaction of this type. It will be interesting, however, to follow our own case to note whether or not further metastases appear.

TABLE 2.—*Unusual Locations of Carcinoids**

Location	No. of Cases
Rectum	56
Stomach	23
Colon (exclusive of rectum)	22
Meckel's diverticulum	7
Ovarian teratoma	6
Gallbladder	3
Mesentery	2
Diverticulum (unspecified)	1

* Taken from Pautler and Scotti.[11]

SUMMARY

An unusual and uncommon case of a rectal carcinoid metastatic to the broad ligament is reported, which caused considerable speculation and concern in the identification of the first tumor nodule removed. It is our belief that this is the first reported case of malignant rectal carcinoid with known metastasis of four years' duration without spread to other organs. It is one of seven reported cases of malignant carcinoids of the rectum, and the patient in the case reported here is the youngest with proved metastasis.

It is pointed out that even the malignant carcinoids, both of the rectum and other locations, may at times have a remarkably chronic course. The frequency (64%) with which rectal carcinoids appear on the anterior wall of the rectum is mentioned. We believe, after reviewing the literature, that a rectal carcinoid should be suspected when a small tumor of the rectum is found in which the mucous membrane appears intact over the surface and that such tumors should be treated by local excision unless they are diffuse (as in Rigdon's case) or are infiltrating annular lesions (as in Pearson and Fitzgerald's case) or metastatic spread is suspected.

ADDENDUM

At the time of the last examination of this patient on May 20, 1952, 18 months after the abdominoperineal resection, she was well and apparently free of recurrent disease.

800 Medical Arts Bldg. (Dr. Epps).

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22. (*a*) Horn.[8] Pearson and Fitzgerald.[10b] Stout.[9] Wilson.[13] (*b*) Ackerman, L. V.: Personal communication to the authors.

23. (*a*) Stout.[9] (*b*) Brunschwig, A.: Argentaffin Tumor (Carcinoid) of the Rectal Colon, J. A. M. A. **100**: 1171-1172 (April 15) 1933. (*c*) Helwig, E. B.: Benign Tumors of the Large Intestine—Incidence and Distribution, Surg., Gynec. & Obst. **76**: 419-426, 1943. (*d*) Dukes, C. E.: Peculiarities in the Pathology of Cancer of the Ano-Rectal Region, Proc. Roy. Soc. Med. **39**: 763-765, 1946. (*e*) Yaker, D. N.: Carcinoid of the Rectum, Clinics **3**: 1055-1058, 1944.

24. Ariel, I. M.: Argentaffin (Carcinoid) Tumors of the Small Intestine: Report of Eleven Cases and Review of the Literature, Arch. Path. **27**: 25-52 (Jan.) 1939.

25. Klemperer, P., in discussion on Goldberg, S. A.: Unusual Neoplasms of the Small Intestine, Am. J. Path. **14**: 663-664, 1938.

Yellow Fever.—Yellow fever, like typhus, is not dead in spite of all of the conquests and conquerors of this disease, nor is it likely to die out so long as the tropical and subtropical forests maintain their mammalian hosts and insect vectors. Jungle yellow fever remains as a potential source of virus for undoing the conquest of urban yellow fever, wherever Aëdes aegypti is permitted to continue, as witness the 1928-29 outbreak in Rio de Janeiro. . . . In South America, only Chile and Uruguay are free from this disease. . . . A tragic occurrence early in 1951 was an outbreak of jungle yellow fever with over 2,000 cases and several hundreds of deaths, among the newly arrived settlers in the State of Goiaz, Brazil. This area had suffered an outbreak in 1945, at which time extensive vaccination was carried out in the then relatively small population. During the past six years, however, an important agricultural development has brought in large numbers of nonimmunes from Northeast Brazil. . . . Ironically enough, these newcomers came from the previously highly endemic region of aegypti-transmitted yellow fever of Northeast Brazil, where there is no jungle yellow fever. It was here that the Rockefeller Foundation initiated its campaign in Brazil against aegypti-transmitted yellow fever in 1923, with the resultant development of this nonimmune susceptible population. . . . As the years pass and jungle yellow fever continues to be jungle yellow fever, without invasion of the cities, the potential threat seems unimportant to many who do not realize the tremendous effort which has gone, and is going, into the eradication of Aëdes aegypti in all of the towns and cities in close contact with the infected forests.—F. L. Soper, M.D., The Elephant Never Forgets, *American Journal of Tropical Medicine and Hygiene,* May, 1952.

CLINICAL NOTES

LIVER NECROSIS AND DEATH FOLLOWING HEPATIC ARTERY LIGATION

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and
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Recent reports, first by Rienhoff[1] and later by Berman,[2] have indicated that patients with cirrhosis complicated by esophageal varices and ascites may benefit from ligation of the hepatic artery. It has been commonly assumed previously that viability of the liver was dependent on adequate blood supply through the hepatic artery and that ligation of this vessel would result in liver necrosis and death. Markowitz, Rappaport, and Scott[3] reported that in dogs liver necrosis and death following such an operation could be avoided, in most cases, by administration of penicillin, indicating that the necrosis was probably the result of infection from the anaerobic bacteria normally present in the liver of dogs. Grindlay, Mann, and Bollman[4] recently suggested the possibility that in those animals that survived there had not been complete stoppage of the blood supply to the liver through the ligated artery or that collateral channels had been established. These recent reports of successful ligation of the hepatic artery in patients with cirrhosis suggest that in cirrhosis the blood supply to the liver has been so altered that such ligation does not result in damage to the liver and that the function of the liver may even be improved. We have recently observed a case of cirrhosis in which ligation of the hepatic artery was not tolerated and resulted in liver failure and death. This case indicates that ligation of the hepatic artery is not innocuous in all cases of cirrhosis.

REPORT OF A CASE

The patient was a white man, 40 years old, who was admitted to this hospital twice because of massive upper gastrointestinal hemorrhage. He had had an attack of jaundice at the age of 26 and a questionable attack at 34. He admitted excessive use of alcohol for several years, and for the past two or three years, he had had symptoms of indigestion, vertigo, syncope, and edema of the legs.

First Hospitalization.—The patient was first admitted to the hospital on July 12, 1950, after having passed blood in his stools and having vomited a large amount of bright red blood during the preceding four days. Physical examination revealed a soft systolic murmur heard over the entire precordium, several spider nevi over the neck and shoulders, enlargement of the liver 2 cm. below the costal margin, and a small amount of ascites. Laboratory tests made between July 12 and July 20 showed the following results: red blood cell count 2,100,000; gastric analysis showed 20 degrees of free hydrochloric acid; the blood serum contained 1.2 mg. of bilirubin, 5.7 gm. of protein, and 2.8 gm. of albumin per 100 cc.; reaction to the cephalin-cholesterol flocculation test was 2+, and to the thymol flocculation test was negative; a thymol turbidity test showed 5 units, and a zinc turbidity test, 25 units. Tests made between Aug. 20 and Aug. 30 showed the following results: the blood serum contained 0.8 mg. of bilirubin, 7.9 gm. of protein and 4.9 gm. of albumin per 100 cc.; reaction to the cephalin-cholesterol flocculation test was 3+ and to the thymol flocculation test was negative; a thymol turbidity test showed 2 units; and the sulfobromophthalein (bromosulfalein®) sodium retention in the blood was 24%. An x-ray examination revealed a large ulcer crater or pseudodiverticulum of the first portion of the duodenum. Esophagoscopic examination showed large thin-walled varices in the lower esophagus, with no evidence of recent hemorrhage.

The patient's treatment consisted of transfusions, ulcer management, and supportive therapy for cirrhosis. He had no further recurrence of bleeding and was discharged from the hospital on Sept. 5, 1950. He was seen six weeks later, at which time he complained only of mild epigastric discomfort and had had no further episodes of bleeding.

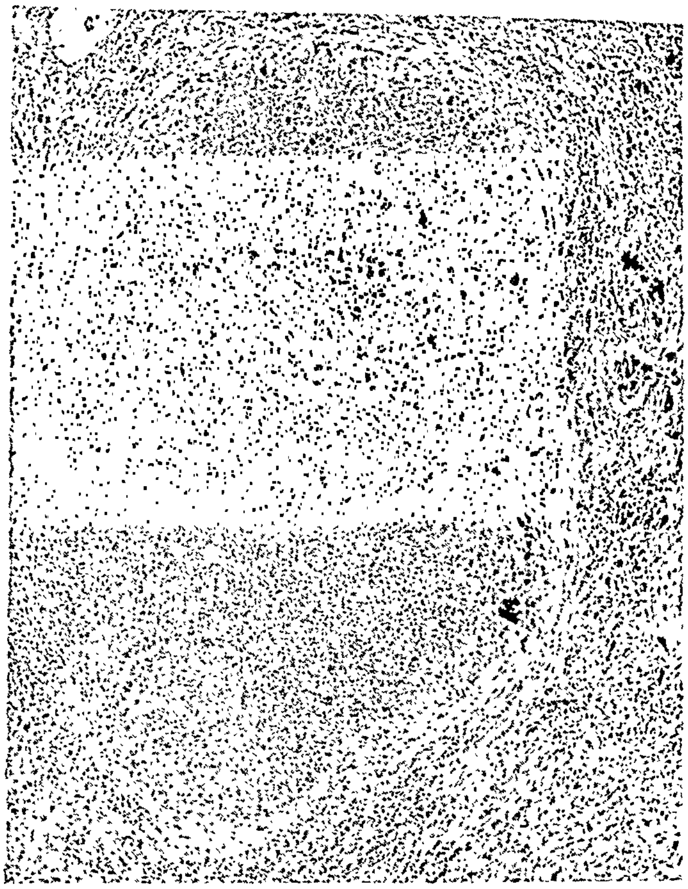

Fig. 1.—Photomicrograph of liver tissue at necropsy showing hemorrhagic infarction of cirrhotic lobule.

Second Hospitalization.—The patient was readmitted to the hospital on March 13, 1951, because of epigastric distress and hematemesis. He stated that he had had no form of treatment after his discharge from the hospital and had had frequent burning pain in the upper abdomen. He had an episode of severe bleeding in December, 1950, and again just prior to admission. Physical examination revealed spider nevi, enlargement of the liver 2 to 3 cm. below the costal margin, moderate epigastric tenderness, and gross evidence of anemia. The patient's blood pressure was 125/85 mm. Hg. An x-ray examination made several days after admission revealed a high degree of spasticity and some deformity of the duodenum. Results of laboratory tests made at this time were as follows: red blood cell count 2,200,000 to 4,000,000; total nonprotein nitrogen content of blood 54 mg. per 100 cc.; prothrombin 58 to 96% of normal; blood serum contained 1.6 mg. of bilirubin, 6.8 gm. of protein, and 3 gm. of albumin per 100 cc.; reaction to the cephalin-cholesterol flocculation test was 4+ and to the thymol flocculation test was

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From the Veterans Administration Hospital, Indianapolis.

[1] Reinhoff, W. F., Jr.: Ligation of the Hepatic and Splenic Arteries in the Treatment of Portal Hypertension with a Report of 6 Cases: Preliminary Report, Bull. Johns Hopkins Hosp. 88: 368-375, 1951.

[2] Berman, J. K.; Koenig, H., and Muller, L. P.: Ligation of Hepatic and Splenic Arteries in the Treatment of Portal Hypertension: Ligation in Atrophic Cirrhosis of the Liver, A. M. A. Arch. Surg. 63: 379 (Sept.) 1951.

[3] Markowitz, J.; Rappaport, A., and Scott, A. C.: The Function of the Hepatic Artery in the Dog, Am. J. Digest. Dis. 16: 344-348, 1949.

[4] Grindlay, J. H.; Mann, F. C., and Bollman, J. L.: Effect of Occlusion of the Arterial Blood Supply to the Normal Liver, A. M. A. Arch. Surg. 62: 806-811 (June) 1951.

negative; a zinc turbidity test showed 24 units; reaction to the colloidal red test was 4+, to the galactose tolerance test was normal, and to a serologic test for syphilis was negative. Esophagoscopic examination again revealed the presence of large, thin-walled varices in the lower esophagus, which had undergone little change since the previous examination.

Because of the recurrent hemorrhages, it was felt that the patient should have surgical treatment, although it had not been determined whether the bleeding was occurring from the varices or the duodenal ulcer. The operation was done April 16, 1951, with the patient under intratracheal ether anesthesia, which lasted four hours; 2,500 cc. of blood was given during the operation. Exploration revealed considerable nodular cirrhosis and some enlargement of the liver. The spleen was about three times normal size and had no unusual adhesions to the diaphragm. There were some moderately dilated veins about the upper stomach. A great many adhesions were present about the free edge of the lesser omentum, making dissection of this area difficult and bloody. A large, posterior, penetrating duodenal ulcer, which was not actively bleeding, was present about 2 cm. from the pylorus. Measurements of portal pressure were taken in a large gastroepiploic vein, using the second lumbar vertebra as a base line; the pressure thus obtained was 430 mm. of water. Temporary occlusion of the portal vein resulted in no change in this pressure; with temporary occlusion of the hepatic artery, a fall of 10 to 20 mm. of water was observed.

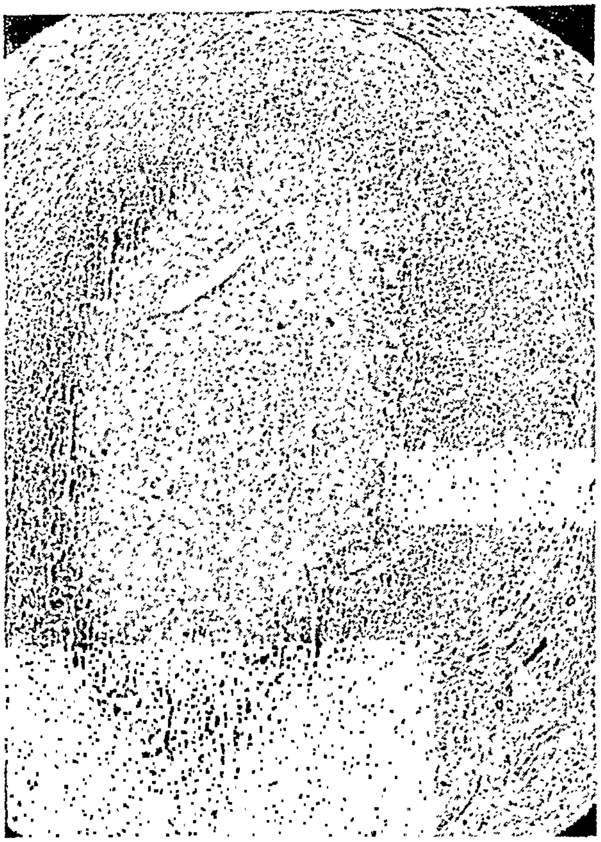

Fig. 2.—Photomicrograph of liver tissue at necropsy showing ischemic infarction of cirrhotic lobule.

It was felt that the source of the bleeding had not been determined with certainty and that prevention of further bleeding from both the duodenal ulcer and the varices should be provided. Accordingly, vagotomy and partial gastric resection were done, and, in addition, the hepatic artery was ligated just proximal to a large gastroduodenal branch. During the operation the patient's condition was fair, although his blood pressure for a long period of time was about 80/60 mm. Hg. The patient's tendency to bleed seemed to increase, and considerbale oozing was encountered, even during wound closure.

The immediate postoperative course was satisfactory. On the evening following the operation the patient's blood pressure fell, and there was considerable oozing of blood from the wound. He was given supportive treatment, with further blood transfusion, and his blood pressure rose to a normal level. On the day after the operation, he became progressively drowsier and more lethargic. In spite of administration of large amounts of vitamin K, the prothrombin level dropped to 23% of normal. The red blood cell count at this time was 4,200,000, and the serum bilirubin content was 9.2 mg. per 100 cc. The patient was treated with plasma, whole blood, vitamin K, human serum albumin, cortical extract, and cortisone, but his course was progressively downhill, with increasing jaundice and lethargy and rising temperature. He died on the second postoperative day.

Necropsy.—Necropsy was performed eight hours after death. The skin and scleras were highly jaundiced. No evidence of hemorrhage was found in the abdominal wound. There was almost 400 cc. of bloody fluid in the peritoneal cavity, and a fibrinous exudate was present over the surface of the liver and greater omentum. The lungs showed beginning bronchopneumonia and minimal edema. No definite esophageal varices could be found. The spleen was enlarged, weighing 450 gm., and appeared to be congested. The liver was enlarged, weighing 2,360 gm., and was extremely cirrhotic, with nodules of varying sizes over the surface and throughout the cut sections. Gross hemorrhage was apparent in most of these regenerated cirrhotic nodules, although occasional lobules presented a yellowish, caseous appearance, with peripheral zones of hemorrhagic discoloration. The portal vein was normal in appearance, and its lumen contained no thrombotic material. The hepatic artery was completely ligated immediately proximal to the gastroduodenal branch, and had no thrombosis, either distal to or proximal to the ligature.

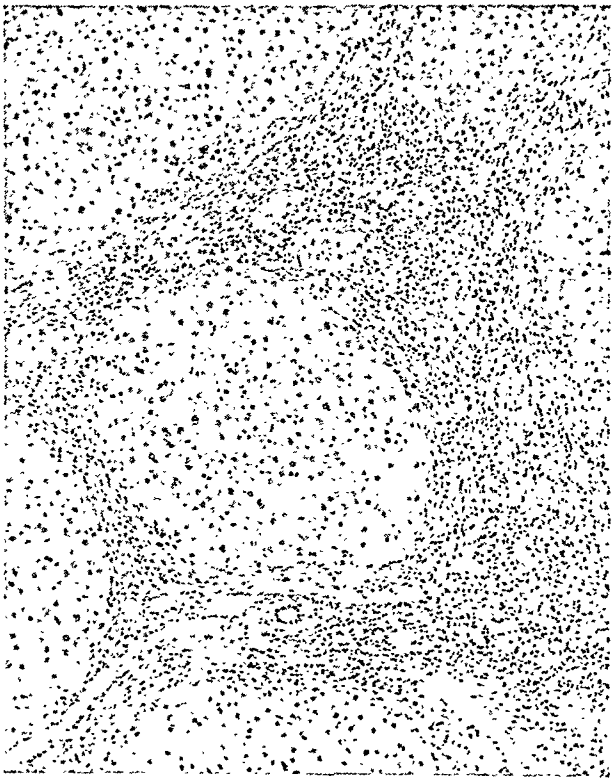

Fig. 3.—Photomicrograph of section of liver taken for biopsy during the operation for ligation of the hepatic artery showing typical portal cirrhosis with viable hepatic parenchyma.

Important microscopic changes were observed in the liver. The underlying condition was typical portal cirrhosis. The architecture was completely disrupted by broad and narrow bands of fibrous connective tissue, in which there were numerous lymphocytes and proliferated bile ducts. These interlacing bands enclosed and rather sharply demarcated lobules of regenerated hepatic parenchyma of varying sizes. In addition, recent superimposed changes of two general types were observed in the regenerated lobules. Most of the lobules were hemorrhagic, with

EXACERBATION OF TUBERCULOSIS DURING TREATMENT WITH CORTISONE

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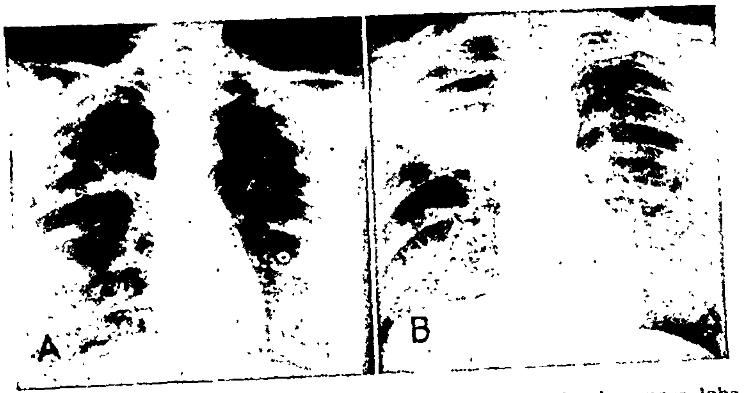
Observations of the unfavorable effects of corticotropin (ACTH) and cortisone on tuberculosis are multiplying. Guinea pigs and rats taking these drugs have succumbed to doses of tubercle bacilli found nonlethal in controls.[1] Pulmonary tuberculosis in humans has also disseminated during steroid administration.[2]

REPORT OF A CASE

S. M., a 37-year-old Negro woman, was seen in the arthritis clinic of Cook County Hospital in July, 1950, because of rheumatoid arthritis of five years' duration. The arthritis, involving all four extremities, was so severe that she was barely able to walk with help. The laboratory tests revealed the presence of hypochromic anemia and an accelerated erythrocyte sedimentation rate, findings commonly encountered in severe rheumatoid arthritis. She had no symptoms referable to the respiratory tract nor abnormal physical findings in the lungs. During case-finding studies she had previously had x-ray studies of her lungs. During two admissions to another hospital for treatment of the arthritis, routine roentgenograms had been made. She had never been told that these indicated pulmonary disease. She had never suspected any lung abnormality until, following discharge from Cook County Hospital in June, 1950, a visiting nurse had advised her to have x-ray studies of her lungs each six months. No reason for this advice was advanced.



A, roentgenogram of the chest showing infiltration in the upper lobe of the right lung before cortisone therapy in May, 1950. *B*, roentgenogram showing marked spread of the lesion during cortisone therapy in January, 1951.

On admission to Cook County Hospital in September, 1950, she was give cortisone (cortone® [Merck]) orally in doses of 300 mg. the first day, 200 mg. the second day, and 100 mg. daily thereafter. Within 24 hours there was a marked decrease in all the symptoms and signs of arthritis. Her appetite, strength and spirits rose correspondingly. Her weight increased 2 lb. (0.9 kg.) within four weeks. There was no demonstrable edema.

Symptoms of a "cold" developed in the 12th week of cortisone therapy. Cough, fever, and expectoration persisted. The patient complained of night sweats. Tubercle bacilli were found in the sputum, and marked extension of the tuberculosis was demonstrated roentgenologically (figure).

Administration of cortisone was discontinued, and the arthritis was temporarily much worse. Subsequently and concurrently with the bed rest enforced because of the tuberculosis, remission of the arthritis has occurred. The roentgenologic evidences of tuberculosis remain the same. The temperature is normal, and there is no cough nor production of sputum.

I have other patients with arrested pulmonary tuberculosis who are receiving cortisone for crippling rheumatoid arthritis without any apparent increase in their pulmonary infection.

CONCLUSION

Patients who are to receive corticotropin (ACTH) or cortisone should be examined for tuberculosis, including by roentgenologic studies. Any findings that suggest the presence of tuberculosis should be carefully and frequently reviewed for possible activation during treatment with corticotropin or cortisone. The presence of pulmonary tuberculosis may contraindicate the use of corticotropin or cortisone.

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[1] (*a*) Michael, M., Jr.; Cummings, M. M., and Bloom, W. L.: Course of Experimental Tuberculosis in the Albino Rat as Influenced by Cortisone, Proc. Soc. Exper. Biol. & Med. **75**: 613 (Nov.) 1950. (*b*) Freeman, S.; Fershing, J.; Wang, C. C., and Smith, L. C.: Effect of ACTH on Patients with Pulmonary Tuberculosis in Clinical ACTH Conference, Proceedings of the First Meeting, edited by J. R. Mote, Philadelphia, The Blakiston Company, 1950, p. 509.

[2] King, E. Q.; Johnson, J. B.; Batten, G. S., and Henry, W. L.: Clinical Notes: Tuberculosis Following Cortisone Therapy: Report of a Case of Rapidly Progressive Pulmonary Tuberculosis Following Cortisone Therapy for Rheumatoid Arthritis, J. A. M. A. **147**: 238 (Sept. 15) 1951. Popp, C. G.; Ottosen, P., and Brasher, C. A.: Cortisone and Pulmonary Tuberculosis, ibid. **147**: 241 (Sept. 15) 1951. Fred, L.; Levin, M. H.; Rivo, J. B., and Barrett, T. F.: Development of Active Pulmonary Tuberculosis During ACTH and Cortisone Therapy, ibid. **147**: 242 (Sept. 15) 1951. Freeman and others.[1b]

TESTICULAR ANDROGEN DEFICIENCY AND SEMEN VOLUME

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and
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The assessment of androgenic activity in male subjects by simple and easily accessible methods is highly desirable. Tests such as the determination of 17-ketosteroids and urinary gonadotropins are not readily available to the general practitioner. Also, the proper clinical evaluation of the secondary sex characteristics (e. g., frontotemporal recession of the hairline, the beard, and pubic and axillary hair) requires familiarity with these signs, and they have the disadvantage of not relating directly to the levels of androgens circulating at the time of examination. We wish, therefore, to call attention to the use of semen volume measurements as an index of androgenic function.

There are, as Huggins[1] pointed out, two different types of male secondary sex characteristics: the self-maintaining (such as beard and voice pitch), which once developed will not atrophy in the absence of androgen, and those depending strictly on a constant presence of this hormone for the maintenance of their anatomic and functional integrity. In the latter category are the sex accessories, such as prostate and seminal vesicles. From this it follows that any serious decrease in the amount of circulating androgen will be reflected in the sex accessories and, therefore, in semen.

Semen is composed of two parts: (1) the spermatozoa, produced by the seminiferous tubules, and (2) the plasma, produced by the sex accessories where prostate and seminal vesicles contribute the largest portion. When there is failure of the seminiferous tubules (or

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From the Cleveland Clinic and the Frank E. Bunts Educational Institute.

[1] Huggins, C.: Prostatic Secretion, Harvey Lect. (1946-1947) **42**: 148, 1947.

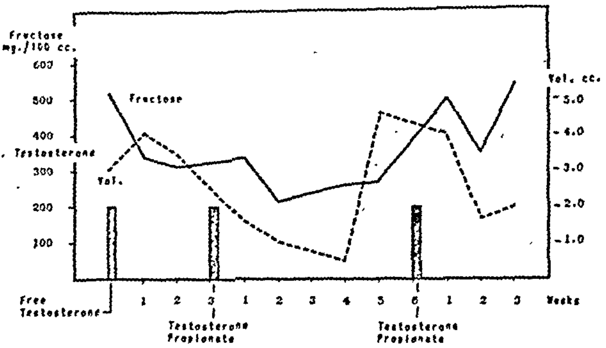
mechanical obstruction distad to them) there may be oligospermia or even azoospermia. However, if Leydig cell function is preserved, as is often the case, semen volume is normal, since the sex accessories are receiving an adequate androgen supply.

There are in plasma a number of chemical substances, characteristic of different portions of the sex accessories, the concentration of which depends on the amount of circulating androgen. In man, the enzyme "acid" phosphatase is elaborated in the prostate [2] and a sugar, fructose, in the seminal vesicles. Both of these substances have been variously utilized as an index of androgenic function.[3] Fructose concentration parallels semen volume up to amounts of 4 to 5 cc. Above this its concentration decreases.[3c] In the accompanying table we have plotted the average fructose concentration in milligrams per 100 cc. of semen as related to the volume of several specimens obtained from patients with hypogonadism treated with testosterone preparations. It will be noted that fructose concentrations increase proportionately with semen volume although the range of variation is great. In some men fructose concentration shows an inverse relationship to semen volume. This is exemplified in the accompanying figure.

Recently Landau and Loughead [4] utilized fructose measurements as an index of androgen activity in eunuchoid or normal persons. In our hands fructose determinations have proved to be no more valid indexes of androgenicity than the semen volume alone. The reasons for this are that the normal individual variations are large (from about 200 mg. per 100 cc. to 800 mg. per 100 cc.) and that there are no significant changes in fructose concentration in the absence of changes in semen volume. In severe, untreated eunuchoidism, the subject is incapable of erection or ejaculation or the semen volume is extremely low (0.1 cc. or less). When androgen is administered the volume gradually increases until it reaches a normal level. Semen specimens should be obtained after four days of continence; in these conditions normal semen volumes vary from 2 to 7 cc. Volumes of more than 3 cc. on repeated examinations and especially volumes over 5 cc. can be considered good evidence that there is no deficiency of circulating androgen. Amounts of less than 2 cc. on repeated occasions raise the suspicion of androgen deficiency. If this has developed in a fully matured person, other signs may not be evident. A more thorough endocrine investigation is then warranted.

Relationship of Fructose Concentration to Semen Volume in Azoospermic, Eunuchoid Patients Treated with Testosterone

No. of Patients	No. of Specimens	Vol. (Cc.)	Fructose Concentration, Mg. per 100 Cc.	Range
6	23	1.0 or less	192	45-560
5	31	1.1-2.0	331.8	170-585
7	21	2.1-3.0	427.1	155-615
6	27	3.1-4.0	431.2	215-655
4	13	4.1-5.0	529.2	270-740



Cryptorchism with eunuchoidism: effect of testosterone propionate therapy on semen volume and semen fructose concentration.

SUMMARY

Attention is called to the fact that in clinical practice the simple procedure of measurement of semen volume may be used to advantage as a screening test for hypogonadism insofar as it relates to testicular androgen production.

2020 E. 93d St.

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[2] Huggins.[1] Mann, T., and Lutwak-Mann, C.: Secretory Function of Male Accessory Organs of Reproduction in Mammals, Physiol. Rev. **31**: 27 (Jan.) 1951.

[3] (*a*) Clark, L. C., Jr.; Beck, E. I., and Thompson, H.: Excretion of Acid Phosphatase as Measure of Prostatic Development During Pubescence, J. Clin. Endocrinol. **11**: 84 (Jan.) 1951. (*b*) Engberg, H.; Andersson, E.; Sury, B., and Raft, J.: Possibility of Determining Androgen Production by Measuring Acid Phosphatase in Semen: Investigations in Cryptorchid Patients, ibid. **5**: 42 (Feb.) 1947. (*c*) Harvey, C.: Relation Between Volume and Fructose Content of Human Semen, Nature **162**: 812 (Nov. 20) 1948. (*d*) Lundquist, F.: Aspects of Biochemistry of Human Semen, Acta physiol. scandinav. (supp. 66) **19**: 7, 1949.

[4] Landau, R. L., and Loughead, R.: Seminal Activity in Man, J. Clin. Endocrinol. **11**: 1411 (Dec.) 1951.

RECURRENT NATURE OF ACUTE BENIGN PERICARDITIS

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R. Bruce Logue, M.D.
and
J. Willis Hurst, M.D., Emory University, Ga.

Despite the extensive literature relating to acute benign pericarditis and the observation of recurrent attacks by various writers,[1] the high incidence and the frequency of recurrences in this illness have not been stressed. It is the purpose of this paper to emphasize this point by presenting a detailed report of one patient and summarized data for eight patients, in whom such episodes of acute benign pericarditis occurred (see the accompanying table). The following case, in which the largest number of recurrences was observed, is presented as an example of this group.

From the Department of Cardiology, Emory University School of Medicine.

[1] (*a*) Burchell, H. B.: Acute Nonspecific Pericarditis, Mod. Concepts Cardiovas. Dis. **16**: [*n.p.*] (March) 1947. (*b*) Logue, R. B., and Wendkos, M. H. Acute Pericarditis of Benign Type, Am. Heart J. **36**: 587-599 (Oct.) 1948. (*c*) Barnes, A. R., and Burchell, H. B.: Acute Pericarditis Simulating Acute Coronary Occlusion, ibid. **23**: 247-268 (Feb.) 1942. (*d*) Wolff, L.: Acute Pericarditis Simulating Myocardial Infarction, New England J. Med. **230**: 422-425 (April 6) 1944. (*e*) Massey, F. C.: Acute Pericarditis Simulating Myocardial Infarction, Northwest Med. **46**: 455-459 (June) 1947. (*f*) Feder, I. A.; Hoffman, J., and Sugar, H.: Acute Primary Pericarditis, Am. J. M. Sc. **220**: 144-155 (Aug.) 1950. (*g*) Evans, E.: Acute Nonspecific Benign Pericarditis, J. A. M. A. **143**: 954-957 (July 15) 1950. (*h*) Carmichael, D. B.; Sprague, H. B.; Wyman, S. M., and Bland, E. F.: Acute Nonspecific Pericarditis: Clinical, Laboratory, and Follow-up Consideration, Circulation **3**: 31-331 (March) 1951. (*i*) Levy, R. L., and Patterson, M. C.: Acute Serofibrinous Pericarditis of Undetermined Cause: Study of 27 Cases, Am. J. Med. **8**: 34-45 (Jan.) 1950.

REPORT OF A CASE

F. J. G., a 27-year-old white woman, a registered nurse, was admitted to the hospital on July 21, 1950, with the chief complaint of sudden onset of severe substernal and precordial pain, accentuated by inspiration and by lying in the supine position but not increased by swallowing. She had her first attack of such pain at the age of 6 years; 19 subsequent attacks occurred. The longest interval between recurrences was four years. Occasionally respiratory infection preceded an attack. Results of physical examination were normal except for temperature of 100.4 F. The cardiac rhythm was regular. There was no murmur, rub, or gallop rhythm. Examination of the lungs revealed no abnormality. There was no mediastinal crepitation.

The sedimentation rate was 24 mm. per hour (Westergren). The white blood cell count was 10,650 per cubic millimeter, with a normal differential count. Roentgenograms of the gallbladder, gastrointestinal tract, cervical and thoracic spine, and chest were normal. Results of fluoroscopic examination of the chest were normal. The electrocardiogram revealed shortening of the T vector in the frontal plane with isoelectric T waves in V_3 and V_4. The patient had low-grade fever for two days. The pain gradually disappeared, and she was asymptomatic when she was discharged, on the fourth hospital day.

One month later the same symptoms reappeared. Physical examination revealed a faint pericardial friction rub in the fourth anterior intercostal space in the midclavicular line. Her temperature was 99.2 F. An electrocardiogram taken five hours after the onset of the pain revealed marked lowering of the T waves in all leads. The use of narcotics was necessary for the relief of the pain, and she was asymptomatic in six days. The patient was subsequently hospitalized elsewhere on two occasions because of recurrence of the same symptoms.

Summary of Data

Patient	Age, Yr.	Sex	Antecedent Infection	No. of Recurrences	Chill	Character of Pain
J. H.	31	M	Measles 3 weeks prior to first attack	3 (1) 1942 (2) 1948 (3) 1949	....	Severe substernal, radiating to shoulder, lasting 10 days, not relieved by glyceryl trinitrate therapy, aggravated by deep breathing, movement, and walking
H. W.	33	M	Acute sinusitis 10 days prior to first attack	4 (1) 1948 (2) 1948 (3) 1949 (4) 1950	Yes	Epigastric and substernal, stabbing, radiating to neck, arms, fingers, aggravated by movement, swallowing, breathing; patient acutely ill
R. I.	25	M	None	9 between Nov., 1950, and May, 1951	No	Dysphagia, pain radiating from pharynx to left shoulder
F. J. G.	27	F	Occasional infection	19 since age of 6 yr.	No	Sharp, substernal and precordial pain, aggravated by cough, movement, and respiration
M. M. B.	45	F	None	8 between 1945 and 1949	Yes	Intermittent sharp, midsternal pain, radiating to arms; constant dull ache; patient acutely ill; sat upright
W. F. G.	50	M	None	2	Yes	Sudden, severe substernal, nonradiating pain aggravated by respiration, movement, and swallowing; acutely ill; intravenous use of morphine required
Case No. 14 [1b]	25	M	None	History of attack 1 yr. previously	....	Sharp precordial pain, aggravated by inspiration, coughing, and turning
Case No. 15 [1b]	46	M	Upper respiratory infection for 1 mo.	4	No	Pain in left shoulder, anterior chest, neck, increased by movement, cough, and sneezing

* Rourke-Ernstene method.

COMMENT

The symptoms, physical findings, laboratory data, and the electrocardiographic changes have been discussed elsewhere.[2] The pain is usually substernal or precordial, and from the onset the striking characteristic of the pain is its close relationship to inspiration, bodily movements, cough, and sometimes swallowing. Occasionally the patient may be acutely ill and may present the picture of peripheral collapse. Frequent examinations may be necessary for the detection of the pericardial friction rub. Cardiac dilatation is believed by some to occur,[3] but the increase in the cardiac silhouette is more likely due to pericardial effusion, which is probably present in small amounts in most cases.[4] Pleural effusion may at times be noted.

In acute benign pericarditis an injury current due to diffuse subepicardial myocarditis is reflected on the electrocardiogram as concave S-T segment elevation in one or more leads.[1b] It is important to differentiate these changes from the persistent S-T elevation seen occasionally in normal subjects having T vectors of large magnitude, with early forces of repolarization giving rise to S-T elevation. The T waves may be tall and peaked at the onset, but after a short time they are flattened and later may become inverted. The T-wave changes may be

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2. (*a*) Burchell.[1a] Wolff.[1d] Carmichael and others.[1h] (*b*) Nay, R. M., and Boyer, N. H.: Acute Pericarditis in Young Adults, Am. Heart J. **32**: 222-233 (Aug.) 1946.

3. (*a*) Barnes and Burchell.[1c] Levy and Patterson.[1i] (*b*) Wolff, L.: Acute Pericarditis with Special Reference to Changes in Heart Size, New England J. Med. **229**: 423-431 (Sept. 9) 1943.

4. (*a*) Porter, W. B.; Clark, O., and Porter, R. R.: Nonspecific Benign Pericarditis, J. A. M. A. **144**: 749-753 (Oct. 28) 1950. (*b*) Nathan, D. A., and Dathe, R. A.: Pericarditis with Effusion Following Infections of the Upper Respiratory Tract, Am. Heart J. **31**: 115-130 (Feb.) 1946.

the only electrocardiographic abnormality present. QRS abnormalities do not occur.

Of most importance in the differential diagnosis is myocardial infarction. The early appearance of the pain of pericarditis and the fever and pericardial friction rub without electrocardiographic changes of myocardial infarction help establish the diagnosis of acute benign pericarditis. Tuberculous pericarditis is more often painless, and the onset is usually gradual, associated with antecedent weakness, night sweats, fever, and weight loss. The pericarditis of rheumatic fever is almost always associated with signs of myocarditis, which are not seen in patients with acute benign pericarditis. Nay and Boyer have stressed the importance of coincident joint manifestations and leukocytosis in acute pericarditis due to rheumatic fever.[2b]

in Eight Cases

		Maximum				Electrocardiographic Changes	
Friction Rub	Pulse Rate	Temperature (F)	White Blood Cell Count	Sedimentation Rate	Chest Roentgenographic Observations	*S-T Segment*	*T Wave*
History of rub with 1st attack	70	Low-grade fever for 2 days	......	..	....	Elevated in all leads	Inverted in leads 1, V_5, and V_6
With first attack only	70-100	100.8	11,800	52	Small amount of fluid in costophrenic angles; cardiac silhouette normal	Elevated in leads 1 and 2, depressed in 3	....
Present	100	.....	8,000	15	Cardiac enlargement; infiltration in left base, with fluid	....	....
Faint during one attack	100	100.4	10,650	24	Normal	....	Very low in all leads
....	100	103	12,600	4	Small amount of fluid in left costophrenic angle	Slightly elevated in leads 2 and 3	Inverted in lead 1; isoelectric in 2 and 3
Yes	84	102.2	19,100	28	Normal except for slight cardiac enlargement	Elevated in leads 1, 2, 3, and V_4-V_6	Flattened in lead 1 and in left precordial leads
Yes	...	100.2	......	0.8 *	....	Elevated in leads 1, 2, and 3	Inverted in leads 1, 2, 3, and CF_4
Yes	108	.....	10,150	1.2 *	Obliteration of left costophrenic angle; apical pleural thickening	Elevated in leads 2, 3, and CF_4	Flattened in leads 1, 2, and 3; later inverted in 1, 2, and CF_4

The cause of acute benign pericarditis remains obscure. In some cases it is possible to elicit a history of preceding infection, usually in the upper respiratory tract.[5] Acute benign pericarditis is sometimes seen in association with atypical pneumonia.[6] Barnes and Burchell stated the belief that occasionally a young adult may have a benign, apparently self-limited form of pericarditis, which may possibly have been due to tuberculosis.[1c] Nathan and Dathe suggested a hypersensitive response to an offending organism as a possible causative factor.[4b] It would seem more likely that the disease is due to a virus infection. If there is an immunologic response on the part of the host, it would appear to be a transitory one, in view of the repeated attacks in some patients.

The present treatment is symptomatic. Bed rest is recommended for one week, with gradual increase in activity over a period of two to three weeks. Various antibiotics,[7] chemotherapeutic agents,[6b] and antihistaminic drugs [8] have been used in the treatment of the disease, but their evaluation is difficult because the natural course of the illness is toward recovery. Taubenhaus and Brams expressed the opinion that the improvement noted in their three patients treated with aureomycin was prompt and impressive enough to warrant its further use.[9] One of our patients received aureomycin for five days during a recurrence, and another received terramycin for seven days. The pain subsided coincidentally with the administration of these antibiotics, but we do not believe that it is possible to attach any significance to this. During one acute attack the patient described above received 50 mg. of corticotropin (ACTH) without any relief of the pain.

Although further study and observation of cases will be needed before definite conclusions can be drawn, it is generally believed that the prognosis for patients with acute benign pericarditis is good and that constrictive pericarditis does not occur. Of a series of 27 patients with acute benign pericarditis described by Levy and Patterson, 23 were reexamined at intervals of 6 months to 16 years following the acute illness.[11] All showed complete recovery without sequelae. Barnes and Burchell followed 14 patients, none of whom has shown residual damage from previous pericarditis.[10] The patient described above, after having experienced 19 attacks of acute benign pericarditis over a period of 19 years, showed no evidence of residual damage at the time of last examination.

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[5] Barnes and Burchell.[1c] Evans.[2c] Levy and Patterson.[3] Porter, Clark, and Porter.[4a] Nathan and Dathe.[4b] Willius, F. A.: Cardiac Clinics: Clinic on Acute Serofibrinous Pericarditis Secondary to Acute Pharyngitis: Comment, Treatment, Course, Proc. Staff Meet., Mayo Clin. **9**: 637-640 (Oct. 17) 1934.

[6] (*a*) Wolff.[3a] Wolff.[2b] (*b*) Finkelstein, D., and Klainer, M. J.: Pericarditis Associated with Primary Atypical Pneumonia, Am. Heart J. **28**: 385-394 (Sept.) 1944.

[7] Evans.[2c] Porter, Clark, and Porter.[4a]

[8] Zivitz, N., and Oshlag, J. A.: Eosinophilic Pleural Effusion and Pericarditis with Effusion in Allergic Subject, J. Allergy **20**: 136-143 (March) 1949.

[9] Taubenhaus, M., and Brams, W. A.: Treatment of Acute Nonspecific Pericarditis with Aureomycin, J. A. M. A. **142**: 973-975 (April 1) 1950.

SUMMARY

The recurrent nature of acute benign pericarditis is emphasized. A report of a case and summarized data in a series of eight cases of the disease, in which the number of recurrences ranged from 2 to 19, are presented. The clinical aspects of the disease are briefly reviewed.

[10] Footnote 1 *a* and *c*.

SEGMENTAL FORCEPS

Richard H. Overholt, M.D., Boston

A special forceps has been designed for use on the uncovered surface and edge of pulmonary segments. The new segmental forceps has a narrow bite with fine-tooth edges. The shafts are flared above the tip, which automatically throws the loop of thread down over its end and into lung tissue as the knot is tightened. The possibility of including the tip of the instrument in the ligature is eliminated (fig. 1).

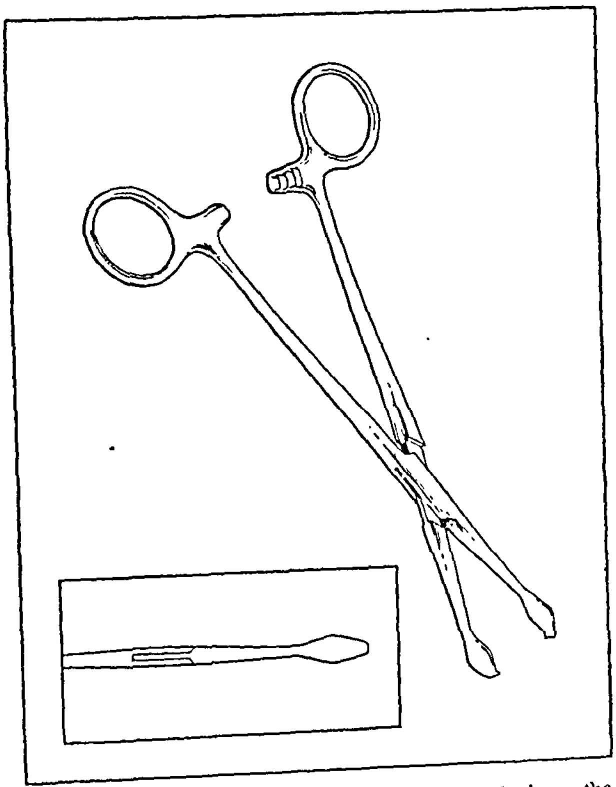

Fig. 1.—Segmental forceps, showing the shafts flared above the tips.

In sealing air leaks on segmental surfaces devoid of pleura, there is a need to grasp small amounts of tissue about the point of escape of air and to set a circumferential ligature beyond the point of the instrument (fig. 2*B*). The lung must be expanded to prove air tightness, and leaks should be sealed with segments in their normal state of inflation. If a leak is present, needle suturing produces more leaks. Ordinary hemostats, either straight or curved, gather up too much tissue, thus tending to reduce the volume of the segment and deform its surface.

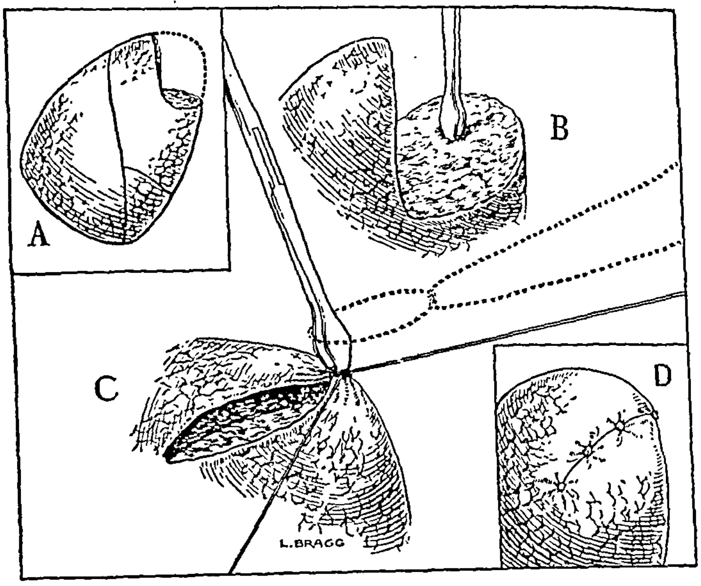

Fig. 2.—Uses of segmental forceps. *A*, apical segments after enucleation. *B*, forceps grasping small amounts of tissue to seal air leaks. *C*, segments grasped by forceps, showing ligature in position. *D*, segments anchored with ligatures.

The segmental forceps has a second use; namely, the approximation of the edges of segments or lobes. Whenever possible, it is desirable to bring two denuded segmental surfaces together; for example, after enucleation of an apical segment (fig. 2*A*), the surfaces of the anterior and posterior segments can be brought together by fastening edges at three or four points. This should be done with the segments fully inflated (fig. 2*C* and *D*).

It is also desirable to anchor a single remaining segment of one lobe to the adjacent intact lobe. This can be done by a slight shift in its position with abutment of the denuded surface of the segment against the interlobar surface of the intact lobe. This prevents rotation of the single segment, aids in sealing small air leaks, and minimizes the formation of adhesions between lung and chest wall. The fastening of segment to lobe must be done when they are inflated. The segmental forceps grasps the edge of each at three or four sites, and the tissue included in each bite is anchored with a ligature, which slips over the end of the instrument.

1101 Beacon St. (Brookline, Mass.).

The forceps was manufactured by Codman & Shurtleff, Inc., 104 Brookline Ave., Boston.

From the Department of Surgery, Tufts College Medical School, Boston. Member of the staff of New England Deaconess Hospital and New England Center Hospital, Boston.

Brain Growth.—The human skull yields to the normal outward push of the growing brain during infancy and earliest childhood. At this period, therefore, brain injury or brain destruction results in overlying cranial smallness. The rate of growth is astonishingly rapid at first for the brain increases from an average weight of 300 gm. at birth to 500 gm. at six months. At two years the weight may normally reach 900 gm., and at puberty 1,200 gm., after which there is ordinarily an increase of no more than 100 gm.—Wilder Penfield, M.D., Cranial Clues to Intracranial Abnormality, *The American Journal of Roentgenology*, April, 1952.

THROMBOCYTOPENIC PURPURA INDUCED BY SULFISOXAZOLE (GANTRISIN®) THERAPY

REPORT OF A CASE CONTROLLED BY PLATELET TRANSFUSION

Jacob Geiger, M.D., New York

Sulfisoxazole (gantrisin®), 3,4-dimethyl-5-sulfanilamido-isoxazole, introduced in 1946, is widely used in treatment of bacterial infections, especially those of the urinary tract. The drug is said to be one of the safest of the sulfonamides. It is described by the manufacturer as a "single drug—not a mixture of several sulfonamides—so that there is less likelihood of sensitization" and as "having greater solubility and higher blood-levels." As is the case with other members of the sulfonamide group, however, reports of toxic reactions have appeared in the literature. Aside from reports of nausea, vomiting, headache, fever, and dermatitis, a number of instances of toxic effects on the bone marrow have been reported. Haunz, Cardy, and Graham [1] collected eight cases of leukopenia following the use of gantrisin,® and recently Hagerman and Franzblau [2] added another. A careful search of the literature for cases of hemorrhagic manifestations of toxicity has yielded one report [3]—that of a case of petechial hemorrhages following the administration of gantrisin,® 75.5 gm. given over a period of 12 days. In this instance there was moderate leukopenia but no thrombocytopenia. The present case is of interest because of the occurrence of toxic thrombocytopenia with its sequelae and the dramatic response following cessation of treatment with the drug and one transfusion of blood platelets.

Results of Blood Studies from Onset of Thrombocytopenic Purpura *

		Dec. 4		Dec. 5 †					
Date	Nov. 23	3 P.M.	4 P.M.	10 A.M.	5:30 P.M.	Dec. 6 10 A.M.	Dec. 7 10 A.M.	Dec. 10 10 A.M.	Dec. 11 10 A.M.
Hemoglobin, gm.	15.8	12.8	Trans-	12.8	......	11.0	10.2	11.6	......
Red blood cells, millions	5	4.2	fusion,	4.48	......	3.33	3.43	3.96	......
White blood cells	10,400	12,990	500 cc.	19,800	......	18,000	11,000	11,700	......
Platelets	......	20,000	whole	40,800	51,000	61,800	85,500	210,000	218,500
Bleedin	......	......	blood	9	1½	2½	2½	1	......
Heparin	......	......	......	31	19	21	21	18	......
Serum	......	......	......	20	57	45	52	......	......
Tourniquet test	......	......	......	>100 petechiae	Negative	Negative	Negative	Negative	......
Clot retraction	......	......	......	Absent	Present	Present	Present	Present	......

* Sternal marrow biopsy (Dr. Carl Reich) on Dec. 4 showed a granular cell hyperplasia; megakaryocytes were diminished. On Dec. 10 the sternal marrow showed hyperplasia of all the elements.

† On Dec. 5, at 5 p.m., a transfusion of blood platelet concentrate (450 billion platelets) was given.

REPORT OF A CASE

P. C., a 69-year-old man, was admitted to the hospital on Nov. 25, 1951, with a history of difficulty in voiding for the past two years. During this period he had received hormone injections. The past history revealed an episode of precordial pain with syncope about 10 years previously but was otherwise irrelevant. Results of the physical examination were normal, except with respect to the prostate, which was firm, irregular and diffusely enlarged. X-ray examination of the lumbar portion of the spine and the pelvis showed no evidence of neoplastic metastasis. The preoperative diagnosis was prostatic obstruction possibly due to carcinoma. On Dec. 3, a bilateral vasectomy and transurethral prostatectomy were performed with the patient under general anesthesia. Histological examination showed a carcinoma of the prostate. The patient reacted well. Medication consisted of the administration of sedatives, when indicated, and gantrisin,® 1 gm. three times daily, given from Nov. 25 to Dec. 4 (a total of 21 gm.). Penicillin had been ordered but was not given because the patient stated he was allergic to it. On the morning of Dec. 4, the first postoperative day, edema and what appeared to be purpuric spots on both ankles and hematuria were noted. Within a few hours, when I saw the patient, the rash had become distinctly purpuric, extending over the lower and upper extremities. There was bleeding from both nostrils and the mouth, with severe hematuria and bloody stools. The diagnosis of toxic thrombocytopenia induced by the use of gantrisin® seemed justifiable, and treatment with the drug was discontinued. Hematological examinations and a sternal bone marrow biopsy were made while the patient was being prepared for transfusion. Five hundred cubic centimeters of whole blood (18 hours old) was administered an hour later. The condition remained unchanged for the next 24 hours. Bladder irrigation yielded large blood clots; bleeding from two areas on the inside of the cheeks and from the nose continued. In the meantime a platelet concentrate was prepared from 5 units of fresh blood.[4] At 5 p. m. on Dec. 5 the patient received a transfusion consisting of approximately 450 billion blood platelets suspended in 300 cc. of isotonic sodium chloride solution. Three hours later the bladder washings were considerably less bloody, yielding only a few small clots. Hematological findings indicated pronounced improvement, and it was the general feeling, both from a clinical and laboratory standpoint, that effective hemostasis had been established and that further hemotherapy was not indicated. On Dec. 7, the urine was grossly clear, the mouth lesions had not bled for two days, and there was gradual fading of the purpuric rash. On Dec. 10 the patient was out of bed. He was discharged on Dec. 12 in good condition.

COMMENT

It is reasonable to assume that the thrombocytopenic purpura was induced by treatment with gantrisin.® Administration of a large number of platelets brought about prompt arrest of bleeding, and cessation of treatment with the drug permitted spontaneous regeneration of the bone marrow. The platelet count did not show an immediate rise, but this was to be expected since the platelets are probably withdrawn from the circulation as fast as they are administered, in the process of restoring a normal blood-clotting mechanism.

SUMMARY

A case of toxic thrombocytopenic purpura induced by treatment with gantrisin® (3,4-dimethyl-5-sulfanilamido-isoxazole) is reported. Recovery occurred with dramatic suddenness after cessation of treatment with the drug and a transfusion of blood platelets.

1070 Park Ave.

From the Achelis Laboratory, Lenox Hill Hospital.

1. Haunz, E. A.; Cardy, J. D., and Graham, C. M.: Agranulocytosis Due to Gantrisin®: Report of a Case with Recovery, J. A. M. A. **144**: 1179 (Dec. 2) 1950.

2. Hagerman, D. D., and Franzblau, S. A.: Gantrisin® (Sulfisoxazole)-Induced Granulocytopenia, J. A. M. A. **147**: 1657 (Dec. 22) 1951.

3. Gale, G. L.: Petechial Hemorrhages with the Use of Gantrisin (NU-445), Canad. M. A. J. **64**: 252 (March) 1951.

4. The platelet concentrate was prepared in the Laboratory of Experimental Cytology, Lenox Hill Hospital, by the method of Minor and Burnett (to be published).

COUNCIL ON PHARMACY AND CHEMISTRY

REPORT TO THE COUNCIL

Some months ago in a random sampling 50 physicians in California were asked the question, "If a narcotics addict came to you for treatment, what disposition would you make of the case?"

Inasmuch as four of the physicians queried indicated that they would treat the addict, while the others gave varied answers, the Council, believing that physicians in general would appreciate a discussion of the question, has authorized publication of the following statement. This statement has been prepared by the Committee on Drug Addiction and Narcotics of the National Research Council with the assistance of Dr. Harris Isbell, Director, National Institute of Mental Health Addiction Research Center, United States Public Health Service Hospital, Lexington, Kentucky.

R. T. STORMONT, M.D., *Secretary.*

WHAT TO DO WITH A DRUG ADDICT

A recent questionnaire circulated in southern California revealed that many physicians were uncertain as to the proper course to be taken when drug addicts appeared in their offices. This paper has been prepared to provide information on the proper procedure in this situation. It will deal only with addiction to those substances covered by the Harrison Narcotic Act and similar laws, such as the Marihuana Tax Act. Specifically, the drugs concerned are opium and all mixtures containing opium, morphine, heroin, dihydromorphinone (dilaudid®), methyldihydromorphinone (metopon), 3-hydroxy-N-methylmorphinan (dromoran®), codeine, dihydrocodeinone (hycodan®), dihydrohydroxycodeinone (eukodal), meperidine (demerol®), methadone (dolophine,® adanon®), marihuana, and cocaine.

PERTINENT FEDERAL REGULATIONS

All physicians who prescribe and dispense narcotics are furnished by the Bureau of Narcotics, U. S. Treasury Department, with Regulations No. 5 relating to the "dealing in, dispensing and giving away of opium or cocoa leaves, isonipecaine or opiates, or any compound, manufacture, salt, derivative or preparation thereof" and with Pamphlet No. 56, "Prescribing and Dispensing of Narcotics under the Harrison Narcotic Law." All physicians should be familiar with these two publications and, furthermore, should realize that the synthetic narcotics, meperidine, methadone, and dromoran,® have been legally defined as opiates and subjected to the same regulations as are morphine and its derivatives. The most pertinent statement concerning addiction is Article 167 of Regulations No. 5, which reads in part as follows: "An order purporting to be a prescription issued to an addict or habitual user of narcotics not in the course of professional treatment, but for the purpose of providing the user with narcotics sufficient to keep him comfortable by maintaining his customary use, is not a prescription within the meaning and intent of the act; and the person filling such an order, as well as the person issuing it, may be charged with violation of the law."

This regulation is interpreted in Pamphlet No. 56, which reads in part as follows: "The responsibility for the proper prescribing and dispensing of narcotic drugs under the Harrison Narcotic Law rests upon the physician in charge in any given case . . . [and] corresponding responsibility rests upon the druggist who fills the prescription to determine, in good faith, that the prescription was issued in the course of professional practice and not for the purpose of gratifying addiction . . . The good faith of the physician and the bona fides of his treatment in a given case will be established by the facts and circumstances of the case and the consensus of medical opinion with regard thereto, based upon the experience of the medical profession in cases of similar nature. Physicians will be expected to exercise such care in every case where narcotic usage is indicated, that the patient under treatment shall receive no quantity of narcotic drug greater than that sufficient for bona fide medical needs in order that there may be no surplus available for possible diversion by the patient to illicit use . . . Mere addiction alone is not regarded or recognized as an incurable disease. It is well established that the ordinary case of addiction yields to proper treatment and that addicts can remain permanently cured when drug taking is stopped and they are otherwise physically restored to health and strengthened in will power."

Many of the individual states have passed narcotic control laws and the physician must be familiar with the laws and regulations of his particular state as well as with the Federal laws. In general, the physician will be acting in accordance with the consensus of medical opinion with regard to addiction and will be complying with the letter and spirit of the regulations if he follows two principles: (1) Ambulatory treatment of addiction should not be attempted as institutional treatment is always required; (2) Narcotic drugs should never be given to an addict for self-administration.

The reasons for establishing these principles are set forth in a committee report adopted by the House of Delegates of the American Medical Association in 1924. The following are excerpts from this report: "Your committee desires to place on record its firm conviction that any method of treatment for narcotic drug addiction, whether private, institutional, official or governmental, which permits the addicted person to dose himself with the habit-forming narcotic drugs placed in his hands for self-administration, is an unsatisfactory treatment of addiction, begets deception, extends the abuse of habit-forming narcotic drugs and causes an increase in crime . . .

"In the opinion of your committee, the only proper and scientific method of treating narcotic drug addiction is under such conditions of control of both the addict and the drug that any administration of any habit-forming narcotic drug must be by, or under the direct personal authority of the physician with no chance of any distribution of the drug of addiction to others, or opportunity for the same person to procure any of the drug from any source other than from the physician directly responsible for the addict's treatment."

DIAGNOSIS OF ADDICTION

Most frequently, addicts who appear in a physician's office are transients who are unknown to the physician. Such persons are likely to appear when circuses and carnivals are present in a community. Less commonly, addicts may be nontransient persons who are fairly well known to the physician. Frequently, nontransient addicts are neurotic persons or are patients who are known to have been chronic alcoholics. It is not unusual for the nontransient addict to be a physician. In recent years, it has not been unusual in certain areas for an adolescent boy or girl to be brought to the physician by relatives or by representatives of social organizations for advice relative to the treatment of addiction.

Most frequently, the diagnosis of addiction is made at the onset of the interview by the patient's statement that he is addicted to and needs drugs. The addict may attempt to conceal his addiction and may present a glib story of some physical illness; most frequently mentioned are atypical angina pectoris, kidney colic, migraine, or hemorrhoids. Generally, the story culminates with the suggestion that other physicians have found that the only adequate remedy is a prescription for narcotic drugs. Frequently, addicts of this type may appear armed with a formula which they state was given to them by another physician and which they have found very effective for the relief of their alleged symptoms. The formula will usually contain morphine, laudanum, or cocaine. If refused morphine, many addicts will ask for either methadone or meperidine. They will state that these drugs are not opiates but are synthetic drugs and are nonaddicting. All too frequently, uninformed physicians will be taken in by this story and will prescribe the synthetic analgesics. It is not unusual for addicts to attempt to obtain narcotics on the basis of some mild, chronic, nonfatal disease, such as asthma, arthritis, or chronic osteomyelitis, for which narcotics are not usually required or given. They will state that these diseases cause them terrible pain, that physicians in another town have been prescribing narcotics, and that their own physicians are away and they need drugs only until he returns. Occasionally, transient addicts may appear who are suffering with serious physical diseases, such as advanced cardiovascular diseases or emphysema. These cases present particularly difficult problems of treatment since the stress of abrupt withdrawal might prove fatal.

Nontransient addicts who are well known to the physician sometimes attempt to develop stories similar to those used by the transient, veteran, criminal type of addict. Nontransient addicts, however, are more likely to be sincere than the transient addict. Many such patients truly desire to be cured of their addiction and should be given all possible help. All too frequently, a nontransient addict will be a physician or a nurse. In such cases, the most likely drug of addiction is now meperidine largely because of the widespread belief among the medical profession that this substance is not addicting.

The presenting situation may also be that of an adolescent girl or boy who is brought to the physician by his parents or by other responsible persons. Generally, such adolescents will admit the use of drugs and present no particular problem in diagnosis. In such cases, the physician should exercise a certain degree of care since, because of the widespread publicity concerning addiction in adolescents, many parents may wrongly attribute normal or abnormal adolescent behavior to drug addiction.

When the history leads one to suspect addiction, a complete physical examination is of the utmost importance. A transient addict is frequently surprised when the physician indicates a desire to perform a complete physical examination and may refuse the examination and immediately leave the office. It is important to realize that there are no pathognomonic physical signs of addiction. The opiate drugs do not cause drunkenness as do the barbiturates and alcohol, so mental confusion, emotional instability, nystagmus, and ataxia are not to be expected. Since complete or partial tolerance to many of the effects of morphine develops during the course of addiction, doctors must not expect that addicts presenting themselves in their offices will show the same effects as might be expected in persons not addicted, namely, constriction of the pupils and sedative effects. An addict may or may not show pupillary constriction depending on how recently he has received a dose of morphine. Superficially, tolerant addicts will appear to be physically and mentally normal unless they have been without drugs for some time and signs of abstinence have appeared.

The most important findings are the presence of old and recent needlemarks. These needlemarks should be sought over the veins in the antecubital spaces, the deltoid region, the abdomen, the anterior surface of the thighs, and along the veins of the legs and hands. Multiple abscesses or old abscess scars are also suggestive. Miosis is not a completely reliable sign since partial tolerance to pupillary constriction caused by morphine develops during addiction. Addicts who are using either meperidine or methadone are likely to have induration and inflammation of large areas of the skin, particularly over the deltoid region and the anterior surface of the thighs. Presence of long scars or tattooing over superficial veins is extremely suggestive, particularly if fresh needlemarks are present. One important feature may be that no physical findings are present which satisfactorily explain the serious symptoms detailed by the patient when the history was taken.

Occasionally, persons who are suspected of taking cocaine or marihuana are brought to the physician for an opinion. Neither cocaine nor marihuana produces physical dependence. Since such patients are usually brought to the physician long after the drug effects have worn off, findings are generally scanty. The signs of cocaine intoxication, however, include mydriasis, sweating, tachycardia, increase in blood pressure, increase of deep tendon reflexes, nervousness, tremor, an unblinking stare, and auditory and visual hallucinations. Intoxication with marihuana is characterized by injection of the conjunctivae, a sleepy appearance, excessive giggling, silly behavior, absence of marked ataxia, and a strong odor on the breath resembling that of cubeb cigarettes.

There are no laboratory procedures generally available to the average physician which are helpful in the diagnosis of addiction. Reliable tests for the presence of morphine and cocaine in the urine have been developed, but these methods are quite complex and are not suitable for use in the ordinary laboratory. There are no methods available for the detection of morphine in blood and no laboratory procedures which are sufficiently specific for the detection of synthetic analgesics and marihuana in either blood or urine. Furthermore, if such procedures were available, a positive test of the presence of drugs in the urine would mean only that the person had recently taken or been given a dose of a drug and would not necessarily mean that he was addicted to that drug.

A definitive diagnosis of addiction to morphine or similar drugs depends upon the demonstration of the characteristic signs of abstinence following complete and abrupt withdrawal of drugs. In order to prove the presence of physical dependence, the addict must be isolated in an environment so well controlled that there is no possibility of any narcotic drugs, other than those prescribed, being obtained by the addict. If such an environment is available, isolation of the addict and withholding of all narcotics will prove the presence or absence of dependence on these drugs. The detection of physical dependence requires familiarity with the signs of abstinence from morphine.

If morphine is abruptly withdrawn from a patient who has been receiving as much as 0.26 to 0.39 gm. (4 to 6 grains) daily for a period of 30 days or more, few signs are observed during the first 16 hours of abstinence. The patient is likely to go into a restless, tossing sleep which may last for several hours. About 14 to 18 hours after the last dose of the drug was given, the addict will begin to yawn; rhinorrhea, perspiration, and lacrimation will appear. These mild signs increase in intensity during the first 24 hours of abstinence; thereafter, they become constant. Dilatation of the pupils then appears and, on close observation, recurring waves of gooseflesh will be seen. About 36 hours after the last dose of morphine was given, uncontrollable twitching of the muscles occurs. The patient will become extremely restless, will move from side to side in bed, and tremor of the face and tongue will be evident. The patient will complain of severe cramps in his legs, abdomen, and back; he will be unable to eat or to sleep for any period of time and vomiting and diarrhea frequently occur. Rectal temperature rises about 2 F, respiratory rate is usually elevated to 20 to 30 per minute and is increased in depth. Systolic blood pressure is usually elevated about 15 mm. of mercury, and patients will lose 5 to 15 pounds of weight during the second 24 hours of abstinence. A reduction in eosinophils per cubic millimeter of blood closely parallels the intensity of abstinence symptoms, and, at the peak intensity of the withdrawal illness, the eosinophil count will be nearly zero. This constellation of signs and symptoms constitutes as clear-cut and well delineated a syndrome as is ever seen in clinical medicine. Once it has been observed, it scarcely can be mistaken for any other condition. Acute signs and symptoms reach maximum intensity 48 hours after the last dose of morphine has been given and will remain intense until 72 hours have passed. Thereafter, they gradually subside and, after 5 to 10 days, completely disappear, although the addict may still be weak and sleeping poorly.

It is necessary to remember that the intensity of the abstinence syndrome varies from person to person and is, within limits, dependent on the dose that the addict has been receiving. Not all patients will exhibit all the signs of abstinence listed above. Severe grades of abstinence symptoms are practically always seen in addicts who have been receiving as much as 60 to 90 mg. (1 to 1.5 grains) of morphine four or more times daily. The intensity of abstinence symptoms becomes milder as the average daily dose declines. Since most adolescent addicts actually have been receiving only small amounts of opiates, the effects of abstinence are usually quite mild in juvenile addicts and very close examination may be required to detect the minor signs which are present.

Symptoms of abstinence from other opiates and the synthetic analgesics differ from morphine chiefly in intensity and duration. Effects of abstinence from heroin, dihydromorphinone, and metopon appear very rapidly, reach maximum intensity in 12 hours or less, are somewhat more severe than those from morphine, and subside more rapidly. Abstinence symptoms from codeine appear slowly and are more prolonged than those from morphine. Abstinence symptoms from dihydrocodeinone and eukodal are less intense than those from morphine, but more intense than those from codeine. The symptoms from dromoran® are quite similar in course and intensity to those from morphine.

Effects of abstinence from meperidine appear rapidly and are detectable two to three hours after the last dose of meperidine has been administered. Abstinence from meperidine has an effect somewhat similar to that of abstinence from morphine, but restlessness is even greater, and uncontrollable twitching and jerking of the muscles is extreme. The course of abstinence from meperidine is quite short and symptoms usually disappear two to three days following withdrawal.

Signs of abstinence from methadone come on slowly and are usually not detectable until the third or fourth day after the last dose of the drug has been given. Autonomic signs, such as sweating and mydriasis, are not very prominent. For this reason, the detection of abstinence symptoms is more difficult. The course of abstinence from methadone, however, is quite prolonged. Because of this, withdrawal of methadone is regarded by many addicts as being more uncomfortable than abstinence from morphine.

Frequently, because of the lack of suitable facilities, it may not be possible to isolate the addict and prove the presence of addiction by allowing the signs of abstinence to appear. It is almost useless to attempt to carry out diagnostic withdrawal in a general hospital, or even in mental hospitals, since addicts frequently manage to smuggle drugs into these environments. Diagnostic withdrawal may also be impossible because of the addict's refusal to undergo the procedure and, in certain cases, it may be unwise if the patient has some serious physical disease.

Where adequate isolation for diagnostic withdrawal is not possible, the diagnosis of addiction can be established only tentatively.

A new drug, N-allylnormorphine, which is a very effective antidote for morphine, has recently been shown to precipitate signs of abstinence in addicted persons within 15 minutes following its administration. This agent, therefore, may provide a means of quickly establishing the presence and degree of physical dependence on opiate drugs, but sufficient information on the safety and reliability of the procedure is not available at this time to permit a recommendation for general use of N-allylnormorphine as a diagnostic agent in suspected addiction.

REPORTING OF ADDICTS

The physician is under no legal obligation to report cases of addiction to Federal officials. The Bureau of Narcotics, however, welcomes voluntary reporting of addicts by physicians.

DISPOSITION OF THE ADDICT

Once a tentative diagnosis of addiction has been made, the physician would be well advised to seek consultation for confirmation. The problem then becomes one of advising the addict in accordance with the two cardinal principles of treatment set forth above—institutional treatment is necessary, and drugs must not be given to the addict for self-administration. The addict must be advised to seek admission to an institution for treatment. If he will not accept this advice, the physician may ethically refuse to take any further action. If the patient indicates willingness to undergo institutional treatment, steps should be taken immediately to obtain admission to the nearest and most available institution. Frequently, this can be accomplished immediately by telephoning the institution selected. The physician should refuse to administer any narcotics unless the patient shows the utmost diligence in obtaining entrance into an institution. Such excuses as time for arranging business and personal affairs or preference for one institution over another should not be accepted as reasons for the administration of narcotics; nor should the presence of signs of abstinence be a valid reason for immediate administration of narcotics unless the patient has such serious physical disease that stress of abstinence might prove fatal.

The physician must be prepared to resist the demands of addicts for narcotics and must exercise great care and judgment in determining whether physical disease which requires the immediate administration of opiates is present. If a physician yields to the importunities of an addict and gives him morphine for mild asthma, his waiting room will soon be cluttered with other addicts, all of them complaining that they have asthma or some similar condition. Such a situation can lead only to embarrassment to the physician and, perhaps, to difficulties with the authorities.

When the patient has agreed to go to an institution and has presented satisfactory evidence that he has taken steps to obtain admission (mere statement of the addict is not sufficient evidence of this), the problem is then reduced to the immediate management of the patient. The second principle, drugs should not be given to the addict for self-administration, applies here. It may be possible to place the patient in a general hospital where narcotics can be administered under direct supervision while final arrangements for entrance to the specialized institution are being made. The physician, in any event, should not give the addict a prescription for narcotics but should administer personally such drugs as are appropriate for immediate need only. It may be emphasized that it is always wise to seek consultation with another physician relative to the diagnosis of addiction and/or the presence of serious physical disease. A special record of narcotics administered should be kept, and, as has been previously mentioned, temporary administration of drugs should not be extended beyond the minimum time absolutely necessary for arrangements for admission to an institution to be completed. When the addict leaves his home community, he should be accompanied to the institution, whenever possible, by some responsible person, preferably a physician or a nurse.

No absolute rule can be laid down for the amount of narcotics to be given during the time arrangements for admission to a proper institution are being made. However, no amount of narcotic should be used in excess of that necessary for the immediate need of the patient. Either morphine or methadone may be used since these two drugs will adequately control abstinence from any of the other narcotics. It is advisable to limit the initial dose to 16 mg. (¼ grain) of morphine or 10 mg. (⅙ grain) of methadone. It practically never should be necessary to exceed as a single dose 60 mg. (1 grain) of morphine or 30 mg. (½ grain) of methadone. Even in patients with very severe heart disease, there is little danger of abstinence from morphine causing death if the above dosage schedule is followed. The type of drug given and the dose should be unknown to the addict and all possible precautions should be taken to prevent the addict from obtaining narcotics from other sources.

THE ADOLESCENT ADDICT WITH OR WITHOUT DEMONSTRABLE DEPENDENCE ON NARCOTIC DRUGS

Despite their ages, adolescent addicts must be separated from their usual environments. Institutional treatment for adolescents with demonstrable dependence is just as necessary as for adult addicts and the same procedures should be followed. Adolescents without physical dependence might possibly be sent to a camp, farm, or some other environment where rehabilitative treatment may be attained rather than sent to institutions where contact with older, more hardened addicts is unavoidable.

PATIENTS WITH INCURABLE, FATAL, PAINFUL DISEASES

Persons in this class are usually patients who are dying with advanced carcinoma, tuberculosis, or some other chronic disease. The problem in such instances is completely different from those described above. The physician is properly concerned, primarily, with relieving suffering and, only secondarily, with the addiction. Federal and State narcotic laws were not designed to prevent narcotics from being prescribed in such cases. Proper, ethical medical practice, however, demands that certain principles be followed.

Physicians prescribing narcotics for such patients should personally be attending the patient. A diagnosis of a painful, incurable disease should be confirmed by consultation with another physician. All means of relieving pain other than prescription of narcotics should be exhausted. Such measures include the use of drugs other than narcotics, physical therapy, and surgical procedures designed to relieve pain. When administration of narcotics becomes necessary, the physician should initially use drugs of lesser potency, such as codeine. When use of more potent narcotics is required, they should be given in the smallest possible dose and the interval of administration should be as long as possible. Precautions should be taken to ensure that the amounts of narcotics prescribed are no greater than those actually required for the particular patient so that there will be no surplus for diversion to illicit use. Whenever possible, the drugs should be given orally rather than hypodermically. Drugs should not be given directly to the patient for self-administration. The status of the patient and his disease should be reviewed periodically to make certain that the diagnosis is correct and that definitive, curative therapy is not possible.

COCAINE AND MARIHUANA ADDICTS

Since physical dependence on these drugs does not develop and there is no withdrawal illness, the patient should simply be advised to seek treatment in a properly staffed institution. Hospitalization until admission to an institution can be obtained is not necessary or advisable.

CHOICE OF INSTITUTION

The choice of an institution depends upon the type of case, the financial situation of the patient, and other factors. Many private sanatoriums in the United States make a specialty of the treatment of narcotic drug addiction. Advice concerning these institutions can be obtained from local medical societies, the American Hospital Association, The American Psychiatric Association, or the American Medical Association. Alternatively, the physician may investigate the possibilities of having the patient admitted to a public, local, or state institution. Information concerning such institutions can be obtained by contacting local or state health departments. Lack of suitable local facilities remains one of the difficulties in the treatment of addiction. Physicians should support the establishment of such facilities in states in which no provision for addicts has yet been made. Where local facilities are not available, addicts can be referred to the two federal hospitals maintained by the U. S. Public Health Service at Lexington, Kentucky, and Fort Worth, Texas. Persons addicted to opiates, synthetic analgesics, cocaine, and marihuana are eligible for admission to these institutions. Patients who are addicted to alcohol, barbiturates, or bromides are not eligible for admission to these federal hospitals unless they are also addicted to morphine, synthetic analgesics, marihuana, or cocaine. If the patient is indigent, treatment is available without charge. If the patient has funds, he is required to pay $5.00 daily for his treatment. Addicts entering these institutions are asked to remain at least 135 days before being discharged. Ordinarily, there is no waiting list for male patients and admission can be arranged readily by writing or telephoning the Medical Officer in Charge of either hospital. Only the institution in Lexington has facilities for female patients, consequently, women usually must wait for a short time before they can be admitted.

THE NEED FOR COMPULSION

Unfortunately, many addicts will not go to an institution or remain there until treatment is completed unless compelled to do so by legal means. There is a great need for legislation in most of the states which would make it possible to commit addicts to institutions where they would be forced to remain until maximum benefit of treatment had been obtained. The Federal Bureau of Narcotics has drafted a text of such a proposed law and will distribute copies to interested persons on request. Legislation has also been proposed which would make it possible for the two U. S. Public Health Service Hospitals that treat addicts to accept and hold addicts committed under state laws. Passage of such laws deserves the support of all physicians.

TREATMENT FOLLOWING DISCHARGE FROM INSTITUTION

The follow-up treatment after the patient has been discharged from an institution is usually the weakest link in the overall treatment of addiction, and it is in this particular phase of the problem that the ordinary physician can make the greatest contribution. The physician should do what he can to assist the addict to find a job following discharge from an institution. He can attempt to make arrangements, utilizing such social agencies as are available, to separate the addict from the environment which played a role in engendering the addiction. He should encourage the former addict to participate in the activities of community groups, such as churches and clubs. The physician can advise the relatives of a former addict respecting environmental and familial factors which may be contributing to the patient's difficulties. He can also administer such forms of psychotherapy as he is qualified to carry out. When suitable facilities are available, intensive psychotherapy by a qualified psychiatrist is of great value provided the patient is willing to accept such treatment. The addict should remain under close supervision for at least two years following discharge from an institution. Although the tendency to relapse is very great in addiction, the physician should maintain an optimistic attitude even in the face of repeated recurrence and should continually encourage and support the addict.

MANAGEMENT OF DISEASE IN FORMER ADDICTS

When a person who formerly has been addicted to morphine become afflicted with a disease for which narcotics are usually prescribed (this situation usually develops when a surgical operation becomes necessary), he should be handled just as if he had never been addicted. Since the former addict has been withdrawn from narcotics and has lost his tolerance to them, narcotics should be prescribed in the same doses and at the same intervals as is customary with persons who have never been addicted. Larger doses are not required. Narcotics should never be given to the former addict for self-administration and use of narcotics should be discontinued as soon as possible. The addict should then be supervised closely for a month or so in order to avert relapse to uncontrolled use of drugs.

MANAGEMENT OF ADDICTS WHO HAVE RELAPSED

The management of patients who have relapsed to abusive use of narcotic drugs should follow the same lines as detailed above. The only difference is that the period of supervision following discharge from an institution should be extended.

NEW AND NONOFFICIAL REMEDIES

The following additional articles have been accepted as conforming to the rules of the Council on Pharmacy and Chemistry of the American Medical Association for admission to New and Nonofficial Remedies. A copy of the rules on which the Council bases its action will be sent on application.

R. T. Stormont, M.D., *Secretary.*

Aurothioglucose (See New and Nonofficial Remedies 1952, p. 441).

Schering Corporation, Bloomfield, N. J.

Suspension Solganal in Oil: 1.5 cc. ampuls. A suspension in sesame oil containing 6.7 mg., 16.7 mg., or 33.3 mg. of aurothioglucose in each cubic centimeter with 2% aluminum monostearate. Preserved with 0.1% propylparaben.

10 cc. vials. A suspension in sesame oil containing 10 mg., 50 mg., or 100 mg. of aurothioglucose in each cubic centimeter with 2% aluminum monostearate. Preserved with 0.1% propylparaben. U. S. Trademark 261,372.

Gamma Benzene Hexachloride (See New and Nonofficial Remedies 1952, p. 74).

Commercial Solvents Corporation, New York

Lotion Kwell 1%: 60 cc. and 500 cc. bottles. A lotion containing 10 mg. of gamma benzene hexachloride in each cubic centimeter.

Bismuth Glycolylarsanilate (See New and Nonofficial Remedies 1952, p. 145).

Winthrop-Stearns, Inc., New York

Tablets Milibis: 0.5 gm. U. S. Patent 1,934,017.

Penicillin for Injection for Prolonged Action (See New and Nonofficial Remedies 1952, p. 126).

Merck & Company, Inc., Rahway, N. J.

Procaine Penicillin G for Aqueous Injection: 300,000, 1,500,000, and 3,000,000 unit vials. Buffered with sodium citrate.

Human Pertussis Immune Serum-N.F. (See New and Nonofficial Remedies 1952, p. 380).

Junior League Blood Center of Milwaukee, Inc., Milwaukee

Pertussis Immune Serum (Human): 10 cc. and 20 cc. vials.

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CAUTION IN THE CHEMOTHERAPY OF TUBERCULOSIS

Three recently developed antituberculosis drugs, isoniazid (isonicotinic acid hydrazide), iproniazid (1-isonicotinyl-2-isopropyl hydrazine), and pyrazinamide, are now undergoing extensive clinical testing. These drugs have shown definite but limited therapeutic activity against human tuberculosis,[1] but there is much concern among workers in this field that their potential value may rapidly be neutralized if they are used improperly or indiscriminately by those not trained in the treatment of tuberculosis. The recent increase in the availability of isoniazid has brought this problem into acute focus.

One of the major threats to the effective use of the newer forms of tuberculosis chemotherapy lies in the possibility that physicians will attempt to use these drugs as the sole form of therapy. As the American Trudeau Society,[2] the American College of Chest Physicians,[3] and other organizations dealing with tuberculosis have repeatedly emphasized, these compounds are not in themselves cures in spite of the earlier optimistic publicity given to some of them. They, like other forms of chemotherapy, must be used as adjuncts to the time-tested measures of bed rest, collapse therapy, and surgical procedures. Moreover, as adjuncts they have not yet proved clinically superior to, or even so effective as, the combination of streptomycin and *p*-aminosalicylic acid in the treatment of tuberculosis.[2]

Another threat to the continued effectiveness of these drugs arises from the fact, now well established, that the tubercle bacillus is capable of developing resistance to them rapidly both in vitro and in vivo. In the clinic, bacilli resistant to isoniazid, iproniazid [4] and pyrazinamide [1b] have appeared in some cases after only two to four weeks of therapy, although a somewhat longer period is usually required. Such organisms seem to develop especially rapidly in patients with far-advanced cavitary pulmonary tuberculosis, and their appearance is associated with a recurrence of symptoms despite continued treatment. In the laboratory, bacilli with a 5-fold increase in resistance to iproniazid [5] and a 16-fold to 4,000-fold increase in resistance to isoniazid [6] have been obtained by serial passage on media containing these drugs. Resistance to isoniazid, like that to streptomycin, may develop in a single step [7] and may be present in organisms never previously exposed to the drug.[8]

These observations emphasize the need for care in the use of the new drugs. Steenken and co-workers,[9] and Grace and associates (see this issue, page 1241) warn that if these compounds are used alone they may benefit the patient only temporarily and may prejudice his chance of future response to combined drug therapy, surgery, or both. This practice may also result in the widespread appearance of strains of tubercle bacilli resistant to these drugs and in the eventual infection of others with organisms resistant to both streptomycin and isoniazid or one of the other compounds. It is suggested by Grace and associates that these drugs be employed only in multiple combinations with streptomycin and *p*-aminosalicylic acid or other chemotherapeutic agents active against mycobacteria and not related by cross resistance. They base this suggestion on the well-established fact that the production of resistant variants of an organism can be inhibited by simultaneous (but not consecutive) exposure of that organism to at least two drugs with different modes of action. That this general principle applies specifically to isoniazid is suggested by the observation that no tubercle bacilli resistant to either isoniazid or streptomycin have been encountered in cultures exposed simultaneously to therapeutic concentrations of the two drugs,[10] although strains resistant to both have been obtained by successive exposure to these compounds.[7b]

Still another hazard in the use of these drugs is that of inadequate dosage. Since there has not yet been time for adequate long-term evaluation of different dosage schedules for the different forms and stages of tuberculosis, it is obvious that no statement about optimum dosage

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1. (*a*) New Antituberculosis Drugs, editorial, J. A. M. A. **148:** 1034 (March 22) 1952. (*b*) Yeager, R. L.; Munroe, W. G. C., and Dessau, F. I.: Pyrazinamide (Aldinamide) in the Treatment of Pulmonary Tuberculosis, Am. Rev. Tuberc. **65:** 523 (May) 1952.

2. Committee on Therapy of American Trudeau Society: Developments in Antimicrobial Therapy of Tuberculosis, May 26, 1952, Am. Rev. Tuberc. *To be published.*

3. Committee on Chemotherapy and Antibiotics of the American College of Chest Physicians: Isoniazid (Isonicotinic Acid Hydrazide), read [illegible] the meeting of the American College of Chest Physicians, Chicago [illegible] 5, 1952.

4. Steenken, W., Jr.; Meade, G. M.; Wolinsky, E., and Coates, E. O., Jr.: Demonstration of Increased Drug Resistance of Tubercle Bacilli from Patients Treated with Hydrazines of Isonicotinic Acid, Am. Rev. Tuberc. **65:** 754 (June) 1952.

5. Steenken, W., Jr., and Wolinsky, E.: Antituberculous Properties of Hydrazines of Isonicotinic Acid (Rimifon, Marsilid), Am. Rev. Tuberc. **65:** 365 (April) 1952.

6. (*a*) Buck, M., and Schnitzer, R. J.: The Development of Drug Resistance of M. Tuberculosis to Isonicotinic Acid Hydrazide, Am. Rev. Tuberc. **65:** 759 (June) 1952. (*b*) Karlson, A. G., and Ikemi, Y.: The Occurrence of Human-Type Tubercle Bacilli (H37Rv) Resistant to Isonicotinic Acid Hydrazide in a Culture Not Previously Exposed to the Drug, Proc. Staff Meet., Mayo Clin. **27:** 239 (June 4) 1952.

7. (*a*) Pansy, F.; Stander, H., and Donovick, R.: In Vitro Studies on Isonicotinic Acid Hydrazide, Am. Rev. Tuberc. **65:** 761 (June) 1952. (*b*) Szybalski, W., and Bryson, V.: Bacterial Resistance Studies with Derivatives of Isonicotinic Acid, Am. Rev. Tuberc. **65:** 768 (June) 1952.

8. Hobby, G. L., and Lenert, T. F.: Resistance to Isonicotinic Acid Hydrazide, Am. Rev. Tuberc. **65:** 771 (June) 1952. Karlson and Ikemi.[6b]

9. Steenken, W., Jr.; Meade, G. M.; Wolinsky, E., and Coates, E. O., Jr.: Drug Resistant Tubercle Bacilli from Patients Treated with Isonicotinic Acid Hydrazides, correspondence, J. A. M. A. **149:** 187 (May 10) 1952.

10. Middlebrook, G.: Sterilization of Tubercle Bacilli by Isonicotinic Acid Hydrazide and the Incidence of Variants Resistant to the Drug in Vitro, Am. Rev. Tuberc. **65:** 765 (June) 1952. Pansy, Stander, and Donovick.[7a]

or duration of administration can be made. It should be equally obvious that, until such information is available, use of the drugs should be limited to institutions where rigidly controlled observations on different treatment schedules can be made, recorded, and pooled with those of other institutions for final evaluation. Random use of the drugs on an empirical basis can result only in waste, inefficiency, inconclusive results, and harm to some patients.

While there have been few toxic reactions to these drugs to date with the doses used, such reactions must be watched for carefully during and after therapy. According to the Committee on Therapy of the American Trudeau Society, "isolated instances of intractable asthma, persistent hypotension, and increased susceptibility to the effects of other drugs (particularly epinephrine, ephedrine, atropine, and demerol) and even deaths have been reported in patients receiving isoniazid or its isopropyl derivative, but the causal relationship has not been clearly established. Minor untoward effects which have been reported are hyperreflexia, constipation, postural hypotension and dizziness, difficulty in starting micturition, eosinophilia, and the appearance of casts, albumin, and reducing substances in the urine. Routine precautions during treatment should include frequent neurologic examinations, blood counts, urinalyses, and tests for renal and hepatic insufficiency. As both drugs are excreted through the kidneys, impairment of renal function may result in the cumulation of toxic concentrations of the drug in the body. For this reason, the renal function should be measured before treatment and, if it is impaired, the dose should be reduced accordingly." [2]

These facts must be considered carefully by every physician contemplating the use of one of the new drugs in a patient with tuberculosis.

FULL COST TUITION IN MEDICAL SCHOOLS

One of the remedies that has been suggested for the present financial problems that confront the medical schools is that the schools charge their students for the full cost of their education. This suggestion usually is advanced by those not close to medical education, but it has received at least passing attention by a few schools in recent years. Fortunately, the great majority of medical schools have been quick to see the dangers of such a program and have refused to give it serious consideration.

Elsewhere in this issue, page 1243, the Council on Medical Education and Hospitals has an important statement summarizing the many damaging effects that could result from attempts to charge medical students for the full cost of their education. The Council points out that a plan of charging full cost tuition differs only in technical details from the practices of the proprietary medical schools at the beginning of the present century. The Council further gives several reasons why such a program would probably give only temporary relief and in time would actually make the situation of the medical schools worse. It is predicted that such a program would soon discourage support of the medical schools by other sources that have been so carefully cultivated and to whose interest and support much of the present excellence of our medical schools is due.

The school adopting such a program would itself soon suffer greatly. It would be at a serious disadvantage in the competition for able students and would probably find itself largely a school of last choice.

The Council properly fears that the consequences to the ideals and ethics of medical practice that might result from such a plan would be one of its most disastrous features. If medical education is placed on a dollar for dollar basis, the graduate laboring under a large debt could easily be tempted to regard his practice as a business enterprise purchased in the market place to be exploited and commercialized accordingly. Because of these and other inherent and potential dangers, the Council very rightly concludes its statement by condemning plans for charging full cost tuition and by urging the medical schools to continue to cultivate and expand accepted sources of support.

CLOTHING PROTECTION AGAINST RADIATION HAZARDS

In his endless struggle to protect himself against the demands of an exacting environment, man has successfully utilized various types of protective clothing to oppose the destructive action of fire, water, and noxious chemicals. Recently, Holden and Owings [1] called attention to the role of protective clothing in reducing some of the hazards of atomic and radiological warfare. Although clothing does not afford protection against the high neutron and gamma fluxes from an atomic bomb blast, it can protect against alpha, beta, infra-red, and ultraviolet radiations. Following an atomic explosion, an area of variable size is quickly contaminated by radioisotopes emitting alpha, beta, gamma, and x-radiations; in contrast, the hazard found in radiological warfare is due principally to a single type of radiation.

Alpha radiation emanating from an area contaminated by an atomic bomb burst causes damage when it affects the body internally. Effective protection against external contact with alpha radiation is made possible by wearing thin clothing. Ordinarily clothing will reduce a beta-ray flux from combined fission products, but shielding against gamma radiation by means of wearing apparel is impractical today. Since radiation produced by an atomic blast includes beta and gamma rays, this circumstance is analogous to one in which a warrior wearing medieval armor is exposed simultaneously and equally to a combined discharge of arrows and bullets. The armor provides full protection against arrows but incomplete protection against bullets. Similarly, clothing protects adequately against beta rays but inadequately against gamma radiation. All types of radioisotopes are fraught

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1. Holden, F. R., and Owings, A. F.: Protection Afforded by Clothing Against Radiation Hazards, A. M. A. Arch. Indust. Hyg. 6:67 (July) 1952.

with peril when inhaled or ingested, alpha rays being more damaging locally to internal body structures than beta rays or gamma rays, and beta rays more destructive than gamma rays. Ingestion is due to the transference of radioactive materials from clothing to the mouth via the hands.

While clothing provides limited protection against external radiation, the controlled use of clothing serves not only to lessen body contamination and reduce the dangers of ingestion and inhalation but also to prevent the spread of radiocontaminants. There are numerous instances in which radioactive substances have been transported into previously uncontaminated areas by shoes. The spread of radioisotopes is the atomic age counterpart of Typhoid Mary.

In conclusion, it may be pointed out that the value of protective clothing in atomic and radiological warfare lies in the control of contamination rather than in providing protection against external radiation. The civilian population should be taught to care for itself when trained personnel and decontamination centers are not available. The basic requirements of a decontamination center are laundry facilities, monitoring instruments, and showers that have segregated clean and contaminated areas on either side of them. As for clothing itself, wearing apparel should be light in weight, nonporous, washable or expendable, and should completely cover the body, especially at the wrists, ankles, and the neck. Finally, light-colored clothing reflects infra-red radiation, thereby minimizing burns.

SURVEY OF DISCHARGED MEDICAL PERSONNEL

An increasing number of physicians are being discharged each month from military service after completion of their statutory tour of duty. In some instances dissatisfaction has been expressed with the utilization of medical personnel by the armed forces and the type of duties assigned. In an effort to evaluate the validity of the complaints, the Council on National Emergency Medical Service is contacting all physicians discharged from service since July 1, 1951. The questionnaire being distributed is designed to determine the use made of physicians by the armed services, the amount of time spent on the treatment of other than military personnel, and ways in which the American Medical Association can better serve medical personnel in uniform.

In addition to providing a service to physicians on active duty and those about to enter the armed services, it is hoped that the results of the survey will provide a sound basis for recommendations concerning the "doctor draft law." This law, which was originally enacted on Sept. 9, 1950, with the approval of the American Medical Association, was designed to supply physicians for the armed services. Inasmuch as this act will expire on July 1, 1953, it is necessary to establish a position as to whether legislation of this type should be extended and, if so, the form that it should take.

It is the earnest desire of the American Medical Association to apprize itself of the facts in this regard, to attempt to correct any justifiable source of complaint, and to formulate sound and constructive recommendations with respect to future draft legislation.

SAMPLE OF 1951 PHYSICIANS' INCOMES

On Wednesday of this week the Office of Business Economics of the U. S. Department of Commerce published the results of its interim professional income surveys for physicians, dentists, and lawyers. Unlike the bench mark study of 1949, which involved the analysis of 30,000 replies, the current survey of physicians' incomes is based on less than 1,000 replies from a sample of approximately 5,000 physicians selected by the Bureau of Medical Economic Research of the American Medical Association. The mean net incomes for 1950 and 1951 were $11,538 and $12,518 as compared with $11,058 reported in the big survey of 1949 income.[1]

The key sentence in the report of the 1949 survey published in the Survey of Current Business [2] was: "During the same period [1929-1949], all earners in the general population (wage and salary workers, as well as independent business and professional workers) recorded almost the identical relative increase (109 as compared with 108 percent) as all physicians." The same relationship held in 1950 and 1951 according to the results of this small sample; that is, the average net incomes of physicians are just keeping pace with the rise in the incomes of all earners in the general population. The incomes of the physician and the patient continue to grow apace.

THE NEW KEOGH-REED BILLS

Elsewhere in this issue (page 1244) is an explanation of the new features of the revised Keogh-Reed bills to establish a voluntary pension system. This revised version was introduced in the House of Representatives on June 27, too late to be considered in the closing days of the session. The bills as revised incorporate all the amendments suggested by the American Medical Association. Physicians should acquaint themselves with the provisions of the revised bills so that they can discuss these (identical) bills with other self-employed persons and pensionless employed persons such as lawyers, dentists, farmers, retailers, when they are asked for their opinions. They may also be asked for an opinion by their local candidates for the House and the Senate, as this measure is important to professional men and women.

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[1] Dickinson, F. G., and Bradley, C. E.: Survey of Physicians' Incomes, J. A. M. A. **146**: 1249-1255 (July 28) 1951. Reprinted as Bulletin 84, Bureau of Medical Economic Research, American Medical Association, Chicago, 1951.

[2] Weinfeld, W.: Income of Physicians, 1929-49, Survey of Current Business, (July) 1951, U. S. Department of Commerce.

THE PRESIDENT'S PAGE

A MONTHLY MESSAGE

In going about the country, I have become increasingly convinced that the general public and our own members still need to know more about the American Medical Association and its activities. In certain areas it is regarded by the public as a pressure organization devoted to the interests of management and industry and its own selfish interests. It has the reputation of being undemocratic. Its own members are often too little aware of the vast amount of work done by the Association in education, public health, and in eliminating fraud and charlatanism. The public in many cases believes that the Association limits the number of doctors and is against the socialization of medicine only for pecuniary reasons.

This is a shocking situation, and our public relations must continue to educate both the profession and the public as to the actual facts and must disabuse their minds of all these impressions, none of which are based on fact. In this and succeeding President's pages, I plan to further outline the real status and policies of the Association.

First of all, let us follow an idea that has germinated in the mind of an individual member. This member desires to see the A. M. A. adopt a certain policy that he has outlined in a resolution. There are two courses he can follow. First, he may take the resolution to his county society and introduce it there. He may present his arguments in open meeting, where he can be asked questions. A debate follows. If it is a good idea, his county society may approve the resolution and send it to the state society house of delegates with instructions to the county society delegates to introduce it. It may also circularize other county societies and enlist their support.

This resolution then is introduced into the house of delegates of the state, where it is immediately referred to a reference committee. The proponents of the resolution then appear before the reference committee and argue their case. One does not have to be a member of the house to appear before a reference committee and be heard. The reference committee then accepts, amends, or rejects the resolution and reports to the house. Final action is determined by the house. Again, if it meets with the approval of the majority of the house, it is forwarded to the A. M. A. House of Delegates, where the state's delegates are instructed to introduce it and work for its passage. Again, this may be circulated to other states before the meeting.

In the A. M. A. House of Delegates the same procedure is followed. Again, any member of the A. M. A., whether or not a member of the House, may appear before the Reference Committee and argue on the merits or demerits of the resolution. If the majority of the House concurs, the resolution becomes a part of the policies of the A. M. A. and the Board of Trustees is directed to carry out the policy. A single member, therefore, who holds no office or official position in any body can follow his idea right to the top. What could be more democratic?

Another method that can be followed is for the individual member to ask a member of the House to introduce the resolution direct, where it will follow the usual procedure. I followed this method once, myself. Twenty-three years ago, when I was in the government service, I desired the support of the A. M. A. in a method that was being pursued in the physical selection of airplane pilots. I asked a member of the A. M. A. House to introduce the resolution. I convinced him that approval of it was desirable. He introduced it, and both he and I appeared before the Reference Committee to support it. He was known by the Committee, but I was not, and I did most of the talking and answered most of the questions. I could not have received more courteous attention had I been President of the A. M. A. instead of a completely unknown member. The Reference Committee brought in a favorable report, and the House adopted the Resolution unanimously. Do you know of any other organization in which the individual member has more democratic rights?

Next month I expect to continue this discussion of the A. M. A., its policies, and its relation to the public.

LOUIS H. BAUER, M.D., HEMPSTEAD, N. Y.

ORGANIZATION SECTION

FEDERAL COMMUNICATIONS COMMISSION POSTPONES EFFECTIVE DATE FOR REPLACEMENT OF DIATHERMY EQUIPMENT

The effective date for compliance with the rules of the Federal Communications Commission has been postponed for one year. Physicians or hospitals now using nonconforming short wave diathermy apparatus have until June 30, 1953, to replace the diathermy equipment that does not conform with the rules of the Federal Communications Commission.

The Council on Physical Medicine and Rehabilitation forwarded a petition to the Federal Communications Commission on March 14, 1952, requesting an extension of the final date for one and one-half years. The following reasons were given for an extension. 1. Several thousand physicians and hospitals would be in violation of the rules of the commission or would have to deprive their patients of the therapeutic benefits of diathermy apparatus. 2. The present capacity of the industry to produce ample diathermy appliances was insufficient owing to cut-backs of materials and difficulty in obtaining labor. The Council was supported by several medical and industrial organizations in its request for an extension of time.

In Public Notice 77631, dated June 26, 1952, the Federal Communications Commission announced the extension of the effective date to June 30, 1953. In extending the date the commission pointed out that any further requests for extension of time would be subject to special investigation of individual cases. The commission pointed out the importance of physicians and hospitals equipping themselves with conforming diathermy equipment.

Requests have come to the Council on Physical Medicine and Rehabilitation concerning the possibility of converting short wave diathermy apparatus to conform with the rules of the F. C. C. The Council has been informed by reliable agencies that this is a difficult electrical and mechanical procedure and that the costs involved were as much, if not more, than replacing the equipment with council-accepted and the F. C. C.-approved type of short wave diathermy apparatus. The reconversion of diathermy equipment must be certified by reliable engineers. Surgical diathermy equipment for the generation of electric cutting currents, electric desiccation, and electric coagulation, used only for these medical purposes, are exempt and do not have to be replaced.

HANDBOOK FOR DOCTORS' SECRETARIES

An attractive booklet entitled "Winning Ways with Patients" is the newest A. M. A. public relations aid for physicians. It is a public relations handbook for the physician's secretary, receptionist, or medical assistant, designed to teach medical personnel how to make every patient's contact with the physician or his office staff as pleasant and satisfying as possible.

Because many patients complain "the doctor keeps me waiting too long," one section of the booklet is devoted to techniques for scheduling appointments, fitting patients without appointments and referred patients into the schedule, and keeping patients happy while they are in the waiting room.

Brightly illustrated, "Winning Ways with Patients" discusses telephone techniques, the best methods of billing and collecting medical fees, and other suggestions for improving doctor-patient and secretary-patient relationships. Personality traits, proper office dress, the importance of keeping office confidences, and the various responsibilities of the doctor's assistant are stressed.

This booklet, prepared by the Public Relations Department of the American Medical Association, will be mailed to all physicians in the United States after Aug. 1, and will also be made available to medical societies sponsoring courses for medical secretaries and assistants.

A. M. A. TO SPONSOR PUBLIC RELATIONS INSTITUTE

The American Medical Association's first Public Relations Institute will be held Sept. 4-5 at Chicago's Edgewater Beach Hotel. Featuring clinics covering a variety of practical PR problems, the institute will stress "Putting PR to Work" in local medical society programs. Subjects to be discussed at the two day meeting include increasing member-participation, developing better press-radio-TV relations, working with community groups, strengthening public service activities, carrying on field service activities, editing a society newsletter, utilizing direct mail techniques, and planning radio and TV programs.

Designed primarily for lay public relations personnel of state and county medical societies, the institute will supplement the annual Medical Public Relations Conference to be held Dec. 1, prior to the A. M. A.'s Clinical Session in Denver.

STATE MEDICAL LEGISLATION

Louisiana

Bills Enacted.—H. 219, has become Act No. 202 of the Acts of 1952. It amends and re-enacts the law relating to depositions and discovery by providing, among other things, that in an action in which the mental or physical condition of a party is in controversy, the court in which the act is pending or in which the judgment was originally rendered may order him to submit to a **physical or mental examination by a physician.** The order may be made only on motion for good cause shown and upon notice to the party to be examined and to all other parties and shall specify the time, place, manner, condition, and scope of the examination and the person or persons by whom it is to be made. Reports of such examination shall be delivered to the person examined, or the results of the examination may not be used as evidence at the trial. H. 428 has become Act No. 150 of the Acts of 1952. It provides, among other things, that no body shall be **cremated** except upon the permit or approval of the coroner. H. 429, has become Act No. 151 of the Acts of 1952. It provides, among other things, that by mutual agreement between two-thirds of the coroners of any parish included in any congressional district of the state approved by the police jury of the parish in which such coroner has agreed, there may be established a **forensic laboratory** for the mutual use and benefit of such coroners which shall be at the disposal of the coroners and law enforcement officers of the parish. It also provides that following an autopsy, the coroner may retain any specimens or organs of the deceased which in his discretion are desirable or needful for anatomic, bacteriologic, chemical, or toxicological examination as well as for possible evidence before a grand jury or court. H. 864, has become Act No. 373 of the Acts of 1952. It places all nursing homes under jurisdiction of the state department of health and directs the department of health to provide rules and regulations governing the operation of such homes. It also makes it unlawful for anyone operating a nursing home to administer, **without a doctor's prescription,** any **sleeping potion or medicine or give hypodermic injections** to any inmate of such nursing home. S. 165, has become Act No. 288 of the Acts of 1952. It authorizes charity hospitals of the state to accept as patients persons suffering from **alcoholism.** S. 194, has become Act No. 296 of the Acts of 1952. It puts on a prescription basis the sale and distribution of **barbiturates** and other hypnotic and somnifacient drugs and also **central nervous system stimulants** such as amphetamine and desoxyephedrine. S. 279, has become Act No. 297 of the Acts of 1952. It provides that at least five members of the **state board of health** shall be duly qualified and registered physicians.

Massachusetts

Bills Introduced.—H. 2510, (Appendix E) and H. 2558, propose the creation and **establishment of a state school of medicine** and a state school of dentistry in the City of Boston or vicinity.

Bills Enacted.—H. 2525, Appendix B, has become Resolve No. 84 of the Laws of 1952. It provides for the continuation of a special commission for the purpose of continuing its investigation and study relative to **high blood pressure** and the problem of its control. S. 209, has become Chapter 535 of the Laws of 1952. It authorizes the department of mental health, in connection with the adjudication of restoration of soundness of mind to appoint two physicians certified by the **American Board of Neurology and Psychiatry,** Incorporated, to examine the subject and advise the court of his present mental condition.

Prepared by the Bureau of Legal Medicine and Legislation.

MEDICAL NEWS

CONNECTICUT

Fluoridation of Water Supplies.—The joint conference committee of the Connecticut State Medical Society and the Connecticut State Dental Association has endorsed fluoridation of public water supplies to aid in the control of tooth decay and will recommend to its parent organizations that an educational program be developed to further acquaint the public and community leaders with the benefits of fluoridation. The Connecticut Health League, an organization of 25 state and voluntary health agencies, endorsed water supply fluoridation during its semiannual meeting June 20 at the University of Connecticut, Storrs. Two communities in the state, New Britain and Cromwell, already are fluoridating their water supplies.

Emergency Medical Services.—According to Dr. Edward J. Whalen, Hartford, president of the Connecticut State Medical Society, emergency medical services are now being sponsored by medical associations for more than 1 million Connecticut residents, half the state population. These 24 hour telephone emergency systems are operating in 12 principal communities and are part of a long-range community service program sponsored by the society and its eight component county medical associations. Cities of more than 50,000 population that now have medical association emergency systems in operation include Hartford, New Britain, Waterbury, Bridgeport, and Stamford. Smaller communities where the new systems are operating are Torrington, Danbury, Greenwich, Norwalk, Manchester, Norwich, and Putnam. In addition to the new telephone emergency plan, there are plans mutually arranged between physicians and local hospitals by which patients may obtain emergency medical attention. These plans have proved a valuable service in communities where facilities are inadequate for operation of the emergency telephone systems. The three principal requirements of the new plans are (1) that the system be sponsored and supervised by a medical group or association, (2) that emergency services be available at all times through a central telephone number, and (3) that an organized panel of physicians be available for emergency calls.

ILLINOIS

Dr. Geen Honored.—Dr. James S. Geen of Utica, who has practiced medicine for 50 years and has been a member of the board of the LaSalle County Tuberculosis Sanatorium for 30 years, was honored at a banquet, May 27, in Ottawa. Dr. Geen, who was named to the board of trustees three years after the sanatorium was opened, has served continuously since that time by appointment of the county board of supervisors. He is now president of the board of directors.

State Hospital Needs Help.—The Illinois Department of Public Welfare has issued a call for 153 medical workers to augment the staff of Chicago State Hospital. According to Mr. Fred K. Hoehler, department director, the institution needs 119 attendants, 14 physicians, 3 supervising nurses, 6 psychiatrists, 4 fellows in psychiatry, 1 bacteriologist, 2 dietitians, 1 hydrotherapist, and 3 senior medical students to help in the care and treatment of more than 5,000 patients. The positions are under civil service.

Chicago

Dr. Dragstedt Named Thomas D. Jones Professor.—Dr. Lester R. Dragstedt, chairman of the department of surgery of the University of Chicago Medical Center since 1947, has been named the Thomas D. Jones Distinguished Service Professor of Surgery, to succeed the late Dr. Dallas B. Phemister.

Dr. Dragstedt served as the Nathan Smith Davis professor of physiology and pharmacology and chairman of the department at Northwestern University from 1923 to 1925, when he was appointed to the surgery staff of the University of Chicago. Dr. Dragstedt received the gold medal of the American Medical Association in 1950, the silver medal of the Association in 1947, and the gold medal of the Chicago Medical Society for his work on gastric ulcer.

Cancer Incidence in Chicago Area.—A study of cancer incidence among residents of the Chicago area showed no real change from 1937 to 1947, according to a survey just released by the National Cancer Institute. The study showed a numerical increase, but that was attributed to the aging of the population. The survey results differ from those obtained in five previously published studies, dealing with the metropolitan areas of Atlanta, Denver, New Orleans, Pittsburgh, and San Francisco, where more significant increases in cancer incidence were noted. Dr. John R. Heller, National Cancer Institute director, said, "At present we can only speculate on the meaning of the 'no change' report for the Chicago area. It may, in part, denote geographical differences in cancer incidence. On the other hand, the differences in cancer incidence between Chicago and the other cities may be due in part to improved case-finding and diagnostic techniques. In Chicago there is some indication that diagnostic techniques were already at a fairly high level in 1937."

In the Chicago area, cancer occurs 100 times as often in persons over 65 as in children under 15; however, certain cancers were found to be relatively common in younger persons, notably the leukemias and lymphomas, and soft tissue, bone, and brain tumors. In women, the commonest cancers are those of the breasts, 25%; genital organs, 23%; and digestive system, 27%. In men, 40% of all cancers originate in the digestive system, 13% in the respiratory system, 10% in the genital organs, 8% on the skin, and 8% in the urinary tract. In cancer of the uterus, diagnosed in its early, localized stage, 90% of the patients were found to survive for a year. The survival rate drops to 71% when diagnosis is made after regional involvement, and to 35% when diagnosis follows metastasis. Further, the Chicago data indicate that cancer of the uterus, which is an easily accessible site, is discovered in a localized stage in only 54 of 100 cases. Here, then, is a great need for earlier diagnosis, the report finds. The study also notes that uterine cancer occurs 58% oftener in nonwhite women than in white women. This may be related to the number of pregnancies or the medical care received by mothers during childbirth. The Chicago survey was carried out through the Public Health Service with the cooperation of the Cook County Medical Society, the Chicago Hospital Council, and city and state health departments.

LOUISIANA

Advancement in Rank at Tulane.—Twenty-three advancements in rank among Tulane University's school of medicine faculty have been announced. Faculty members advanced to full professorships include John Adriani (surgery), Paul C. Beaver, Ph.D., (parasitology), Frederick F. Boyce (clinical surgery), Manuel M. Garcia (clinical radiology), Chapman G. Johnson (clinical obstetrics and gynecology), Otto Schales, D.Sc., (biochemistry), Walter S. Wilde, Ph.D., (physiology), and Willard R. Wirth (clinical medicine). New medical associate professorships were granted to Clarence T. Ray (medicine), Isidore L. Robbins (clinical medicine), John R. Snavely (medicine), and Alan G. C. White, Ph.D., (biochemistry). Raised to the rank of assistant professors were Elaine Louise Allen (clinical pediatrics), Jack R. Anderson (clinical otolaryngology), Roger B. Bost (clinical pediatrics), Louis S. Charbonnet (clinical surgery), John K. Hampton Jr., B.S., (physiology), Lee C. Schlesinger (clinical orthopedics), Charles C. Sprague (medicine), Harold G. Tabb (clinical otolaryngology), Walter G. Unglaub (medicine), Richard P. Vieth (clinical pediatrics), and Samuel Zurik (clinical otolaryngology). Dr. Clifford G. Grulee Jr., assistant professor of pediatrics, has been named director of the division of graduate medicine.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

MARYLAND

Hospital News.—Dr. Russell A. Nelson has been appointed director of the Johns Hopkins Hospital, Baltimore, to succeed Dr. Edwin L. Crosby, director of the hospital since 1946, who has resigned to become executive head of the Joint Commission for the Accreditation of Hospitals. Dr. Nelson since 1947 has been assistant to Lowell J. Reed, Ph.D., vice-president of the Johns Hopkins University and Hospital.

Narcotic Violation.—Dr. John C. Stewart of Baltimore pleaded not guilty in the state court at Baltimore to a violation of the Uniform Narcotic Drug Act of Maryland and on April 18 was found guilty and sentenced to serve three years. His sentence was suspended, and he was placed on probation, the provisions of which were that he should surrender his narcotic registration. He was also fined $1,000.

MASSACHUSETTS

Society News.—At the annual business meeting of the Massachusetts Heart Association, Dr. Lewis M. Hurxthal of Newton was elected president and Dr. James C. McAdams of Fall River, vice-president. Dr. Benedict F. Massell of Brookline and Mr. Charles E. Cotting of Boston were reelected secretary and treasurer, respectively.

Dr. Hertig Appointed Shattuck Professor.—Dr. Arthur T. Hertig of Boston has been appointed Shattuck Professor of Pathological Anatomy at Harvard Medical School, Boston, to succeed Dr. S. Burt Wolbach, who held that office from 1922 until his retirement in 1947. In 1949 Dr. Hertig and Dr. John Rock, Brookline, won the award of the American Gynecological Society for their study of the development of the early human ovum. Dr. Hertig has been teaching and conducting research in pathology at Harvard Medical School and its associated hospitals since 1932, except for two years (1934-1936) when, as a national research fellow at the Carnegie Institution of Washington, D. C., he was engaged in studying the pathology of embryos. He has been professor of pathology at the Boston Lying-In Hospital and the Free Hospital for Women, Brookline, since 1948 and also has served as consultant to the U. S. Naval Hospital at Chelsea and to the Armed Forces Institute of Pathology.

MISSOURI

Dr. Oscar V. Batson Honored.—The University of Missouri, Columbia, recently conferred the honorary degree of doctor of science on Dr. Oscar V. Batson, professor of anatomy and chairman of the department in the Medico-Chirurgical College, Graduate School of Medicine, University of Pennsylvania, Philadelphia, since 1928, and also assistant professor of otolaryngology at the University of Pennsylvania School of Medicine. Dr. Batson served as instructor in pathology at the University of Missouri School of Medicine, St. Louis, and as instructor in anatomy at the University of Wisconsin, Madison. He later became professor of anatomy at the University of Cincinnati and also at the Ohio College of Dental Surgery before accepting appointment in the Pennsylvania Graduate School of Medicine. He was awarded the Wilhelm Conrad Medal by the American Roentgen Ray Society in 1941 and the bronze medal by the American Medical Association in 1942.

NEW YORK

Society News.—Dr. Carlton E. Wertz, Buffalo, has been appointed chairman of the convention committee for the 1953 annual meeting of the Medical Society of the State of New York to be held next May at Hotel Statler, Buffalo, and Dr. William J. Orr, also of Buffalo, has been designated chairman of the local committee on arrangements.

Gift of Rare Book.—The Lancet Society, an organization of physician-specialists, has presented to the State University of New York College of Medicine at Syracuse a rare book containing the anatomical illustrations of Andreas Vesalius. This volume, given in memory of the late Dr. Henry H. Haft, one of the founders of the society, and clinical professor of medicine at the college of medicine, is one of a number of copies, published jointly by the New York Academy of Medicine and the Library of the University of Munich in Germany, with reproductions from the original wood blocks. These blocks were later destroyed in the bombing of Munich during World War II.

New York City

Hartford Memorial Chapel.—The Pauline A. Hartford Memorial Chapel, a new interdenominational chapel for the use of patients, their relatives, and the hospital staff, was recently dedicated at Columbia-Presbyterian Medical Center. The chapel, a gift of the late John A. Hartford, former board chairman of the Great Atlantic and Pacific Tea Company, is named in honor of his wife, who was a patient at Presbyterian Hospital shortly before her death in 1948.

Alumni Elections.—The Alumni Association of the New York University College of Medicine has elected as president Dr. Anthony S. Bogatko, assistant professor of clinical surgery of the College of Medicine. Dr. Harold Brandaleone, assistant clinical professor of medicine at the college and medical director of the Third Avenue Transit System, was named vice-president.——Dr. James W. Smith, clinical professor of ophthalmology, New York University Post Graduate Medical School, was recently elected vice-president of the Alumni Federation of New York University.

NORTH CAROLINA

Man of the Year.—Dr. Thomas Craven of Huntersville was named "Man of the Year" at a dinner of the Huntersville Lions Club. A scroll was presented by Dr. Edward R. Hipp of Charlotte, president of the Mecklenburg County Medical Society, of which Dr. Craven has been a member for more than 30 years. Dr. Craven has served several terms as mayor of Huntersville and is a member of the Mecklenburg Board of Health, the board of the Mecklenburg Tuberculosis Sanatorium, and the Huntersville School Board.

Postgraduate Program.—The 1952-1953 program of year-round postgraduate medical education at James Walker Memorial Hospital in Wilmington began July 1, with Dr. George T. Harrell, professor of internal medicine and director of the department, Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, as visiting chief. Licensed physicians and medical students are invited to attend the daily conferences held from 5 to 6 p. m., and the clinical pathological conference held each Wednesday at 7:30 p. m. No registration fee is required, but all physicians residing outside New Hanover County are requested to register at the information desk in the lobby of the James Memorial Hospital. During the remainder of 1952, the following visiting chiefs will participate in the program: Aug. 19-22, Dr. Robert L. Sanders, Memphis; Sept. 9-12, Dr. Robert B. Lawson, Winston-Salem; Oct. 7-10, Dr. Richard W. TeLinde, Baltimore; Nov. 4-7, Dr. Charles C. Lund, Boston; Dec. 2-5, Dr. Bradley L. Coley, New York.

OHIO

Doctor's Orchestra.—The Doctor's Orchestra of Akron, under the directorship of Dr. Alexander S. McCormick, in June ended its 26th season with its 159th concert. The next season will open with rehearsals in October.

Centennial of Death of Daniel Drake.—Cincinnati recently observed the centennial of the death of Dr. Daniel Drake, the first physician in Cincinnati to receive a diploma (1805). Dr. Drake founded what is now known as the Cincinnati General Hospital and the University of Cincinnati College of Medicine, serving as its first professor of medicine. He was a pioneer in medical journalism west of the Allegheny mountains, notable among his contributions being the treatise on "The Principal Diseases of the Interior Valleys of North America."

Dr. Marion A. Blankenhorn, professor and chairman, department of internal medicine, University of Cincinnati College of Medicine, was the principal speaker at the Drake tombstone in Spring Grove Cemetery, where a bronze plaque was dedicated, replacing the worn inscription. At a banquet at the Vernon

Manor Hotel, Dr. Frank H. Mayfield, immediate past president, presented to Dr. Cecil Striker, president of the Academy of Medicine, the Daniel Drake gavel, made from the doorsill of Drake's home on Third Street.

A resolution, previously adopted, provides that the new hospital building at the Hamilton County Home is named the Daniel Drake Memorial Hospital. Honorary degrees, conferred by President Raymond Walters of the University of Cincinnati on distinguished guests Dr. Ralph Major of Kansas City, Kan., and Dr. Emmet F. Horine of Brooks, Ky., were previously reported in The Journal. The *Cincinnati Journal of Medicine* entitled its June, 1952, issue the "Daniel Drake Memorial Edition."

PENNSYLVANIA

Physicians Needed.—The Philadelphia Quartermaster Depot, 2800 South 20th Street, Philadelphia, is in need of a full-time (40 hour week) civilian physician. The annual salary is $5,060. For information telephone Mr. Arthur J. Jones, HO 5-2000. A physician is also needed for a summer camp at Pocono Highland, July 27 to Aug. 27. For information telephone Mr. Weinberg, RI 6-0748.

Society News.—The following medical schools of Pennsylvania have made favorable reply to a resolution approved by the House of Delegates of the Medical Society of the State of Pennsylvania recommending the establishment of departments of physical medicine and rehabilitation at the undergraduate and graduate level: the University of Pennsylvania School of Medicine, Temple University School of Medicine, Woman's Medical College of Pennsylvania, Jefferson Medical College, and Hahnemann Medical College, all of Philadelphia, and the University of Pittsburgh School of Medicine.

Philadelphia

Isotope Laboratory.—The Goldman Isotope Laboratory of the Hahnemann Medical College and Hospital of Philadelphia was officially dedicated June 4 at a banquet in honor of Mr. William Goldman, a trustee of the college, who made possible the establishment of the laboratory. Ralph T. Overman, Ph.D., chief of the training section of the Oak Ridge Institute for Nuclear Studies, was the principal speaker.

Pittsburgh

Society Election.—At the meeting of the Pittsburgh Roentgen Society held June 11, the following officers were elected: Dr. William T. Rice, president; Dr. Joseph E. Malia, vice-president; Dr. Donald H. Rice, secretary; Dr. Newton Hornick, treasurer; and Dr. Samuel G. Henderson, counsellor to the American College of Radiology.

SOUTH DAKOTA

Indian Agency Agreement.—The Bureau of Indian Affairs and the South Dakota State Medical Association have entered into an agreement for use of the fee schedule approved by the association, under which medical services of private physicians will be provided. This agreement is for patients referred to private physicians by the superintendent of the reservation involved and for emergency treatment of eligible Indians who may need care away from the reservation. Authorizations issued by the superintendent of the unit involved must accompany each patient referred to private physicians.

WASHINGTON

Haverhill Fever.—Haverhill fever (epidemic arthritic erythema) has been reported in Clark County, Washington. Two cases have been proved by agglutination in high dilution. Discovery of these cases has been credited to Dr. Edward C. Heyde of Vancouver.

Society News.—At the meeting of the Washington State Obstetrical Association, May 3, in Vancouver, B. C., Dr. Clarence W. Knudson of Seattle was elected president; Dr. Kenneth E. Norris of Yakima, vice-president; and Dr. Lincoln B. Donaldson of Seattle, secretary-treasurer.

WEST VIRGINIA

Joint Health and Welfare Meeting.—The West Virginia Welfare Conference, Inc., and the West Virginia Public Health Association will hold a joint meeting at Jackson's Mill, Aug. 22. Dr. Fred J. Holter, Ph.D., graduate advisor in the school of physical education at West Virginia University, will discuss "Community Health Councils." Mr. H. William Millington of Charleston, executive secretary of the Kanawha Welfare Council, will speak on welfare councils; and Mr. Charles Lively, executive secretary, West Virginia State Medical Association, will present a résumé of the association's fifth annual rural health conference held in June. The joint meeting of the 28 member board of the Welfare Conference and the 16 member council of the PHA, the first in the history of the two organizations, has been arranged to correlate the related programs of the two groups.

GENERAL

Society Election.—At the annual meeting of the American Laryngological Association in Toronto, Canada, May 23-24, Dr. Louis H. Clerf, Philadelphia, was elected president; Dr. J. Mackenzie Brown, Los Angeles, first vice-president; Dr. Edwin N. Broyles, Baltimore, second vice-president; Dr. Harry P. Schenck, New York, secretary; and Dr. Gordon F. Harkness, Davenport, Ia., treasurer.

Summer Course in Gerontology.—The New York University summer school at Chautauqua, N. Y., offers a workshop on problems of gerontology and adult education. The course will be given from July 28 to Aug. 8 under the co-direction of Prof. John Carr Duff of the N. Y. U. School of Education and Wilma T. Donahue, Ph.D., director of the division of gerontology at the University of Michigan Human Adjustment Center. Dr. Karl A. Menninger, Topeka, Kan., Harry A. Overstreet, professor emeritus of philosophy, College of the City of New York, and Mrs. Bonaro Overstreet are among the scheduled lecturers.

Outbreaks of Gastroenteritis.—According to the U. S. Public Health Service, an outbreak of food poisoning was reported by the California Department of Public Health. In a fruit pickers' camp about 75 persons became ill after eating sandwiches prepared from ground bologna and boiled eggs. Laboratory examination revealed Staphylococcus albus and alpha hemolytic Streptococcus. The Massachusetts Department of Health reports an outbreak of gastroenteritis in which 84 of 125 persons became ill after a high school banquet. Examination revealed Salmonella typhimurum in nine cases. Another outbreak occurred two weeks later after a banquet served by the same caterer in another high school.

Traffic Accidents in May.—According to the National Safety Council, more Americans were killed by automobiles this May than in any other May on record. The death toll of 3,160 was 12% over May in 1951. The previous high for May was 3,066 deaths in 1937. The May toll boosted the average increase for the first five months of this year to 4% more than for the comparable period last year. Of 42 states reporting for May, 30 had more deaths than in May a year ago, and 12 had fewer.

In contrast with the sharp increase in deaths reported by the states, 441 reporting cities had 5% fewer deaths than in May of last year. Of the 441 cities, 308 reported no traffic deaths in May. The three largest were Providence, R. I. (248,700); Hartford, Conn. (177,400), and New Haven, Conn. (164,400). For five months 156 still had perfect records. The three largest were: Little Rock, Ark. (102,200); Davenport, Ia. (74,500), and Evanston, Ill. (73,600).

Research at Brookhaven National Laboratory.—One hundred and fifty-four visiting scientists and students will participate in the research program at Brookhaven National Laboratory, Upton, L. I., N. Y., this summer, according to Robert A. Patterson, Ph.D., assistant director of the laboratory. This is nearly as many as the total for the 1950 and 1951 summer programs. The summer program will place Brookhaven's special research facilities in the field of atomic energy at the disposal of scientists and students. Of the 1952 group, 63 are scientists who will participate in research projects related to work they conduct at 35 different universities, colleges, or industrial organizations which they represent. Ninety-one are either graduate students or undergraduates from 38 different universities and colleges, who will spend the summer as research assistants in study of atomic energy. Many of the scientists will work in the biology or reactor departments. Brookhaven is the northeastern center for nuclear research and development operated under contract with the AEC by Associated Universities, Inc., a nonprofit educational corporation sponsored by nine leading eastern universities.

Prevalence of Poliomyelitis.—According to the National Office of Vital Statistics, the following number of reported cases of poliomyelitis occurred in the United States, its territories and possessions in the weeks ended as indicated:

Area	July 5, 1952	June 28, 1952	July 7, 1951
New England States			
Maine	2	3	..
New Hampshire	..	..	..
Vermont	1	1	1
Massachusetts	2	1	5
Rhode Island	..	..	..
Connecticut	2	2	5
Middle Atlantic States			
New York	21	21	17
New Jersey	2	3	5
Pennsylvania	4	4	7
East North Central States			
Ohio	45	22	7
Indiana	2	9	3
Illinois	4	8	16
Michigan	13	13	21
Wisconsin	3	10	12
West North Central States			
Minnesota	4	2	4
Iowa	27	20	..
Missouri	10	5	6
North Dakota	1	1	1
South Dakota	2	..	1
Nebraska	16	2	7
Kansas	17	19	5
South Atlantic States			
Delaware	..	..	1
Maryland	..	..	1
District of Columbia	..	1	3
Virginia	10	..	4
West Virginia	5	6	1
North Carolina	8	9	6
South Carolina	..	1	3
Georgia	11	16	11
Florida	10	10	10
East South Central States			
Kentucky	2	4	..
Tennessee	12	9	4
Alabama	17	8	22
Mississippi	26	26	16
West South Central States			
Arkansas	10	9	15
Louisiana	19	36	30
Oklahoma	26	22	21
Texas	186	206	58
Mountain States			
Montana	1	5	..
Idaho	1	..	..
Wyoming	..	2	1
Colorado	6	5	21
New Mexico	16	6	2
Arizona	7	2	7
Utah	1	..	2
Nevada	..	..	..
Pacific States			
Washington	11	14	2
Oregon	9	2	2
California	43	39	39
Territories and Possessions			
Alaska	..	..	..
Hawaii	18	6	..
Puerto Rico	..	..	..
Total	639	590	408

Meeting of Persons Subjected to Laryngectomy.—An Institute on Voice Pathology and the first International Meeting of Laryngectomized Persons are announced by the Cleveland Hearing and Speech Center, an affiliate of Western Reserve University. The meeting will be held Aug. 1 and 2 at Harkness Chapel, Bellflower and Ford Drive, on the campus of the university in Cleveland. It is sponsored by the American Cancer Society (Cuyahoga Unit), Cleveland Academy of Medicine, National Cancer Institute of the Institutes of Public Health, Cleveland Otolaryngological Club, the Lost Chord Club of Cleveland, Office of Vocational Rehabilitation of the Federal Security Agency, and the School of Medicine and Speech Department of Western Reserve University.

On Friday, meetings will be held by the sections on laryngeal disease and voice therapy, research on voice, and dysphonias. Saturday morning the sections on surgery in laryngectomy and on rehabilitation of laryngectomized persons will meet. The latter section will present a panel discussion on "Methods of Teaching Esophageal Speech." On Saturday afternoon the first International Meeting of Laryngectomized Persons will be held. Guest speaker at this meeting will be Dr. Charles S. Cameron of New York, president of the American Cancer Society.

The Cleveland Hearing and Speech Center announces that in 1951, 1,992 children and adults received direct therapy and 19,370 persons received indirect service in recreational rehabilitation, public education and training activities. Since 1945, when the center was formed as the result of a merger of the Cleveland Hearing Center and Western Reserve University Speech Clinic, more than 150 laryngectomized persons have been trained in esophageal speech and have participated in the center's Lost Chord Club.

College of Surgeons Meets in New York.—The annual clinical congress of the American College of Surgeons will be held at the Waldorf-Astoria, New York, Sept. 22-26 under the presidency of Dr. Alton Ochsner, New Orleans. On Tuesday a symposium on trauma will be held from 2 to 5 p. m., and at 8:30 p. m. there will be a motion picture symposium on surgery of the heart. On Wednesday a symposium on cancer will be held at 2 p. m. Five panel discussions in gynecology and obstetrics will be held Wednesday, Thursday, and Friday afternoons; problems of neurologic, thoracic, orthopedic, and plastic surgery, and urology will be considered in two panel discussions each on Friday afternoon; and sessions on ophthalmology and otolaryngology will be held Tuesday, Wednesday, and Thursday.

There will be 12 sessions of the forum on fundamental surgical problems, with about 155 papers. Attendance at seven postgraduate courses, to be given from 9 a. m. until noon Tuesday through Friday, is limited to persons registered for them. There will be courses in gynecology and obstetrics, management of fractures, nonmalignant diseases of the gastrointestinal tract, pre and postoperative care of the surgical patient, surgical aspects of pulmonary disease, trauma of the skull and spine, and cardiovascular surgery. The presidential dinner Monday will be followed by an assembly and the presidential meeting. Dr. Donald C. Balfour of Rochester, Minn., will give the 7th Martin Memorial Lecture on "The Evolution of Gastric Surgery." On Wednesday Dr. Sumner L. Koch, Chicago, will give the annual trauma oration and on Friday evening Sir Cecil Wakely of London, England, president of the Royal College of Surgeons, will give the fellowship address at the convocation. A cruise to the Island of Bermuda following the congress has been arranged. Participants will leave New York on Sept. 27 aboard the Queen of Bermuda, returning to New York Oct. 3. Information regarding rates may be obtained from Mr. Leon V. Arnold, 36 Washington Square West, New York 11.

EXAMINATIONS AND LICENSURE

NATIONAL BOARD OF MEDICAL EXAMINERS

NATIONAL BOARD OF MEDICAL EXAMINERS: *Part I*, Sept. 8-10. All centers where there are five or more candidates. Exec. Sec., Dr. John P. Hubbard, 225 S. 15th St., Philadelphia 2.

EXAMINING BOARDS IN SPECIALTIES

AMERICAN BOARD OF ANESTHESIOLOGY: *Oral*. Swampscott, Mass., Sept. 28-Oct. 1. Sec., Dr. C. B. Hickcox, 80 Seymour St., Hartford 15.

AMERICAN BOARD OF DERMATOLOGY AND SYPHILOLOGY: *Written*. Various Centers, September 1953. Final date for filing applications was May 1. All candidates are now required to take the written examination. *Oral*. Philadelphia, October 1953. Ex. Sec., Miss Janet Newkirk, 66 East 66th St., New York 21.

AMERICAN BOARD OF INTERNAL MEDICINE: *Written*. Various centers and overseas, Oct. 20. Closing date for the acceptance of applications for the written examination was May 1. *Oral*. San Francisco, Sept. 3-6; Philadelphia, Nov. 13-14 and Washington, D. C., Nov. 17-18. Exec. Sec.-Treas., Dr. William A. Werrell, 1 West Main Street, Madison 3.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY: *Written*. Various Centers, Feb. 6, 1953. Final date for filing applications is Nov. 1. Sec., Dr. R. L. Faulkner, 2105 Adelbert Road, Cleveland 6.

American Board of Ophthalmology: *Written*. Various Centers, January 1953. Final date for filing applications was July 1. *Practical*. Chicago, Oct. 6-10 and New York City, June 6-10, 1953. Sec., Dr. Edwin B. Dunphy, 56 Ivie Road, Cape Cottage, Maine.

American Board of Orthopaedic Surgery: The deadline for the receipt of applications for the 1953 Part II examinations is Aug. 15. Sec., Dr. Harold A. Sofield, 122 S. Michigan Ave., Chicago.

American Board of Otolaryngology: *Written*. Chicago, Oct. 6-10. *Oral*. Chicago, Oct. 6-10 and April 21-25, 1953. Sec., Dr. Dean M. Lierle, University Hospital, Iowa City.

American Board of Pathology: *Written and Oral*. Pathologic Anatomy and Clinical Pathology examination. Chicago, Oct. 9-11. Sec., Dr. William B. Wartman, 303 E. Chicago Ave., Chicago 11.

American Board of Pediatrics: *Oral*. Chicago, Oct. 24-26; Boston, Dec. 5-7. Exec. Sec., Dr. John McK. Mitchell, 6 Cushman Road, Rosemont, Pa.

American Board of Preventive Medicine and Public Health: Cleveland, Oct. 18-20. Sec., Dr. E. L. Stebbins, 615 N. Wolfe St., Baltimore.

American Board of Proctology: *Oral and Written*. Philadelphia, Sept. 27-28. Sec., Dr. Louis A. Buie, 102-110 Second Ave., S.W., Rochester, Minn.

American Board of Psychiatry and Neurology: New York City, Dec. 15-16. Final date for filing applications is Sept. 15. Sec.-Treas., Dr. David A. Boyd, Jr., 102-110 Second Ave. S.W., Rochester, Minn.

American Board of Radiology: *Oral*. Dec. 1-7. Final date for filing application was June 1. Sec., Dr. B. R. Kirklin, 102-110 Second Ave., S.W., Rochester, Minn.

American Board of Surgery: *Written*. Various Centers. October 1952. Final date for filing application was July 1. *Written*. Various Centers, March 1953. Final date for filing application is Dec. 1. Sec., Dr. J. Stewart Rodman, 225 S. 15th St., Philadelphia.

American Board of Urology: *Written*. Various Centers, December. *Oral*. Chicago, Feb. 7-11. Sec., Dr. Harry Culver, 30 Westwood Road, Minneapolis 16, Minn.

Board of Thoracic Surgery: *Written*. Various Centers, Sept. 5. Sec., Dr. William M. Tuttle, 1151 Taylor Ave , Detroit 2.

MEETINGS

Alaska Territorial Medical Association, Anchorage, Aug. 21-23. Dr. William P. Blanton, P. O. Box 2569, Juneau, Secretary.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, The Homestead, Hot Springs, Va., Sept. 4-6. Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Texas, Secretary.

American Congress of Physical Medicine, Roosevelt Hotel, New York, Aug. 25-29. Dr. Frances Baker, One Tilton Ave., San Mateo, Calif., Secretary.

American Hospital Association, Philadelphia, Sept. 15-18. Mr. George Bugbee, 18 East Division St., Chicago 10, Executive Director.

American Physiological Socety, New Orleans, Sept. 4-6. Dr. E. F. Adolph, University of Rochester School of Medicine and Dentistry, Rochester, N. Y., Secretary.

American Society of Maxillofacial Surgeons, Hotel del Coronado, San Diego, Calif., Sept. 10-13. Dr. Casper M. Epsteen, 25 East Washington St., Chicago 2, Secretary.

Biological Photographic Association, Hotel New Yorker, New York, Sept. 10-12. Mr. Lloyd E. Varden, Pavelle Color, Inc., 533 W. 57th St., New York 19, Secretary.

Medical Society of Delaware, Rehoboth, Sept. 8-10. Dr. Andrew M. Gehret, 822 North American Bldg., Wilmington, Secretary.

Montana Medical Association, Florence Hotel, Missoula, Sept. 18-21. Mr. L. R. Hegland, 104 North Broadway, Billings, Executive Secretary.

National Medical Association, Chicago, Aug. 11-15. Dr. John T. Givens, 1108 Church St., Norfolk 10, Va., General Secretary.

Neurosurgical Society of America, The Cloister, Sea Island, Ga., Sept. 18-20. Dr. C. D. Hawkes, 22 North Manassas St., Memphis 5, Tenn., Secretary.

Nevada State Medical Association, Reno, Aug. 21-23. Dr. William A. O'Brien III, 505 Chestnut St., Reno, Secretary.

New Hampshire Medical Society, Mt. Washington Hotel, Bretton Woods, Sept. 7-9. Dr. Deering G. Smith, 44 Chester St., Nashua, Secretary.

North Pacific Pediatric Society, Challenger Inn, Sun Valley, Idaho, Sept. 19-20. Dr. S. Gorham Babson, 1107 S.W. 13th Ave., Portland 5, Ore., Secretary.

North Pacific Society of Internal Medicine, Sun Valley, Idaho, Sept. 19-20. Dr. Robert L. King, 1115 Terry Ave., Seattle 1, Wash., Secretary.

North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, Texas, Sept. 17. Dr. E. Aubrey Cox, Hamilton Bldg., Wichita Falls, Texas, Chairman.

Reno Surgical Society Conference, Reno, Nevada, Aug. 21-23. Dr. Olin C. Moulton, 130 N. Virginia St., Reno, Nevada, Chairman.

Rocky Mountain Radiological Society, Denver, Aug. 7-9. Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Neb., Secretary.

U. S. Chapter, International College of Surgeons, Conrad Hilton Hotel, Chicago, Sept. 2-5. Dr. Arnold S. Jackson, 1516 Lake Shore Drive, Chicago, Executive Secretary.

Utah State Medical Association, Salt Lake City, Sept. 4-6. Dr. T. C. Weggeland, 42 S. Fifth East St., Salt Lake City, Secretary.

Vermont State Medical Society, Mt. Washington Hotel, Bretton Woods, N. H., Sept. 7-9. Dr. James P. Hammond, 128 Merchants Row, Rutland, Secretary.

Washington State Medical Association, Olympic Hotel, Seattle, Sept. 13-17. Dr. Bruce Zimmerman, 338 White-Henry-Stuart Bldg., Seattle, Secretary.

INTERNATIONAL

American Congress of Industrial Medicine, Rio de Janeiro, Brazil, Sept. 20-28. General Secretarial Office: Calle Arenales No. 981 Buenos Aires, Argentina.

Australasian Medical Congress, Melbourne Victoria, Australia, Aug. 22-30. Dr. C. H. Dickson, Medical Society Hall, 426 Albert St., East Melbourne, Victoria, Australia, General Secretary.

European Congress of Cardiology. University of London, Bloomsbury, W.C.1, England, Sept. 10-12. Dr. K. Shirley Smith, 35 Wimpole St., London, W.1, England, Secretary.

European Society of Cardiovascular Surgery, Strasbourg, France, Oct. 3-4. Sir James Learmonth, Department of Surgery, University New Buildings, Edinburgh 8, Scotland.

Interamerican Cardiological Congress, Buenos Aires, Argentina, Aug. 31-Sept. 7. Dr. Blas Moia, Larrea 1132, Buenos Aires, Argentina, Secretary-General.

Inter-American Congress of Public Health, Havana, Cuba. Sept. 26-Oct. 1. Pan-American Sanitary Bureau, 2001 Connecticut Ave., Washington, D. C.

Inter-American Congress of Radiology, Mexico City, Mexico, Nov. 2-8. Dr. Guido Torres Martinez, Marsella No. 11, Mexico, D.F., Secretary-General.

International Congress of Anthropology and Ethnology, Vienna, Austria, Sept. 1-8. For information write: Secretaries, Royal Anthropological Institute, 52 Upper Bedford Place, London, W.C.1, England.

International Congress on Diseases of the Chest, Rio de Janeiro, Brazil, Aug. 24-30. Mr. Murray Kornfeld, 112 East Chestnut St., Chicago 11, Illinois, Executive Secretary.

International Congress of Haematology, Mar del Plata, Argentina, Sept. 21-26. Dr. Carlos Reussi, Anchorena 1710, Buenos Aires, Argentina, Secretary.

International Congress of the History of Medicine, Nice-Cannes, France, and Monaco, Sept. 8-14. Dr. F. A. Sondervorst, 124 Avenue des Allies, Louvain, Belgium, Secretary-General.

International Congress on Hydatid Disease, Santiago, Chile, Nov. 21-24. Organizing Committee, P. O. Box 9183, Santiago, Chile.

International Congress of Internal Medicine, Friends House, London, N.W.1, Eng. Sept. 15-18. Sir Harold Boldero, 12 Pall Mall East, London, S.W.1, England, Secretary.

International Congress of Medical Press, Venice, Italy, Oct. 4-5. Dr. Bruno de Biasio, S. Marco 4646, Venice, Italy, Secretary.

International Congress on Medical Records, London, England, Sept. 7-12. Miss Gwen Perkins, 120 C Street S.E., Washington, D. C., Chairman.

International Congress of Military Medicine and Pharmacy, Montevideo, Uruguay, March 1-7, 1953. Dirreccion General del Servico de Sanitad Militar, 8 de Octubre y Mariano Moreno, Montevideo, Uruguay, Secretariat of the Congress.

International Congress on Neuropathology, Rome, Italy, Sept. 8-13. Dr. Armando Ferraro, 722 W. 168th St., New York, N. Y., U. S. A., Secretary General.

International Congress of Otorhinolaryngology, Amsterdam, Netherlands, June 8-15, 1953. Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam, Netherlands, Secretary.

International Congress of Urology, The Waldorf-Astoria, New York, N. Y., U. S. A., Sept. 15-18. Dr. John A. Taylor, 2 East 54th St., New York, N. Y., U. S. A., Secretary-General.

International Hospital Congress, London, England, May 25-30, 1953. Capt. J. E. Stone, 10 Old Jewry, London, EC2, England, Hon. Secretary.

International Study Conference on Child Welfare, Bombay, India, Dec. 5-12. For information write: All India Save the Children Committee, 5 Carmichael Road, Bombay, India.

International Union Against Tuberculosis, Rio de Janeiro, Brazil, Aug. 24-27. Prof. Etienne Bernard, 66 Blvd. St. Michel, Paris 6e, France, Secretary-General.

Medical Women's International Association, Vichy, France, Sept. 12-16. Dr. Margaret M. Wurts, 27 East Wellesley Road, Montclair, N. J., U. S. A., National Secretary.

Neuroradiologic Symposium, Stockholm, Sweden, Sept. 17-20. Docent Ake Lindbom, Symposium Neuroradiologicum, Serafimerlasarettet, Stockholm K., Sweden, Secretary.

World Congress of Jewish Physicians, Jerusalem, Israel, Aug. 10-14. Secretariat: ℅ Israel Medical Association, Rehov Mesilat Yesharim, Beth Kovatz, Jerusalem, Israel.

World Congress of Medicine, Montecatini, Italy, Oct. 16-18. Secretariat: Corso Trieste, 65, Rome, Italy.

World Federation of Mental Health, Brussels, Belgium, Aug. 25-Sept. 1. Dr. J. R. Rees, 19 Manchester St., London, W.1, England, Secretary.

World Medical Association, Athens, Greece, Oct. 12-16. Dr. Louis H. Bauer, 2 East 103d St., New York 29, N. Y., Secretary General

DEATHS

Ireland, Merritte Weber ⊕ major general, U. S. Army, retired, Washington, D. C., formerly, for more than 12 years, Surgeon General of the Army, died in Walter Reed Hospital July 5, aged 85. Born in Columbia City, Ind., the son of a physician, General Ireland received his first degree in medicine from the Detroit College of Medicine in 1890. He received a second degree from Jefferson Medical College in Philadelphia the following year and then was commissioned an assistant surgeon in the Army. He served in Cuba during the Spanish-American War. During the Mexican expedition led by Gen. John J. Pershing he was post surgeon at Fort Sam Houston, Texas and later went to France with General Pershing, where he headed the medical service of the American Expeditionary Force. General Ireland was largely responsible for the development of modern concepts of field medicine during World War I. He served as Surgeon General from Oct. 4, 1918, until his retirement from the Army on May 31, 1931. His term of office saw the creation of the Army Medical Center on the grounds of Walter Reed Army Hospital. Among General Ireland's awards were the Distinguished Service Medal, Commander of the Legion of Honor of France, Companion of the Order of the Bath of Great Britain, and Grand Officer of the Order of Polonia Restituta. In 1945 he was awarded the William Freeman Snow Medal for distinguished service to humanity from the American Social Hygiene Association, and in April, 1945, received a citation for distinguished service to humanity from the Medical Society of the District of Columbia. General Ireland served as a delegate from the U. S. Army to the American Medical Association in 1908, 1909, and 1910, and later was a member of its Council on Medical Education and Hospitals. He was past president of the National Board of Medical Examiners, Association of the Military Surgeons of the United States, and the American College of Surgeons. He was formerly secretary of the Gorgas Memorial Institute of Tropical and Preventive Medicine, a fellow and ex-governor of the American College of Physicians, a fellow of the Royal College of Surgeons of Edinburgh, and a member of the American Psychiatric Association. He had been a director of the Columbia Hospital for Women and Lying-In Asylum, Garfield Memorial Hospital, and the United States Soldiers Home Hospital. He delivered the William Potter Memorial Lecture at Jefferson Medical College in 1939. In 1947 he resigned as president of the Army Mutual Aid Association after 18 years of service and as a director of that organization for 32 years. General Ireland received LL.D. degrees from Jefferson in 1919, Gettysburg (Pa.) College in 1922, and Syracuse (N. Y.) University in 1935 and an A.M degree from the University of Michigan in Ann Arbor in 1920.

LeBel, Louis Jean Baptiste ⊕ Nutley, N. J.; born in Nutley, N. J., Jan. 26, 1895; University of Virginia Department of Medicine, Charlottesville, 1921; specialist certified by the American Board of Dermatology and Syphilology; founder and a past president of the Northern New Jersey Dermatological Society; member of the American Academy of Dermatology and Syphilology; member of the professional staff, Council on Medical Education and Hospitals, American Medical Association in Chicago; at one time taught at Columbia University College of Physicians and Surgeons in New York; for many years affiliated with St. Mary's Hospital in Passaic, where he was president of the medical staff from 1939 to 1941 and where he died May 20, aged 57, of subacute bacterial endocarditis.

Curtis, Elmer Eaton ⊕ medical director, captain, U. S. Navy (retired), Vallejo, Calif.; born in Hiawatha, Kan., Aug. 7, 1877; St. Louis University School of Medicine, 1905; commissioned a lieutenant (jg) in the Medical Corps of the U. S. Navy on July 12, 1907; attained the rank of captain on July 1, 1929, and on Sept. 1, 1941, was placed on the retired list; during World War I served aboard the transports USS Powhatan and USS Mount Vernon, engaged in ferrying troops across the Atlantic; after attending the Naval War College, Newport, R. I., in 1933 was assigned to the staff of the commander, Battle Force, U. S. Fleet, as force medical officer; recalled to active duty at the outbreak of World War II, and assigned as medical officer in command of the Naval Dispensary, Treasure Island, Calif.; in 1942, assumed command of the Naval Hospital, Treasure Island, San Francisco; was released from active duty in 1945 and returned to the retired list; died in the Naval Hospital, Mare Island, May 5, aged 74, of lymphosarcoma of small intestine, peritonitis and bronchopneumonia.

Levy, Joseph ⊕ New Rochelle, N. Y.; born in New York April 10, 1901; Johns Hopkins University School of Medicine, Baltimore, 1927; specialist certified by the American Board of Internal Medicine; fellow of the American College of Physicians; member of the Latin American Congress of Physical Medicine, Aero Medical Association, International Society of Internal Medicine, American Academy of Sciences, American Geriatrics Society, Israel Medical Association, Westchester Heart Association, and New Rochelle Hospital Association; member of the Phi Delta Epsilon Fraternity and a founder of Tau Alpha Omega, a fraternity at City College; during World War II a flight surgeon with the rank of major in the Army Medical Corps, serving in Africa and the Philippines; adjunct physician, Montefiore Hospital for Chronic Diseases in New York; attending physician, New Rochelle Hospital; died in San José, Costa Rica, recently, aged 50, of coronary occlusion, after delivering a paper at the fourth annual convention of the Latin American Congress of Physical Medicine.

Jostes, Frederick Augustus ⊕ St. Louis; born in St. Louis Aug. 14, 1895; Washington University School of Medicine, St. Louis, 1920; specialist certified by the American Board of Orthopaedic Surgery; member of the American Orthopaedic Association, Clinical Orthopaedic Society, and the American Academy of Orthopaedic Surgeons; fellow of the American College of Surgeons; president of the Missouri Society for Crippled Children and Adults and first vice-president of the Mississippi Valley Medical Society; served during World War II; assistant professor of clinical orthopedic surgery at his alma mater; affiliated with St. Louis County Hospital in Clayton, Bernard Free Skin and Cancer Hospital, Evangelical Deaconess, St. Louis Children's, and Jewish hospitals, and the Barnes Hospital, where he died May 19, aged 56, of lymphosarcoma.

White, Vivian Parley ⊕ Salt Lake City; born in Salt Lake City, Sept. 12, 1896; Harvard Medical School, Boston, 1923; specialist certified by the American Board of Otolaryngology; member of the American Academy of Ophthalmology and Otolaryngology and the Pacific Coast Oto-Ophthalmological Society, of which he had been vice-president; immediate past president of Utah State Medical Association; past president of the Utah State Ophthalmological Society; clinical lecturer in surgery (otolaryngology) at the University of Utah College of Medicine, where he had been instructor in anatomy of the head and neck from 1925 to 1927; affiliated with Salt Lake County General Hospital and Dr. W. H. Groves Latter-Day Saints Hospital, where he died May 13, aged 55, of carcinoma of the intestine.

Albanese, Nicholas Anthony ⊕ Columbus, Ohio; Starling-Ohio Medical College, Columbus, 1914; served during World Wars I and II; affiliated with St. Ann's Maternity Hospital; died April 26, aged 59, of myocardial infarction.

Bartholomew, Alfred Castle ⊕ Fort Wayne, Ind.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1901; served during World War I; died in Methodist Hospital April 27, aged 73, of myocardial infarction.

Bennett, Charles Lenzo ⊕ Kalamazoo, Mich.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1904; past president of the Kalamazoo Academy of Medicine; formerly practiced in Gobleville, where he had been on the village council and school board; formerly vice-president and

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⊕ Indicates Member of the American Medical Association.

director of the State Bank in Gobles; affiliated with Bronson Hospital and Borgess Hospital, where he died April 22, aged 76, of pneumonia and arteriosclerotic heart disease.

Denison, Edward Lyman, Jensen Beach, Fla.; Northwestern Medical College, St. Joseph, Mo., 1892; Illinois Medical College, Chicago, 1895; formerly practiced in Chicago, where he was affiliated with St. Luke's Hospital; died April 27, aged 82, of coronary occlusion.

Elias, Francis Leander ⊕ Wymore, Neb.; University of Kansas School of Medicine, Kansas City, 1910; past president of Gage County Medical Society; affiliated with Lutheran Hospital in Beatrice, where he died March 3, aged 68, of coronary thrombosis.

Flowers, Earl Newton ⊕ Clarksburg, W. Va.; Johns Hopkins University School of Medicine, Baltimore, 1919; served as city health officer; veteran of World War I; died April 12, aged 57.

Gaskill, Henry Kennedy ⊕ Hendersonville, N. C.; Jefferson Medical College of Philadelphia, 1906; member of the Medical Society of the State of Pennsylvania; formerly practiced in Philadelphia, where he was professor of dermatology at the Woman's Medical College of Pennsylvania; served during World War I; died April 11, aged 79 .

Gulbrandsen, Lyder Oliver ⊕ Viroqua, Wis.; Marquette University School of Medicine, Milwaukee, 1927; served during World Wars I and II; died in La Crosse, recently, aged 51, of coronary occlusion.

Hamer, Clemen ⊕ Glendale, Calif.; College of Medical Evangelists, Loma Linda and Los Angeles, 1924; affiliated with Behrens Memorial Hospital, Physicians and Surgeons Hospital, and Glendale Sanitarium and Hospital, where he was a member of the board of directors and where he died April 1, aged 68, of pneumonia.

Hemstead, Werner ⊕ Minneapolis; Omaha Medical College, 1882; died in Rochester, Minn., March 11, aged 91, of bronchopneumonia and cerebral infarcts.

Henriksen, Henry George, Northfield, Minn.; Bennett College of Eclectic Medicine and Surgery, Chicago, 1897; also a graduate in pharmacy; died March 30, aged 82, of carcinoma of the lungs.

Herzog, Charles E. ⊕ Stormville, N. Y.; Long Island College Hospital, Brooklyn, 1922; connected with the Department of Correction; killed April 23, aged 53, when his automobile collided with a truck.

Hirst, Jesse Watson, St. Petersburg, Fla.; Jefferson Medical College of Philadelphia, 1893; for many years a Presbyterian medical missionary in Korea, and at one time court physician to the emperor; died in St. Anthony's Hospital April 28, aged 88, of cerebral vascular accident.

Hively, Howard Dayton ⊕ Parkersburg, W. Va.; University of Louisville (Ky.) Medical Department, 1912; formerly medical director of the West Virginia Coal and Coke Corporation in Omar; at one time practiced in Charleston, where he was affiliated with Charleston General Hospital; died April 7, aged 66, of coronary thrombosis and arteriosclerosis.

Hodgson, Thomas Cady ⊕ Berlin, Conn.; University of Toronto Faculty of Medicine, Toronto, Canada, 1894; served as health officer and medical examiner; died in Hartford April 21, aged 82, of internal carotid thrombosis.

Hofmann, Erich Manfred ⊕ Wooster, Ohio; Thüringische Landesuniversität Medizinische Fakultät, Jena, Thuringia, Germany, 1919; died March 4, aged 63.

Hopper, Henry Paine, Saucier, Miss.; Kentucky School of Medicine, Louisville, 1904; for many years county physician; died in the Veterans Administration Hospital, Biloxi, April 3, aged 78, of pulmonary edema and arteriosclerotic heart disease.

Hutchings, Charles Wyatt ⊕ Syracuse, N. Y.; Syracuse University College of Medicine, 1926; member of the American Psychiatric Association; served during World War II; assistant director of the Syracuse State School of the New York State Department of Mental Hygiene, Fairmount; formerly on the staff of the Marcy (N. Y.) State Hospital and assistant director of the Manhattan State Hospital, Wards Island, New York City; died in Syracuse Memorial Hospital April 15, aged 52, following a gastrectomy.

Imbrie, Clarence Elwood ⊕ Butler, Pa.; Baltimore Medical College, 1904; affiliated with Butler County Memorial Hospital; died April 13, aged 77, of coronary occlusion.

MacDonald, Aeneas E. ⊕ Minneapolis; Queen's University Faculty of Medicine, Kingston, Canada, 1901; affiliated with St. Mary's and Asbury hospitals; died April 11, aged 76, of cerebral hemorrhage and arteriosclerosis.

McGarvah, Joseph Andrew ⊕ Detroit; Detroit College of Medicine, 1905; affiliated with Woman's Hospital; died March 20, aged 69, of carcinoma of the bile ducts.

Madison, William Davis ⊕ Peoria, Ill.; Drake University College of Medicine, Des Moines, 1912; affiliated with Florence Crittenton Home and Methodist Hospital, where he died April 28, aged 69, of coronary thrombosis.

Marcussen, Robert Monroe ⊕ Menlo Park, Calif.; Cornell University Medical College, New York, 1943; city councilman; served during World War II; Commonwealth Fund Fellow in Medicine at his alma mater, 1947-1948; in 1949-1950 Fellow in Neuropsychiatry at Stanford University School of Medicine in San Francisco, where he had been clinical instructor in medicine; died April 13, aged 33.

Martin, Solomon Claiborne Jr., West Palm Peach, Fla.; Barnes Medical College, St. Louis, 1893; died recently, aged 81, of myocardial disease and congestive heart failure.

Massoud, Mittry Abi ⊕ Pineora, Ga.; Maryland Medical College, Baltimore, 1904; died April 30, aged 76, of coronary heart disease.

Maxson, John Child, Campbell, Calif.; University Medical College of Kansas City, Mo., 1893; served during World War I; died in San Jose March 29, aged 85, of mesenteric thrombosis.

Merrill, Adelbert Samuel ⊕ Manchester, N. H.; Tufts College Medical School, Boston, 1908; specialist certified by the American Board of Radiology; member of the American Roentgen Ray Society, New England Roentgen Ray Society and the American College of Radiology; affiliated with Elliott Hospital; died in Concord April 7, aged 76.

Mitchell, Robert Lee ⊕ Vinita, Okla.; University of Arkansas School of Medicine, Little Rock, 1904; served overseas during World War I; affiliated with Eastern State Hospital; at one time on the staff of Veterans Administration Hospital in Houston, Texas; for many years affiliated with Veterans Administration Hospital in Muskogee, where he died May 15, aged 76, of coronary thrombosis.

Moncure, Turner Ashby, Baltimore; Medical College of Virginia, Richmond, 1912; clinic chief of the Presbyterian Eye, Ear and Throat Charity Hospital; died in the Lutheran Hospital April 23, aged 65, of arteriosclerotic cardiovascular disease.

Simmons, James Franklin, Greenville, Miss.; Mississippi Medical College, Meridian, 1907; on the staff of the Kings Daughters Hospital; died March 27, aged 68, of cerebral hemorrhage.

Smith, John McNeill ⊕ Rowland, N. C.; Jefferson Medical College of Philadelphia, 1908; died recently, aged 67, of cerebral hemorrhage and arteriosclerosis.

Townsend, Mary Ella, Atlantic City, N. J.; Woman's Medical College of Pennsylvania, Philadelphia, 1895; died in Camden March 11, aged 83, following an operation for cancer.

White, Thomas Francis ⊕ Baltimore; University of Maryland School of Medicine, and College of Physicians and Surgeons, Baltimore, 1920; served as health officer-at-large for Baltimore City; died March 6, aged 61, of coronary thrombosis.

Willson, Pleas, Newborn, Ga.; Atlanta College of Physicians and Surgeons, 1901; died March 14, aged 71, of cerebral hemorrhage and hypertension.

Young, Louise Sanford, El Paso, Texas; Illinois Medical College, Chicago, 1905; University of Louisville (Ky.) Medical Department, 1905; died April 20, aged 83, of arteriosclerosis.

GOVERNMENT SERVICES

NAVY

Construction Projects to Provide 2,300 Hospital Beds.—Plans to build a new permanent hospital at Norfolk, Va., and major permanent additions to existing hospitals at Great Lakes, Ill., and San Diego, Calif., have been announced by the Navy. The construction, which will begin next year, will provide a total of 2,300 additional beds. The largest of the projects will be a 1,000 bed addition to the naval hospital at San Diego. The addition will be a multistory structure and will include operating rooms, x-ray facilities, and messing areas.

The new hospital to augment medical facilities in the Norfolk area will contain 800 beds initially, with provision for expansion to 1,500 beds. The 500-bed addition to the naval hospital at the Great Lakes Naval Training Center will be a multistory permanent structure. The original hospital was built in 1911.

Study of Protein Metabolism.—Henry Borsook, professor of biochemistry at the California Institute of Technology, addressed the Naval School of Aviation Medicine at Pensacola on July 7 on "Newer Aspects of Protein Metabolism." Dr. Borsook was on his way to Paris, where he will lecture on that subject. While in Pensacola he completed initial planning with Captain Ashton Graybiel (MC), Director of Research, and Capt. James L. Rolland (MC), Commanding Officer, preparatory to setting up a cooperative study on protein metabolism sponsored by the California Institute of Technology and the U. S. Naval School of Aviation Medicine.

Personal.—Capt. C. W. Shilling (MC) director, research division, Bureau of Medicine and Surgery, was guest speaker at the annual open house of the Worcester Foundation for Experimental Biology June 14. Captain Shilling spoke on "The Usefulness of Useless Research." Captain Shilling also attended the dedication of the Aviation Medical Acceleration Laboratory at Johnsville, Pa., June 17.

AIR FORCE

Study of Human Adaptation to High Altitudes.—Brig. Gen. Otis O. Benson Jr., commandant of the Air Force School of Aviation Medicine, and a party of military and civilian scientists, have flown to a remote laboratory in Peru, where human adaptation to high altitudes is being investigated. The Institute of Andean Biology, an outpost of the University of San Marcos in Lima, is perched on the upper slopes of the Andes at the mining town of Morococha, one of the loftiest inhabited spots on earth. The town is 90 miles from Peru's sea-coast capital on a plateau 14,900 feet up.

Dr. Alberto Hurtado, the Peruvian physician who runs the Institute, is a Harvard graduate and former research worker at the Mayo Foundation. He has chosen the Indian descendants of the Incas who live on the towering heights of his native land for his special field of study.

The Indians at Morococha toil in the mines at altitudes up to 18,000 ft., where an Air Force flyer is required to wear an oxygen mask, and for relaxation play football in the hard-driving English style. The changes in their physiological processes after centuries of existence on those dizzy heights are of vital interest to the Air Force.

Dr. Hurtado holds a contract with the Air Force to learn what these changes are, and to search for possible ways in which they might be speeded up. It is to settle the final terms of this contract that General Benson's group is flying to Peru. The School of Aviation Medicine is the Air Force research organization which administers the high-altitude project. With the General are other specialists, among them Col. Paul A. Campbell, director of research at the aeromedical school. From the University of California at Berkeley, Dr. John H. Lawrence, director of the Donner Laboratory of Medical Physics, and William Siri, a research physicist, flew to Randolph Field to join the party. Also in the group is Dr. Peter Hoessly, assistant professor of medicine at the University of Utah.

VETERANS ADMINISTRATION

Increased Pay for Disabled Veterans.—Almost 120,000 blind, crippled and tuberculous veterans of all wars and peacetime service will receive increased payments in their compensation checks due Sept. 1, 1952, under Public Law 427 signed by the President June 30. Veterans who are not on the compensation rolls and are covered by the new law should apply at their nearest VA Regional Office.

Veterans who will receive the increased checks are those who have been awarded statutory allowances for the loss, or loss of use, of either or both hands or feet, for blindness, and for total deafness (in combination with total blindness). Veterans who are receiving a minimum monthly rate of compensation for arrested tuberculosis will also receive increased checks. Statutory allowances are paid in addition to the basic compensation rates, which are based on the degree of disability and range from $15.75 to $172.50.

The new law authorizes for the first time for World War II veterans and veterans with service since the beginning of the Korean conflict a minimum monthly rate for arrested tuberculosis and a statutory allowance for the loss, or loss of use, of a creative organ. Up to now these benefits have been available under existing law to certain World War I veterans only. Following are the increases for veterans disabled in wartime or since the Korean conflict started:

1. Monthly statutory allowance for the anatomical loss, or loss of use, of one hand or one foot, or blindness of one eye (having only light perception) increased from $42 to $47. This is payable in addition to monthly rate of compensation.

2. Monthly rate of compensation for the anatomical loss, or loss of use, of both hands, both feet, blindness, total deafness (in combination with total blindness), and various combinations of such disabilities increased from a range of $240 to $360, to a range of $266 to $400. Additional amounts are payable for a wife, children, or dependent parents in these cases and in all other service-connected cases in which the veteran's disability is rated 50% or more. The monthly rates of such additional compensation in totally disabled cases range from $21 to $91, depending on the number and type of dependents. The new law does not increase these additional amounts.

3. The minimum monthly rate of compensation for service-incurred tuberculosis that has been arrested is increased from $60 to $67 and extended to cover World War II veterans and veterans with service since the start of the Korean conflict.

4. Monthly statutory allowance for loss, or loss of use, of creative organ is increased from $30 to $47 and extended to cover World War II veterans and veterans with service since the start of the Korean conflict.

5. Certain seriously disabled World War I veterans, with service-connected disabilities, will receive increased monthly payments—from $198 to $220 for totally blind veterans; from $258 to $286 for totally blind veterans who have lost one or more limbs; and from $60 to $67 for veterans who need the regular aid and attendance of another person.

These new wartime rates also are payable for peacetime service if the specific disabilities were incurred in line of duty as the direct result of armed conflict or while engaged in extra hazardous service, including service under conditions simulating war. Rates of compensation for disabilities incurred during other peacetime service are 80% of wartime rates.

FOREIGN LETTERS

BRAZIL

International Congress of Surgery.—The International Congress of Surgery was held at Madrid, from May 19 to 25, and was attended by more than 2,000 surgeons of many different specialties, coming from 37 countries. Some 500 papers were presented, and films were shown to demonstrate various surgical techniques. For the first time in Europe, with the cooperation of Remington Rand Inc., color television was produced by the "vericolor" process, and these operations were transmitted on television from the "Cidade Universitaria" (University Town). At the closing ceremony, paying special homage to the memory of the great Spanish scientist Ramon y Cajal, the following speakers were heard: the minister of education, the dean of the university, Prof. Gregorio Marañon, and the secretary of the International Congress, Prof. Alfonso de la Fuente. During this ceremony the head of the Brazilian delegation, Prof. Rodolfo de Freitas, acknowledged the privilege of São Paulo being the place chosen for the 9th International Congress of Surgery and invited surgeons from all over the world to visit Brazil in 1954. At the meeting of the house of delegates it was decided that a building be constructed in São Paulo as a center for meetings of the members of the "Capítulo Brasileiro" (Brazilian chapter) and of the South American zone of the International College of Surgeons. Dr. Rudolph Nissen was elected president of the next congress, and among the future vice-presidents are the Brazilian surgeon Prof. Carlos Gama and Prof. Manuel Manzanolla from Mexico.

Ovarian Pregnancy.—A study on ovarian pregnancy was published by Drs. Domingos Delascio and Luidoso Credidio. They presented a complete historical review commencing with the De Sanctis Maurice case, which is considered to be the first recorded, in the year 1682. They recalled the work of Willigh, who insisted on the necessity for microscopic investigation for a diagnosis of the disease and the criticism of Lawson Tait about cases erroneously diagnosed. The English master referred to a case of a dermoidal tumor described as an ovarian pregnancy. The formulation of the four fundamental conditions for safe diagnosis of this ectopic implantation by Otto Spiegelberg in 1878 is mentioned. Drs. Delascio and Credidio record the study of Catherine van Tussembroeck, in the year 1899, in which, based on microscopic investigations, she was able to establish the diagnosis of ovarian pregnancy. The cause of ovarian pregnancy has been the object of much controversy. The authors believe that ovarian pregnancy may be caused by disturbance of two mechanisms: namely those of ovulation and implantation following pathological processes of the tubes and ovaries.

Cancer of the Esophagus.—A review of 82 cases of cancer of the esophagus and gastric cardia was presented by Prof. José Maria de Freitas and co-workers. The localization of tumor was as follows: interior third plus cardia, 46 cases; inferior third, 17; median third, 12; superior third, 3; and not specified, 4. The outstanding symptoms in these 82 cases consisted of: dysphagia, 69 times; vomiting and regurgitation, 57 times; pain at the inferior third of the retrosternal region, 52 times; and hemorrhage with hematemesis and melena, 10 times. All patients were noted to have lost weight, the loss being more than 10 kg. in the majority of cases.

X-ray examinations of the esophagus and stomach and endoscopic study of the esophagus, larynx, and bronchi are of the utmost importance for the diagnostic confirmation of cancer of the esophagus and cardia and the ultimate indication for surgical therapy. Good results were obtained through endotracheal anesthesia with total curarization, automatic respiration being controlled by the pulmotor; with surgical approach through left thoracotomy with resection of a rib; with esophageal-gastric or esophageal-jejunal anastomosis made with cotton sutures, far apart from each other, in two planes: those of the total inner layer being U-shaped and those of the external layer doubly anchored; systematic use of blood, about 500 cc. at each transfusion, alternating with saline, during the surgical proceedings; closed pleural drainage during 48 hours or more; esophageal-gastric intubation for 48 hours; and systematic use of penicillin and streptomycin locally and parenterally. Feeding is begun early, as soon as the 1st postoperative day, and consists of small amounts of liquid hourly until the 3rd day, reinforced with light foods the 10th postoperative day. Early passive and active exercise soon after the operation and early rising from bed, from the 3rd to the 5th postoperative day, are to be recommended.

The results obtained with surgical treatment (resection of esophagus and cardia) in 39 cases out of the 82 studied were as follows. Two patients who submitted to total resections of the esophagus lived through the immediate postoperative period, as did 19 of the 37 who submitted to transthoracic resection of the esophagus and cardia with immediate anastomosis. Among this same group, 18 died immediately. In the rest of the group, consisting of patients who underwent palliative operations, there were 18 deaths. Two patients died without having been operated on. Causes of death in 19 out of 39 patients who underwent resection were as follows: dehiscence of anastomosis 5, surgical shock 1, postoperative hemorrhage 3, cachexia 6, bronchopneumonia 2, and toxemia 1. The mortality rate tended to diminish in proportion to the speed with which the diagnosis was established and the rate that the authors' experience increased. Two patients underwent total resection of the esophagus six months and three years later, with cervical esophagostomy and gastrostomy, respectively. From those 19 patients who underwent radical operation, 2 persons lived two months, 1 lived three months, and 2 lived six months. This last one has been feeling well and is reasonably well nourished.

ECUADOR

Inquiry into the Health of Apparently Healthy Workers.—The Office of Ecuadorean Social Security, which is charged with giving medical care to private employees and workers, is studying the possibility of initiating services of preventive medicine. For this purpose, an inquiry was made in the city of Guayaquil, under the direction of Dr. Alfredo Valenzuela, into the health of 5,000 employees and workers. The results of this inquiry are interesting, since the employees and workers were apparently healthy.

Of the 5,000 persons examined, 3,317, or 66.34%, were men and 33.66%, women. Their ages varied between 20 and 50 years, but 66% were between 21 and 40 years of age. Seventy-six per cent were public and private employees, and 24% were factory workers. Of all these workers, 71% had a monthly income varying between 200 and 800 sucre, that is, $11 to $46, monthly; it should be noted that the cost of living in this country is somewhat less than that in the United States. Nevertheless, it is evident that the average monthly income is insufficient to satisfy even the most elementary needs.

Intestinal parasites were found in 62.84%, distributed as follows: Trichuris trichiura, 41.32%; Endamoeba coli, 21.08%; Ascaris lumbricoides, 11.68%; Trichomonas intestinalis, 7.60%; Ancylostoma duodenale, 6.36%; E. histolytica, 4.62%; Giardia lamblia, 3.74%; Taenia solium, 3.24%; Strongyloides stercoralis, 2.52%; Hymenolepis nana, 0.5%; with smaller percentages of Chilomastix mesnili, Balantidium coli, Oxyuris, Cercomonas, and Anguillula organisms. In 17.4% two or more of these parasites were found in the feces in association.

In regard to venereal diseases and syphilis, all the women in the inquiry denied ever having suffered such infections. Among the men, 19.54% stated that they had had gonorrhea, and 14%, chancre. Serologic tests in all those suspected and all those who

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The items in these letters are contributed by regular correspondents in the various foreign countries.

had had any venereal infection were positive for syphilis in 8.6%, of whom 6.7% were men and 1.9% women.

There were 105 persons, or 2.1%, who proved to be tuberculous; of these, 48 showed slight tuberculosis, 41 moderately advanced tuberculosis, and 16 very advanced tuberculosis. Red blood cell counts between 3,800,000 and 4,200,000 were found in 76%, which indicated moderate anemia. In 11.24% the count was over 4,500,000 and in the remainder less than 3,700,000. Cardiac diseases had a low incidence: 0.88%, consisting, in most cases, of sinus tachycardia (0.36%). Similarly, there was a low incidence of renal diseases, amounting to a total of 0.83%.

Among the other diseases found, the principal ones were as follows: chronic constipation, 36.19% (22.10% in women); acute pharyngitis, 22.79% (women, 13.49%); tonsillitis, 22.21% (women, 15.80%); arterial hypertension, 10.58%; hernias (inguinal, umbilical, and epigastric), 5.49% (women, 2.66%); articular rheumatism, 5.27%; malaria, 3.9%; colitis, 2.76%; bronchial asthma, 2.44%; hepatitis, 2.27%; chronic bronchitis, 1.68%; varices, 1.58% (women, 1.55%); peptic ulcer, 1.28%; hemorrhoids, 0.69%; epilepsy, 0.06%; and cancer of the neck of the uterus, 0.06%. It is interesting to note the present low incidence of malaria, since the campaign carried on systematically throughout the coastal regions, contrasting notably with the figures of 5 or 10 years ago.

Tuberculosis in Azuay.—Under the direction of Dr. Agustín Landivar, the Ecuadorean Antituberculous League has carried out an investigation on the incidence of tuberculous infection and disease in the inhabitants of the central province of Azuay.

The investigation covered 10,734 persons, among whom 7,959 were found positive to the Mantoux test (40.95% with one plus; 37.25% with two; 13.3% with three, and 8.3% with four pluses). Of the total number, 4.66% were found to have active tuberculosis and 0.78%, inactive forms of tuberculosis.

The Rosenthal multipuncture technique was used in vaccinating 2,946 persons; tuberculin allergy and positive reaction to the Mantoux test were found to have been produced in 91.32%.

FINLAND

Bilberry Cure for Oxyuriasis.—The encouraging experiences of a three day bilberry cure for oxyuriasis at the Children's Hospital in Helsingfors are the outcome of observations of an isolated case. It was that of a 10-year-old boy who came to the outpatient department of the hospital suffering from diarrhea. He was told to eat bilberries for a couple of days. On his return to the hospital a fortnight later he had ceased to discharge pinworms (Enterobius vermicularis). This led to an extensive trial of bilberries as a cure for this condition by Dr. Bernhard Landtman of the Children's Hospital. The patients thus treated were 15 boys and 15 girls between the ages of 2 and 15 years. As many as 25 of them had been treated for oxyuriasis by various other means without success. From about half a liter to two liters of bilberries were eaten daily for three consecutive days, either as such or as a purée or thick soup. When fresh bilberries were not in season they were given in dried or preserved form. They were sweetened with sugar, and about a deciliter of milk was given daily with no restriction of the water intake. But no other food was permitted during these three days.

The success of this treatment depended largely on whether the child was treated in the hospital or at home. In the four cases in which the treatment had no effect the children had remained at home, and it is doubtful that the parents followed faithfully the instructions given them. Indeed, one mother admitted that she gave her child ordinary food on the second day of treatment for fear of starvation. In 18 cases the treatment was given in hospital or some other institution. In most of these cases there was an ample discharge of pinworms in the feces as early as the first day of the cure. As many as 20 patients were kept under observation for more than 3 months after the treatment, and the observation periods ranged from 14 days to 5 years. In none of the 26 successfully treated cases could pinworms be found within a few weeks of the completion of the cure, which seemed to be equally effective whether the bilberries were fresh or preserved. The two children who were found to have suffered a relapse nine months after the cure belonged to the same family and probably represented a new infection. No ill effects or complications were observed in connection with this treatment, which, in the opinion of Dr. Landtman, inhibited the development of the eggs of the pinworm as well as expelled the pinworm itself.

Tapeworm Pernicious Anemia.—The prevalence of the tapeworm in Finland has led to much research in this field, and at the Fourth Medical Clinic in Helsingfors, in the charge of Prof. Bertel von Bonsdorff, investigations have recently been carried out concerning the relationship of this parasite to anemia. Von Bonsdorff has shown that the tapeworm, when lodged in the upper sections of the small intestine, is able to prevent the interaction of the extrinsic and intrinsic factors involved in the development of tapeworm pernicious anemia. In cooperation with Dr. R. Gordin he has lately shown that the tapeworm itself absorbs these particular factors. Another investigation has been carried out at the University Hospital in Helsingfors by Dr. Gösta Björkenheim with special reference to the neurological sequels of the pernicious anemia caused by the tapeworm. During the period 1948-1950 he has carried out neurological examinations at various Finnish hospitals of 95 patients between the ages of 11 and 75 who were suffering from this particular form of pernicious anemia. In as many as 75 of these cases there was evidence of some degenerative process in the nervous system.

A close study of these cases showed that in 34 of them the involvement of the nervous system was severe enough to impair capacity for work, the chief cause of this incapacity being ataxia. A reexamination of 80 of these patients from 1 month to 24 months after vermifuge treatment showed that the blood picture had usually become satisfactory. The condition of the nervous system had become worse only in six patients, while the remainder showed improvement or complete recovery. Indeed, the improvement had been so marked in more than half these cases that a neurological examination revealed nothing amiss. In the opinion of Dr. Björkenheim, this satisfactory response to vermifuge treatment depends on the restored ability of the human organism to make proper use of the essential factors in question. The observation that some lesions of the central nervous system are reversible in cases of tapeworm pernicious anemia must, however, be qualified by the admission that certain of these lesions are permanent. Dr. Björkenheim has found that liver preparations and vitamin B_{12} exert a beneficial influence on these neurological manifestations but not more so than vermifuge treatment alone.

Study of Congenital Syphilis.—At the Haga Welander Home for children suffering from congenital syphilis, Dr. Ole Enkvist of Helsingfors has investigated the problem of whether specific treatment of a syphilitic mother during pregnancy affects the prognosis of her offspring. It has been argued that so-called malignant cases of congenital syphilis, refractory to specific treatment, occur as frequently among the children born to mothers given specific treatment during their pregnancy as among the children for whom no specific prenatal treatment has been given. Dr. Enkvist's material concerns 555 children who suffered from congenital syphilis, who were less than 3 years old, and who could be classified in two groups according to whether their mothers had received specific treatment with bismuth and arsphenamine during their pregnancies. In the first group there were 102 children, and in the second 453.

A comparison of 61 children in the first group with 131 in the second with reference to the date at which the first signs of congenital syphilis appeared showed that there was no appreciable difference in this respect between the two groups. But when they were compared with regard to weights at birth, it was found that the group with untreated mothers was considerably lighter in weight. The mortality among the 102 children in the first group was 26%, whereas among the 453 children in the second group it was as high as 36%. Yet another comparison showed that the number of uncomplicated and relatively mild lesions predominated in the first group, whereas visceral syphilis with a definitely and persistently palpable spleen was commoner in the second group. Syphilis of the bony system was also more frequent in the second group. On the other hand, neurosyphilis was more frequent in the first group, and this observation is in conformity with the well-established finding that the neurosyphilis of adults occurs more frequently among patients given inadequate treatment than among others given no treatment at all.

Dr. Enkvist concludes that, though specific treatment of the mother during pregnancy may better the prognosis for her syphilitic offspring, the benefits obtained are not as great as expected. The number of the patients in his first group is rather small for a statistical analysis such as this, and it is further impaired, as he points out, by the lack of sufficiently exact data with regard to the treatment each of the expectant mothers received.

ITALY

Medicosurgical Society.—The medicosurgical society of the Romagna region held a meeting on Feb. 10, under the chairmanship of Professor Sighinolfi. Professor Musconi, from Imola, reported two cases of echinococcus cysts of the liver in which clinical, radiological, and laboratory findings led to a wrong diagnosis that could not be rectified during the surgical intervention because the complications that occurred within the cysts (hemorrhagic overflowing in one case and biliary infarction in the other) had greatly altered their anatomic and pathological characteristics. In the first patient the surgical intervention was performed for gastric ulcer and a blood cyst of the liver was found. One year later during a second intervention in the same patient multiple echinococcus cysts were found, one of which was located in the lumen of the common bile duct. In the second patient a large mass beneath the liver was diagnosed clinically as dropsy of the gallbladder, since all symptoms of echinococcus infection were absent and cysts of the gallbladder were not revealed by the roentgenograms. During the operation the cyst was not found; histological examination justified diagnosis of echinococcus cyst. Professor Musconi concluded that diagnosis of echinococcus cyst is very difficult and sometimes impossible in cases of dead cysts in which all clinical symptoms are absent.

Professor Santini, from Ravenna, discussed radiological diagnosis of gastric ulcer. If the anatomic and pathological statistics are compared with the clinical and radiological, it is seen that diagnosis of gastric ulcer is not thought of often considering the frequency of this lesion. Of particular interest in the statistical study is the ratio between the frequencies of gastric ulcer and duodenal ulcer: in this field the disagreement between clinical pathologists and anatomic pathologists is pronounced in that the former support a more frequent occurrence of duodenal ulcers, the latter of gastric ulcers. Discussing the diagnosis of gastric ulcer, Professor Santini pointed out that in about half of the cases classic symptoms permitting a diagnosis based only on anamnestic study are not present. Radiological examination is superior to clinical examination, but many times a gastric ulcer also escapes the radiologist.

The only radiological pathognomonic sign of the gastric lesion is the niche, but indirect signs, such as antral gastritis, incisura of the great curve caused by spasm of the gastric muscles, retraction of the small curve, eccentricity of the pylorus compared with the base of the duodenal bulb, the deviation toward left of the duodenal bulb, and segmentary rigidity, are also valuable. Several technical devices may be used to put in evidence signs of ulcer; in order for the niche to become visible it is necessary that the bright bundle of rays be perpendicular to the diametrical plane of the gastric cylinder passing through the niche. The study of the patient in standing, inclined, and right triclinic position may be advantageous in each particular case. Therefore, Professor Santini concluded that it is impossible to recommend a standard position, but each position should be used according to the zone that is being studied and to the constitutional configuration of the stomach. Trendelenburg's position should not be excluded since it is very useful in those cases in which the cephalic pole of the stomach is being studied. This zone is often overlooked.

Comparing the advantages that may be obtained by means of an examination with small doses and large doses of opaque medium, he observed that the former is useful for revealing modifications of the folds of the mucous membranes but is almost always insufficient to reveal an ulcerous niche. Large doses of opaque medium can penetrate in the ulcerous excavation provided that the level of the meal reaches the ulcer. When it is believed that the ulcer is located in a high position, a second meal of barium may be given, or else the patient may be forced to a voluntary abdominal retraction while the radiographs are taken. By these means ulcers of the superior tract of the small curve are often detected that otherwise would not have been seen.

This technique in examining the digestive tract has been followed for many years at the radiological department of the Ravenna Hospital where about 5,000 patients have been examined from January, 1948, to July, 1950. The results reveal that the number of gastric ulcers is greater than that reported by clinical physicians and also by some radiologists and that, therefore, the ratio between the occurrence of duodenal ulcer and gastric ulcer is smaller than that usually thought of.

Drs. Suzzi Valli and Dominici reported on the first case of autochthonous visceral leishmaniasis that occurred in the Romagna region in 1951 in an adult living in a locality of the Forlì province. The Romagna region is the source of endemic centers of cutaneous leishmaniasis from which the disease is progressively spreading, but up to date no case of the visceral form had been reported.

SWEDEN

New Light on Rickets.—The tendency of modern textbooks to oversimplify the etiology of rickets by tracing this disease solely to the lack of sunlight and vitamin D is challenged by Dr. Gert V. Sydow, who presents an analysis of 64 cases of active rickets observed during a four year period at a hospital in Sundsvall since its children's department was opened in September, 1947. In addition to the 64 active cases there were 14 in which the disease had been arrested. This analysis, published in *Svenska Läkartidningen* for April 10, 1952, shows that there were 21 girls and 43 boys suffering from active rickets. The average age of the girls was much higher than that of the boys when rickets was first diagnosed; before the age of 10 months there were only 3 girls as compared with 31 boys. On the other hand, among the 13 cases of rickets diagnosed after the age of 12 months, there were as many as 8 girls. There was also a sex difference with regard to the incidence of tetany, which affected 21 infants, 17 of whom were boys. Dr. V. Sydow suggests that these sex peculiarities, if confirmed by a larger series and statistical analysis, indicate a genetically determined element in the etiology of rickets. His material also includes cases indicating a hereditary and familial element in the etiology of the disease. Another observation is that the persons with rickets are undersized, a condition again suggesting a congenital factor of a genotypic nature. Dr. V. Sydow is unable to confirm the theory that rickets is a function of growth and that the risk of developing rickets increases with the rate of growth of the child. It should be noted that his diagnosis of rickets depended on radiological examinations of the wrists and on serum analyses.

Alcohol and Venereal Disease.—The committee appointed under government auspices to investigate problems concerned with the control of alcohol in Sweden has made a special study of the connection between alcohol and venereal disease. In cooperation with the public health service, it issued a detailed questionnaire to patients receiving treatment for venereal disease in venereal disease centers and from medical officers of health and private practitioners. The recipients of this questionnaire were asked to fill in data concerning their sex, age, occupation, the date when infection occurred, and the place in which infection occurred, i. e., in a hotel, a cellar, a passage, or out of doors. Further, they were to tell whether there was a permanent or a casual relationship with the partner, and, if the latter, whether it was for payment. Many other points were raised, such as the character of the places of entertainment in which the partners had met, whether appointments had been made by telephone, and whether the partner was a fellow citizen or a foreigner. They were asked whether one or both partners were under the influence of alcohol at the time and in what form it had been consumed. The veracity with which the questionnaire was answered may have varied somewhat with the sex and occupation of the recipient. For example, the women were apt to be inaccurate over the character of their partnerships, not liking to be classed as prostitutes. As many as 1,413 questionnaires were answered well enough to be suitable for statistical study. Infections received on Saturdays and Sundays were together more numerous than the infections received during all the rest of the week. More than 60% of the men had been infected by casual or occasional partners. This was so for barely 15% of the women; most of the others were infected by more or less permanent partners. Not one woman considered herself a prostitute. Nearly half of the seamen were infected by prostitutes. About four-fifths of the men infected through prostitution were under the influence of alcohol at the time. During casual relationships, more than half of the men were under the influence of alcohol, and 42% of the women with casual partners consumed alcohol at the time. Patients infected abroad more often were under the influence of alcohol at the time than patients infected in Sweden. It was remarkable that the infection rate was so high in places of entertainment in which no alcohol was served. The 15 pages devoted to this discussion contain many tables and diagrams from which the reader can glean information without drawing any cut and dried conclusions as to the exact share taken by alcohol in promoting venereal disease.

Refusal of Physician to Induce Legal Abortions.—Trouble has lately risen over the refusal of a professor in the gynecologic department of the Sabbatsberg Hospital to induce abortions authorized as legally permissible by the authorities. The matter has been given so much prominence in the lay press that the Swedish Medical Association has felt obliged to publish a memorandum on it. It pointed out that it is the duty of every physician to give the advice and treatment that his patient's condition requires and that is in conformity with scientific knowledge and well-tried experience. He may be held responsible when he ignores these considerations, but no Swedish law obliges a physician to carry out any operation on human beings. Legislation provides for the induction of abortion but does not oblige a physician to induce it when he finds abortion unjustifiable. To threaten such a physician with penalties and dismissal from his post for this reason would be to abandon the principles governing the practice of medicine in the Western world. "It is in dictator states that doctors are obliged in the exercise of their profession to act contrary to their scientific and ethical convictions." It is pointed out in this memorandum that the Swedish Medical Association is concerned only with the principles involved and not with any individual case still before the court.

Prophylaxis of Acoustic Trauma.—In the ear department of the Falun Hospital, Dr. Rolf A. Hillesund has compared the effects of various devices for the prevention of acoustic trauma. With the help of audiograms he noted the protective effects of vulcanite earplugs, ordinary cotton wool, and glass fiber wool on 10 persons with normal tympanums. Vulcanite earplugs are not, in his opinion, bearable as a rule for more than a couple of hours in the day, and they seldom fit well. On the other hand, glass fiber wool can be made to fit well in every case, can be used all day with greater comfort than the vulcanite plug, and is quite cheap. With glass fiber wool in both ears, persons with normal hearing can usually catch a speaking voice several meters away. Dr. Hillesund believes that there is little chance of glass fiber wool provoking eczema in the auditory passages. Referring to the problem of acoustic trauma in general, he pointed out that at the present time the best prophylactic measure seems to be the protection of the individual user rather than the provision of machinery for silencing sounds.

The Influence of Climate on Rickets.—At a meeting of the Swedish Medical Society, several speakers dealt with the diagnosis of rickets by radiological examinations. As these are not always available, clinical criteria become more important than ever, and one speaker referred to craniotabes as the most objective physical test or sign of rickets. The same speaker, Dr. Sixten Haraldson, showed what a profound influence climatic conditions in the north of Sweden can have on rickets. The grotesque skeletal deformities portrayed in the textbooks may be rare, but he has seen a score of cases of craniotabes every year at Kiruna, in the far north of Sweden. After the sunless summer of 1949 there were some three score such cases seen. In the far north much of the penetrating effect of the solar rays is lost, and this loss is not sufficiently compensated for by reflection from the snow or by freedom from industrial pollution of the air. The comparatively sunny months of March and April usually have such low temperatures that little of a child's skin can be safely exposed to sunlight out of doors. The midnight sun in the summer is of course not available for the child who is dutifully sent to bed at night. The sunlight of July and August is often wasted because swarms of mosquitoes often render life out of doors quite unbearable. Dr. Haraldson declared rickets to be one of the causes of the defective teeth and delayed dentition often observed in Kiruna. He was in favor of fish-liver oil as a prophylactic, to be administered with special reference to the climatic conditions in the north of Sweden.

SWITZERLAND

Inquiry on Prefrontal Leukotomy.—The periodical *Médecine et Hygiène*, a Swiss medical journal published fortnightly in Geneva, sent a questionnaire to specialists in psychosurgery from America, France, and Switzerland asking them to express their opinions concerning the indications and contraindications of this therapy, the damage it caused to personality, and the medicosurgical problems it entailed. Answers favorable to prefrontal leukotomy included those from Dr. Freeman, professor at the Psychiatric Institute of Washington, who compared psychosurgery to any other surgical operation devoted to alleviating suffering; from Dr. Wertheimer, professor of neurosurgical clinics at the Faculty of Medicine of Lyons (France), who estimated that leukotomy is an efficient therapeutic act in many cases; and from Professor Lhermitte (Paris), Dr. Bersot (Switzerland), secretary of the European Federation for Mental Health, Dr. Cossa (Nice), and Dr. Feuillet (Nancy) who think that frontal leukotomy is the only medical act capable of opening a way of hope to patients who had before an intolerable life.

Professor Morel, head of the university psychiatric clinic of Geneva, and Dr. Baruk, chief of the clinic of Charenton (Paris), were opposed to leukotomy. The former, founding his opinion on histological facts, estimated that the enormous quantity of fibers cut by the surgical section must provoke the secondary degeneration of the cortical neurons afferent to the sectioned fibers; this opinion is not accepted by Wertheimer, who thinks that there is no degeneration, because of the short fibers of association. Baruk considers that leukotomy results in serious damage to the moral personality of schizophrenic patients and would like to see this operation abolished. Other persons, like Dr. Porot (Alger), admit leukotomy but make reservations for its indications.

Opinion was unanimous that leukotomy should be done only after all other treatment has failed. Leukotomy is indicated only in cases having no tendency to spontaneous healing and showing a behavior that is not compatible with any social life or that is dangerous for the patient (suicide).

Most of the authors, even those who are opposed to leukotomy, do not place much emphasis on the postoperative problems of behavior; if the characteristics change, they change in a favorable rather than an unfavorable way.

Concerning the medicolegal aspect, all believed that the decision must be made by the psychiatrist, but with the agreement, if not of the patient, at least of his family or guardian. The legal responsibility of the patient who has undergone leukotomy varies according to his state of mind, established by a survey, as in any other litigious case.

On the whole, in viewing the gravity of the cases undergoing leukotomy, favorable results, even of modest percentage, though they sometimes reach 20 to 30% of schizophrenias (Wertheimer), must be taken in consideration. In the absence of more effective therapy it would not be wise to recommend abolition of prefrontal leukotomy, even if it is yet only a palliative process.

CORRESPONDENCE

POTENTIAL DANGER OF ISONIAZID RESISTANCE THROUGH FAILURE TO USE MULTIPLE CHEMOTHERAPY IN TREATMENT OF TUBERCULOSIS

To the Editor:—Not since the advent of streptomycin has the medical profession been given an antituberculosis drug with the tremendous therapeutic potentialities of isoniazid. Are these potentialities to be fulfilled, or is isoniazid to become rapidly ineffective owing to the development of bacterial resistance? The answer to this urgent question depends on the attitude of those responsible for its clinical use. Most physicians are already aware of the dangers inherent in the development of isoniazid resistance, but, if a minority is to take the position that bacterial resistance is a laboratory curiosity of no clinical significance, the value of this drug may be greatly lessened. It is a matter of historical record that the wide use of sulfadiazine in naval training centers during World War II eventually resulted in the establishment of streptococcic types resistant to sulfonamides (Epidemiology Unit no. 22: *J. A. M. A.* **129**:921-927 [Dec. 1] 1945). This unfortunate experience in the administration of drugs to relatively segregated groups appears to have been largely forgotten.

While it is true that bacterial resistance to penicillin and sulfonamides in our civilian population has not yet presented serious difficulties, two facts about isoniazid in the treatment of tuberculosis set it apart as a special problem. First, it is significant that patients in tuberculosis hospitals represent segregated groups; furthermore, they have a more or less active chronic disease. Infection with isoniazid resistant strains of extraneous origin may be expected in some hospitals and, later, in the general population. The euphoria and subjective improvement of patients treated with isoniazid will make their control all the more difficult, and the narrow antibacterial specificity of isoniazid will leave secondary infection relatively unchanged.

The second important fact is that isoniazid is the only commercially available chemotherapeutic agent known, other than streptomycin, against which bacteria can develop practically complete resistance in a single step (Szybalski, W., and Bryson, V.: *Am. Rev. Tuberc.* **65**:768-770, 1952). Although this evidence is based on a study of nonpathogenic tubercle bacilli, it has been indicated that human pathogens behave similarly in the development of isoniazid resistance (Middlebrook, G.: *Am. Rev. Tuberc.* **65**:765-767, 1952; Hobby, G., and Lenert, T. F.: *ibid,* **65**:771-774, 1952). Unlike levels of bacterial resistance, the pattern depends on the drug rather than on the species of bacteria (Demerec, M.: *J. Bact.* **56**:63-74, 1948; Bryson, V., and Demerec, M.: *Ann. New York Acad. Sc.* **53**:283-289, 1950). Also, resistance to isoniazid orginates about 1,000 times as rapidly as streptomycin resistance. In terms of mutation rate, an isoniazid resistant bacterial cell will arise on the average in bacterial populations 1,000 times smaller than are required to produce streptomycin resistance. For those who still believe the origin of resistance by mutation rather than by drug contact is an academic hypothesis, the simple isolation of fully isoniazid resistant strains in the complete absence of the drug by the replicate plate method of Lederberg should remove the last vestige of doubt (Szybalski, W., and Bryson, V.: unpublished data). Although reversion to sensitivity through more rapid growth of sensitive mutants may occur in the laboratory following removal of isoniazid from the media, many bacterial generations are required, and there is no assurance that similar reversion will occur in vivo.

In the recent meeting of the National Tuberculosis Association at Boston (May 26-29, 1952) it was reported by attending physicians that extensive tests of isoniazid are in progress or are soon to be made. A few of these tests will contrast the effects of isoniazid used alone against other forms of chemotherapy. This we regard as nothing short of catastrophic. Isoniazid resistant strains of human tubercle bacilli following relatively brief therapy have already been reported by Steenken, Meade, Wolinsky, and Coates (*J. A. M. A.* **149**:187 [May 10] 1952).

When first available, streptomycin was also used alone. The necessity of administering *p*-aminosalicylic acid with streptomycin to retard the origin of resistant strains was learned as a result of much time and effort. When bacterial resistance is a problem, the advantages of using multiple chemotherapy have often been stressed (Carpenter, Bahn, Ackerman, and Stokinger: *Proc. Soc. Exper. Biol. & Med.* **60**:168-171, 1945; Grace, E. J., and Bryson, V.: *Arch. Surg.* **50**:219-222 [April] 1945; Bryson, V., and Grace, E. J.: *New England J. Med.* **237**:683-692, 1947; Klein, M., and Kimmelman, L. J.: *J. Bact.* **54**:363-370, 1947). Mutation to resistance against two drugs used together is theoretically the product of individual mutation rates. In the case of isoniazid and streptomycin combined, a volume of bacteria almost as great as the entire human body would be required for the finding of a cell simultaneously resistant to both agents. On the other hand, the use of drugs in sequence, as resistance develops to each in turn, will not prevent the eventual occurrence of multiple bacterial resistance. If the organisms of human tuberculosis develop very high isoniazid resistance by a single step mutation, no clinically feasible dose of the drug used alone will prevent the selection of resistant strains, providing the bacterial population is moderately large or actively multiplying.

There is no doubt that large-scale tests of isoniazid by hospitals and physicians engaged in private practice will soon begin. In spite of some antagonism of isoniazid with subinhibitory concentrations of streptomycin (Szybalski, W., and Bryson, V.: unpublished data), we suggest most emphatically that isoniazid be employed only in multiple combinations with streptomycin and *p*-aminosalicylic acid or other chemotherapeutic agents active against mycobacteria and not related by cross resistance. For example, streptomycin-*p*-aminosalicylic acid therapy should be compared with and without isoniazid, rather than as an alternative to isoniazid. Otherwise, we predict the early and widespread appearance of isoniazid resistant strains of tubercle bacilli. Such an event will largely neutralize one of the most hopeful developments in the recent history of tuberculosis therapy.

In releasing isoniazid to the medical profession the Food [illegible] Drug Administration has approved package labels stating [illegible] use in the treatment of streptomycin resistant tuberculosis [illegible] close supervision of a physician." While the treated [illegible] temporarily improve with isoniazid, the eventual [illegible] others with organisms resistant to both streptomycin [illegible] azid could make any prolonged medical program [illegible] F. D. A. recommendations a potential threat [illegible] value of the drug. We would prefer to see [illegible] use only in combination with another tu[illegible] tive against tubercle bacilli of the patient [illegible] be between prolonging the lives of a few [illegible] today as opposed to controlling tuber[illegible] within one generation.

POLLEN PREVALENCE AND POLLEN-FREE AREAS

To the Editor:—In the correspondence section of THE JOURNAL May 3, 1952, page 82, Dr. Ralph Bookman of Los Angeles voices his concern about the effect on the average physician of my article "Pollen Prevalence and Pollen-Free Areas," which appeared in THE JOURNAL March 1, 1952, page 716. He is also disturbed by the economic and social dislocation which he predicts for many of those pollen sensitive persons "who heed such advice." If this were the first appearance in medical and lay publications of the facts about quantitative pollen distribution, one might be persuaded to share the doctor's pessimism, since no one denies that facts, unless interpreted with common sense, are often dangerous. But the article in question has only augmented a body of similar observations published in numerous medical journals and books over a period of 25 years by many allergists as well as by their collaborators.

It seems pointless to deny that a pollen sufferer may ever escape pollen allergy by avoidance of the pollens to which he is sensitive. This is the only conclusion to be drawn from Dr. Bookman's effort to brand as fallacies first "that it is possible to escape a pollen allergy, and, second, that there are certain areas, suggested by the accompanying maps, to which a pollen sensitive person may flee." In this he does not agree with the teaching of his fellow allergists or take into account the satisfactory experience of many ragweed sensitive patients. Dr. Morrill Wyman, as early as 1876, recorded cases of ragweed allergy in which the sufferer obtained perfect relief for a whole season by going to Europe or to the wooded areas of Canada during August and September. Tuft, in "Clinical Allergy" (Philadelphia, Lea & Febiger, 1949) says, "Complete elimination of the offending allergen from the diet or environment of the patient is the ideal therapeutic method, since it obviates the necessity of other treatment." Feinberg, in "Allergy in Practice" (Chicago, Year Book Publishers, 1946) says, "The ragweed-sensitive patient may be relieved by living in European countries, Asia, parts of South America, Africa, and Australia—no matter what the climate may be. The ragweed-sensitive patient may find increasing relief the farther north he goes into Canada or by going west of the Rockies or to the southernmost portion of Florida. Persons sensitive to certain other pollens may come to the Midwest with consequent relief." Alexander, in "Synopsis of Allergy" (St. Louis, C. V. Mosby Co., 1947) says, "The ideal treatment of hay fever, like other forms of allergy, is to avoid contact with the offending agent. . . . The availability of pollen counts over several years in essentially all sections of the country offers an excellent guide as to where one may go to escape hay fever."

As for Dr. Bookman's statement that "A person sensitized to eastern pollen who moves to this area will probably become sensitized to our local flora within a period of from 6-18 months," the possibility that ragweed sufferers from the middle and eastern states may in time become sensitized to indigenous California pollens has already been discussed in considerable detail by southern California allergists, as well as by those having clinical experience both in the ragweed belt and in southern California (*J. A. M. A.* **139**:554 [Feb. 19] 1949; ibid. **141**:99 [Sept. 3] 1949). Due attention has also been given to the matter in my chapter in Vaughan's "Practice of Allergy," second edition (St. Louis, C. V. Mosby Co., 1948, p. 470). Suffice it to say that there are no published data available on the frequency with which such new sensitization may occur, so that it is not possible to evaluate Dr. Bookman's use of the term "probably" in this connection. It is, however, pertinent to inquire: How many of those who do change residence without any subsequent difficulty take the trouble to report such a happy outcome to any allergist in the new locality?

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ULTRAFILTRABLE ACTH ACTIVITY OF HUMAN PLASMA

To the Editor:—We have previously published in THE JOURNAL (**147**:525 [Sept. 29] 1951) a paper concerning the adrenocorticotropic activity of human blood, directly testing the plasma with the "ascorbic acid depletion method," according to our own technique. In our paper we called attention to the fact that in comparative experiments losses of adrenocorticotropic activity might occur when the determination is made with fractionated plasma (Bornstein and Trewhella, *Lancet* **2**:678, 1950) since the blood fraction with adrenocorticotropin activity is not totally precipitated from +94% acetone.

Our observations and some reports of other authors that the biologic activity of corticotropin (molecular weight 20,000) is not connected with the intact protein molecule but with one of its fractions (Li, C. H.: *Nederl. tijdschr. geneesk.* **94**:1093, 1950) have induced us to investigate whether the corticotropic hormone is, also in the blood, in a form simple enough to be ultrafiltered.

Until now, studies have been performed on blood of five normal adults. The plasma of every person was tested comparatively, before and after ultrafiltration, with the ascorbic acid depletion method. The volume injected in each animal subjected to hypophysectomy was in every case 0.5 cc. per 100 gm. of body weight. Three animals were used for each experiment.

The plasma ultrafiltration was performed immediately, with a cellophane membrane in an adequate ultrafilter, at a pressure of 15 atmospheres, in inert gas (nitrogen) and at a temperature of +2 C. In 90 minutes it is possible to obtain a volume of ultrafiltrate sufficient to make injections in three animals.

Mean falls in ascorbic acid concentration of adrenals expressed as milligrams per 100 gm. of fresh tissue, according to our experimental conditions, were 35.6±3.6 mg. for the plasma before and 46.6±4.25 after ultrafiltration.

From these results, we conclude that the adrenocorticotropic activity of the blood of normal adults is completely ultrafiltrable. Further results on this subject will be published more extensively in another paper.

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CHOLINERGIC DRUGS IN PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

To the Editor:—An article entitled "Use of Cholinergic Drugs in Paroxysmal Supraventricular Tachycardia," by Furman and Geiger (*J. A. M. A.* **149**:269 [May 17] 1952) has come to our attention. In the "comment" section of the article by Drs. Furman and Geiger, we note the following statement: "In our opinion, neither agent should be used in the average case except after less dangerous methods have been properly employed without success." Using the word "neither" implies condemnation of both, either individually or in combination. From the studies reported in this paper it is obvious that such a statement indicting either drug independently is not warranted, especially since both had been used in each instance. In the last sentence of the same paragraph we find the statement: "Only in such situations in which the patient's condition is so grave . . . do we feel that the use of methacholine is justified." With the primary attention focused repeatedly on methacholine, neostigmine is still mentioned as an accessory drug of hazardous action.

The undersigned independently introduced the use of neostigmine for paroxysmal tachycardia in clinical medicine in 1941 (*M. Rec.* **153**:134 [Feb. 19] 1941; ibid. **153**:209 [March 19] 1941). In our experience of over 1,000 injections of neostigmine, many of these for supraventricular tachycardia of sinus, auricular, or nodal origin, we have yet to witness a severe reaction due to its use. We still recommend the use of neostigmine methylsulfate, 1 mg. subcutaneously, in the management of paroxysmal supraventricular tachycardia.

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COUNCIL ON MEDICAL EDUCATION AND HOSPITALS

The Council on Medical Education and Hospitals has adopted the following statement and voted that it be published.

DONALD G. ANDERSON, M.D., *Secretary.*

FULL COST TUITION

The problem of financing the medical schools, always a difficult one, has been intensified since the end of World War II. To meet this problem, a number of schools have recently considered raising tuition charges to meet or nearly meet the full operating costs involved in the instruction of undergraduate medical students. In support of such action, it is argued that the average medical student will some day receive a comfortable income and that it is reasonable to ask him to pay the entire cost of his education. Several varieties of payment plans with varying exemptions have been suggested, but all are based on essentially the same principle, which is that the medical student, on entering medical school, signs an unsecured note for the difference between the usual tuition fee and the "full cost" to the school of his education, the amount to be repaid over a long period following his entrance into active, private practice. These plans are offered as new and perhaps "revolutionary" innovations in medical school financing. Actually, there is nothing new or unique about them except the techniques of collection.

The principle of full cost tuition is as old as American medical education and was discredited more than 40 years ago. Most of the arguments that may be advanced to support it today were given expression by the proprietary medical schools when an earlier generation of American medical educators stated their conviction that a stable high grade program of medical education could not be financed primarily by student fees. In 1910 Abraham Flexner wrote: "Our conclusion is that established schools . . . may count on fees to the extent of one-third to one-half of the expenditure required to conduct a good school of modern medicine; and that as the department becomes more homogeneously developed, the fees will tend to do even less."

Although the plans referred to assume that a medical school can compute accurately the full cost of educating a medical student, several recent studies have indicated that it is extremely difficult if not impossible for most medical schools to segregate the basic cost of medical instruction from costs incurred in the instruction of other students, in research, and in medical care. Until such figures can be derived, the fairness of any charge that purports to represent the full cost of medical instruction would be open to serious question.

It must be clear that all these plans would be subject to the same dangers and abuses that were inherent in the proprietary schools. It is not unforeseeable that a medical school operating under such a program would be strongly tempted to solve certain of its faculty problems by the simple expedient of raising salaries at student expense. Furthermore, it is but a short step from use of student fees to maintain essential teaching laboratories to the exploitation of such fees for the acquisition of special equipment, for maintaining research, and for the remodeling or even the construction of new facilities. If some of these fears seem farfetched it need only be recalled that they are the very abuses that the leaders of American medicine were struggling but a few years ago to eliminate.

It may be granted that full cost charges would temporarily ease the financial burden of the schools. It is probable, however, that such charges would in time actually make the situation worse. It would mean the abandonment of the established principle that medical education is a social function and is, therefore, rightfully a public trust. Years of effort have been expended in enlisting the interest, sympathy, and support of the general public for medical education, and the splendid development already achieved by the nation's medical schools is a measure of the public's willingness to bear this responsibility. It is entirely possible that placing the whole burden of operating costs on the student might jeopardize or even eliminate other important sources of support by appearing to reduce or to remove the need for support from such sources. Irrespective of the reasons offered in support of such a plan, many will regard the institution of full cost tuition as an abdication of social responsibility by the governing boards of universities.

One of the most serious objections to full cost charges concerns their possible effects on the schools adopting such policies and on the student himself. It is unlikely that a student will either pay these large fees or obligate himself to pay them on long-term contract if he can gain admission to an equally good school at less cost. The full cost school would then be placed at a serious disadvantage in the competition for able students and might easily find itself largely a school of last choice. It is equally possible that many students would choose other careers rather than assume such a long-term financial obligation. The study of medicine would then be left largely to the well-to-do, who could afford outright payment, and medicine would lose the services of many able and deserving persons.

The consequences to the ideals and ethics of medical practice that might follow the practice of putting such a high price on medical education would be most serious. At present, although students of medicine are aware of the financial sacrifice that they make through spending long years in training while their contemporaries are already gainfully employed, they are also aware that others are contributing significantly toward the cost of their education. It is impressed on a student that, while the physician is ordinarily entitled to payment for his services, remuneration must not be the primary consideration on which rendition of service is based. Altruism and compassion are paramount elements and are traditionally considered to be the vital, motivating forces in medical practice. As soon as medical education is placed on a dollar for dollar basis, with a large price attached, those ideals are placed in jeopardy. The graduate, laboring under a large debt, would be sorely tempted to regard his practice as a business enterprise, purchased in the market place, to be exploited and commercialized accordingly.

In view of all these inherent and potential dangers, the Council on Medical Education and Hospitals believes that plans for charging full cost tuition should be condemned as being inimical to the best interests of medical education and the public.[1] It would appear that the future quality of medical schools and of the practice of medicine will be better safeguarded if the medical schools will continue to cultivate and expand accepted sources of support. Recent developments would seem to indicate that there is no need for despair and that, as there is developed an increasing awareness of the social functions and benefits provided by their medical schools, increased support from the general public may be anticipated.

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[1] The Council distinguishes between plans charging the individual medical student the full cost of his education and plans such as those developed by regional educational programs by which a medical school in one state receives from a governmental agency of another state a subsidy, which may be in the form of a full cost tuition charge, for each student it admits from the latter state. Such a program represents essentially the purchase of services by one state from another state and is not subject to the abuses or undesirable effects that would result from charging the individual student for the full cost of his education.

BUREAU OF MEDICAL ECONOMIC RESEARCH

THE REVISED KEOGH-REED BILLS

(H. R. 8390 and H. R. 8391)

Frank G. Dickinson, Ph.D.

The original bipartisan Keogh-Reed bills (H. R. 4371 and H. R. 4373) were introduced into the House of Representatives on June 7, 1951, at the request of the Special Committee on Retirement Benefits for Lawyers of the American Bar Association. The chairman of the committee is Mr. George Roberts, 40 Wall Street, New York City, and the draftsman of the identical bills was Mr. Leslie R. Rapp, a lawyer of New York City. Late in 1951 Mr. Roberts organized a Co-ordinating Committee on Retirement Benefits for the Professions and the Self-employed and has served as its chairman. This committee is composed of representatives of more than a score of organizations—mostly professional—and of the American Farm Bureau Federation. The representative of the American Medical Association is the Director of the Bureau of Medical Economic Research.

The original Keogh-Reed bills have been studied in great detail by the representatives of many national organizations. Several conferences were held with the technical staff of the Joint Committee on Internal Revenue Taxation of Congress. The revised drafts of these identical bills were introduced on June 27, 1952, by Representatives Eugene Keogh (D, N. Y.) and Daniel Reed (R, N. Y.). (In order for a bill in the House of Representatives to be truly bipartisan, two identical bills must be introduced, one by a Democratic Congressman and the other by a Republican Congressman.) They were referred to the Committee on Ways and Means on June 27, but the committee took no action on them before Congress adjourned on July 7. The bills will be introduced again next January. The revised bills (H. R. 8390 by Mr. Keogh and H. R. 8391 by Mr. Reed) resulted directly from the hearing of May 13 by the Committee on Ways and Means of the House of Representatives.[1] At that hearing, a number of amendments and changes were suggested by some of the 19 persons who testified. In the revised draft an attempt was made to follow as many of the suggestions made during the hearing as seemed to be in the public interest.

The general purpose of this bill is to provide a voluntary pension system for self-employed taxpayers who cannot be covered under the terms of section 165 (a) of the Federal Internal Revenue Code and to cover employed persons who for one reason or another are not covered and cannot expect to receive a pension from their employer. In other words, the purpose of this revised measure is to bring about a degree of equity for the taxpayers who are not now covered by pension plans with those who are now covered by pension plans approved under section 165 (a), by pension plans of local, state, and federal units of government, or by plans of nonprofit organizations. It is imperative that the federal internal revenue law cease to discriminate against certain taxpayers in the matter of pensions.

This discussion is not concerned with the Social Security Act, which for the present purposes may be considered the first layer of pensions; rather it is concerned with the second layer of pensions, in which the employer and employee, by a wide variety of plans, seek to provide pensions for the employees to be financed by premiums paid to insurance companies or by amounts set aside in pension and profit sharing trusts. More than 16,000 pension plans have been approved under section 165 (a) since it was enacted in 1942. Ten million of the 60 million gainfully employed are currently outside the scope of this section because they are self-employed. But the self-employed, as they grow older, are just as much subject to the feebleness and vicissitudes of old age as those who during the working years were employed persons covered by employee-employer pensions.

Before the differences between the old and the new versions of this measure are explained, certain basic facts should be set forth. Although it has not been possible to obtain any data from the pension trust section of the Bureau of Internal Revenue, available estimates indicate that several billion dollars paid by employers into a variety of pension plan funds are not currently being taxed to either the employer or the employee, although the employee when he retires will be required to declare as taxable income that portion of his pension that was financed by his employer. It should be clear, therefore, that this measure does not introduce a new factor into our revenue law but rather permits self-employed taxpayers and employed taxpayers not now covered by pension plans to defer taxes on certain portions of their current income. The setting aside of funds by the self-employed alone under this revised measure might possibly exclude a half-billion dollars annually from current taxation; on this sum, of course, taxes would eventually be payable.[2] The wide support given to the pension plans authorized under section 165 (a) by Congress since 1942 clearly establishes a favorable attitude toward the extension of similar privileges to those taxpayers not now covered. There would be no tax avoidance—only tax deferment. For this reason it would be better to extend our revenue legislation to cover them than to repeal section 165 (a), under which 16,000 pension plans have been approved.

The revisions and changes in this measure have occupied the attention of many persons. The special committee of the American Bar Association on Retirement Benefits for Lawyers has supervised the study and consideration during the past year that culminated in the hearing on May 13. The Bar Association committee was ably assisted by representatives of the American Medical Association, the American Dental Association, the American Institute of Accountants, the American Farm Bureau

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Director, Bureau of Medical Economic Research, American Medical Association.

[1] Postponement of Income Tax on Income Set Aside for Retirement, Hearing Before the Committee on Ways and Means, House of Representatives, Eighty-Second Congress, Second Session, on H. R. 4371, H. R. 4373, H. R. 3456, H. R. 1173, H. R. 5847, and H. R. 7426, May 13, 1952, United States Government Printing Office, 1952.

[2] Footnote 1, page 47. According to the Treasury Department analysis of the original bill, the maximum conceivable revenue loss would be 3.7 billion dollars. Their statement is that "if 25% of the eligible individuals were to take full advantage of the bill, the current revenue loss would be nearly one billion dollars." If you change the 25% to 10%, which I consider a more reasonable estimate (one-third of the eligible persons excluding, on the average, one-third of the amount excludable = ⅓ × ⅓, or, roughly, 10%), the resulting current revenue loss would be 0.25 billion dollars. If we assume that 50% of my excludable amount of one-half billion dollars would be a loss in current revenue, then the difference between our two estimates is the difference between my 0.25 billion dollars and the 0.37 billion dollars. This is not a great difference, and I feel reasonably sure that their estimate could be lowered to mine, or even below mine, if they were basing it on the self-employed alone, or if they were basing it on the revised bill.

A half-billion dollars is 1.5% of the 36 billion dollar income of the self-employed in 1950. (The maximum excludable is 10%.) Since an unknown portion was a return on investment and, therefore, not earned income as defined in the Keogh-Reed bill, a considerable portion of that income would not be excludable under this bill. On this basis a half-billion dollars would obviously be a very high estimate. In 1950 the average income of the 10 million self-employed was $3,400 as compared with $3,200 for all 60 million gainfully employed. Personal savings for all persons in 1950 were $10.7 billion or 4.8% of total personal income. If the self-employed saved 4.8%, they would have saved not more than $1.8 billion. If they had put one-fourth to one-third of their personal savings into restricted retirement funds authorized by this bill, they would have excluded roughly a half-billion dollars, a high estimate when one considers the unattractiveness of the restricted retirement fund as an investment. Few eligible taxpayers under 40 to 50 years of age would consider it an attractive investment. Certainly most of the personal savings would still be invested in other forms, such as buildings, working capital, government bonds, and stocks.

Employers paid $1.56 billion as payroll taxes under the Social Security Act, as amended, in 1951. This was business expense that will never be taxable to either the employer or the employee. Probably taxes are being deferred on at least twice that sum on account of the payments made by employers to pension plans approved under section 165 (a). Thus, by several methods of estimate, the latter sum will be increased by less than one-sixth on account of exclusions by the self-employed under the Keogh-Reed bill. Further study will be required for an estimate of the amount that would be excluded by the pensionless employed who would be eligible under this bill because their employers do not have pension plans.

To me, the most significant point in the Treasury's analysis of the original Keogh-Reed bill is the last paragraph, which I interpret to mean that the Treasury admits that 10 million self-employed taxpayers are discriminated against by being excluded from section 165 (a) and that the Treasury would remedy this obvious discrimination by repealing section 165 (a), thereby jeopardizing the pension expectations of employees under more than 16,000 approved pension plans.

Federation, the Author's League of America, the American Federation of Radio Artists, the National Society of Professional Engineers, the Conference of Actuaries in Public Practice, the Association of Stock Exchange Firms, the Society of Industrial Designers, the American Institute of Chemists, the Investment Counsel Association of America, the United Bondholders of America, the American Veterinary Medical Association, the National Federation of Business and Professional Women's Clubs, and many state and local units of these great national organizations, many of whose members are self-employed because of the dictates of custom and law.

The list of changes made by the revised bills follows. No attempt has been made to arrange these changes in order of importance.

1. Several persons who testified on May 13 noted that the original bill provides only one method by which the excludable amounts could be invested, that is, through a trust fund established by ". . . a bona fide agricultural labor, business, industrial or professional association or similar organization for the exclusive benefit of its participating members. . . ." Accordingly, the new draft provides the eligible taxpayer with the option of purchasing a retirement annuity contract from an insurance company, provided the insurance contract contains the same restrictions as the restricted retirement fund. This additional method would avoid forcing any professional man to belong to a professional society as a condition for obtaining benefits under this measure and would, furthermore, give the large life insurance companies an equal opportunity to interest eligible taxpayers in using insurance company annuity contracts as a method of saving for old age. Moreover, the restrictions in this revised measure are so great that the combined efforts of banks, trust, and insurance companies may actually be insufficient to induce large numbers of eligible taxpayers to forego current consumption and save some of their earned income for old age. With more institutions employed in the development of this voluntary pension system, more persons now outside of pension plans will be encouraged—even urged—to set aside for old age some of their earnings during the working years of their lives. During the past 17 years Congress has repeatedly expressed approval of the general objective of inducing more persons to save for their old age. This voluntary pension system would require deliberate family budgeting of such savings.

2. No subject was discussed oftener during the hearing than the limits on the amount excludable annually, which the original bill has set at $7,500 or 10% of earned income, whichever is the lesser. The members of the committee were impressed with the general argument advanced during the hearing of May 13 that these limits were possibly too generous for the young and too severe for those who are now old. Consequently, the revised draft places an over-all lifetime limitation of $150,000 on the excludable amounts, that is, 20 times $7,500. Also, eligible taxpayers now over 55 years of age may exclude more than 10% of their earned income during the period 1953 to 1972.

Since the normal working period of life comprises about 40 years, a lifetime limit of $150,000 means that the maximum amount of $7,500 could be set aside annually every other year, or during half of the traditional 40 working years, by any eligible taxpayer fortunate enough to earn $75,000 in each of those years. For all practical purposes, this would cut the $7,500 in half and permit $3,750 to be excluded in each of the 40 working years, or the equivalent thereof, by this extremely fortunate person. While effectively cutting this annual limit in half, the revised bill still allows some flexibility; moreover, the amount of pension that the man earning $37,500 a year could finance by excluding $3,750 in each of 40 years will, under this new set of limits, be almost as much as the pension that could be financed by a man earning $75,000 a year who would make only 20 maximum payments during his lifetime. Thus, this new set of limits not only effectively reduces the annual limit but gives those who make less than $75,000 a year an advantage by permitting them to exclude 10% of their earned incomes in more years.

There has also been added under the Special Rule a provision for the exclusion of additional amounts by those who are now between 55 and 75 years of age. In many of the 16,000 pension plans approved under section 165 (a), the employer sets aside large sums of money to finance past-service credits for employees who had worked for him for a considerable length of time before the establishment of a pension plan. A practical counterpart for these past-service credits has been provided under the Special Rule, which would be in effect from 1953 to 1972 and would not apply to eligible taxpayers who will attain age 55 after Jan. 1, 1952. It provides that the amount annually excludable between the 55th and 75th birthdays shall be increased by an amount equal to 1% of the taxpayer's earned net income, or $750, whichever is the lesser, multiplied by the number of full years of his age in excess of 55 determined as of Jan. 1, 1952. But his multiple cannot exceed 20, nor can this additional amount be excluded after the taxable year in which the taxpayer attains age 75. Since the youngest eligible taxpayer is now 56 years of age and will attain age 75 in 19 years, it follows that this provision will expire in the calendar year of 1972. A number of tables presented at the hearing indicated that at the premium rates currently being charged by life insurance companies the maximum monthly cash refund annuity starting at age 75 (not at age 65) that could be financed under this Special Rule would be $1,206 a month—for a person now age 59 who would have an earned income of $75,000 in each of his next 17 years. It is hard to imagine such a fortunate person. Since the lifetime limit of $150,000 also applies to eligible persons over age 55 who would come under this Special Rule, the expected monthly pension starting at age 75 for this person now age 59 would be reduced from $1,206 to $1,013 a month.

Now let us turn from the question of limits on the amounts annually excludable from taxable income to the amount of the pension that could be financed. Tables were introduced during the testimony that revealed that very few younger taxpayers under the General Rule could, at present annuity rates, expect to finance a pension of much more than $1,000 a month starting at age 70. As stated before, under the Special Rule almost no one now over age 55 could finance a monthly pension starting at age 75 in excess of $1,000 a month on a cash refund basis. Thus, the revised limitations will prevent any eligible taxpayer from financing a pension of much more than $1,000 a month starting at age 70 or age 75. Testimony was also given indicating that approximately two-thirds of the pension plans approved in recent years under section 165 (a) permitted maximum pensions of more than $1,000 a month—usually starting at age 65, not at age 70 or 75, when the cost is less—and the remaining one-third of the recently approved plans established a maximum of less than $1,000 a month starting at age 65. It was concluded that the monthly pension target of $1,000 a month provided in this revised draft is a very reasonable counterpart of section 165 (a) for the self-employed and those employed not now covered by pension plans.

Simple arithmetic indicates that a pension of $1,000 a month and $12,000 a year is equal to 16% of the $75,000 of earned income a year that would be the minimum amount that a person would have to earn if he made the maximum contribution of $7,500. Do most industrial pension systems allow a maximum pension of more or less than 16% of average earnings? Only 3% of the new pension plans aim as low as a pension of less than 20% of average earnings. The man earning $37,500 could get a pension equal to 32% of his average earnings, and this 32% would be about the highest percentage that anyone could attain under this bill. Hence, by the test of the percentage relationship between the monthly pension and average earnings, this bill is far more restrictive than section 165 (a) in actual practice.

The bill cannot be considered a "rich man's" bill in any sense of that term. If any better set of limitations had been developed, the authors of the bill would not have proposed a lifetime limitation of $150,000 on the amounts excludable from taxable income. The averaging formulas developed under the regulations promulgated under section 165 (a) have prevented discrimination against low-paid employees. But formulas cannot be applied to an individual. The lifetime limitation is merely the best suggested method of providing the counterpart of section 165 (a). There was no necessity for such a limitation in the language of that section, because it applied to groups. Only by examining the limitations in pension plans approved thereunder can the reasonableness of this lifetime limitation be demonstrated.

3. So much for the maximum pension which is highly theoretical because so few persons could earn $75,000 every year for most of their working lifetimes. We now turn to the average pension. Unrefuted testimony supported by elaborate tables was given on May 13 that estimated the average pension a physician, a lawyer, and a dentist could receive under this revised bill on the basis of the Department of Commerce surveys of the incomes of lawyers in 1947, of dentists in 1948, and of physicians in 1949. The average monthly pension on a cash refund basis starting at age 70 for all members of the profession now under 70 years of age would be $208 for physicians, $106 for lawyers, and $140 for dentists. Furthermore, it was estimated that under the Special Rule for those now over age 55 the average monthly pension at age 75—not age 70—would be $131 for physicians, $121 for lawyers, and $72 for dentists. These average amounts of pension for the members of these three professions will give the members of Congress a much clearer idea of the monthly amount of the typical pension under this revised draft than will any consideration of the maximum theoretical pension. Unfortunately, the Committee on Ways and Means was not given any data on May 13 on average expected pensions for the members of other professions and other groups of eligible taxpayers. This was attributed to the unavailability of income surveys of the other professions. The plain fact is that pensions cost a lot of money, and the members of the Committee were more impressed with the estimated average pensions given at the hearing than with the theoretical maximum pensions in weighing the arguments for and against this proposed voluntary pension system. The amounts excludable annually from taxable income when read separately may still seem large to some persons even with the lifetime limitation of $150,000, but anyone who will examine the probable average amount of pensions under this revised draft will not consider the limits too high. Indeed, as time passes it is probable that the limits may need to be raised in order to encourage this group of more than 10 million gainfully employed citizens to provide adequately for their old age out of their own earnings. They have no employer to pay it for them. This bill would encourage citizens to save some of their own money for old age and to pay taxes on those pensions during their old age.

4. So much for maximum pensions and average pensions expected under this revised draft. The vital question regarding this voluntary pension system is neither of these. The vital question is how much of the maximum pension and how much of the average pension would be attributable to the advantage of tax deferment. Each taxpayer is going to pay for these pensions out of his own pocket. No employer, no unit of government is going to pay him one cent for pensions. No one will be credited with additional amounts in his retirement fund when other employees quit or die. The only gift to the taxpayer, the only way in which this bill would encourage the self-employed and those not covered by pension plans to save earnings for old age during their working periods of life is the enticement of paying lower taxes during the retired years on the pensions as they are received than they would have paid on the premiums (excludable amounts) during their working years, that is, the prospect of reducing total lifetime taxes. On the average, no more than one-fifth, or possibly one-fourth, of these pensions could be considered the real benefit being conferred upon the taxpayer by reason of tax deferment. If this estimate may be accepted for the present, the question before Congress is whether such a benefit of one-fifth or one-fourth of these pensions is sufficient to give these citizens who are excluded from section 165 (a) the counterpart of tax deferment benefits that employees are currently obtaining under that section. It must be stressed that the amount of the pension is four or five times the amount of the benefit that this revised bill would confer upon these taxpayers. After 12 months of study there is still reasonable doubt that the true benefits under this revised bill are actually sufficient to provide equality for those outside the scope of section 165 (a).

The term "expected benefits" is used, for no one can predict the level of income tax rates in future years such as 1973 or 1981. Every taxpayer who would exclude sums under this revised bill is gambling with the future of our income tax rates. Presumably, he would pay smaller taxes during his retired years than during his working years. This statement is reasonable if we are thinking about two men in 1952, one of whom is 45 and now at or near the peak of his earning period of life, and the other 75 and probably retired. We must think about future calendar years, for this bill is concerned with persons as they pass through time, and the actual savings in taxes offered by this bill are highly problematical. Income tax rates have moved upward during the last two decades. It is certainly true that if we turn back the pages 15 or 20 years we could find examples of persons who, if this bill had been available to them 15 or 20 years ago and they had set aside sums of money during their working years in the expectation of paying lower taxes during their retired years starting in 1952, would have found that instead of having a tax savings they would have had a tax loss. Who knows the future of income tax rates?

The option of taking the benefits in a lump sum and paying in turn a capital gains tax is provided in section 165 (a). Therefore, it has been provided in this bill. The grave uncertainties about the future level of income tax rates warrant this capital gains option. For that matter, the future level of the capital gains tax rate is also uncertain.

5. In the original bill any taxpayer covered by a pension plan was permitted to exclude under the bill the difference between the limits of (10% and $7,500 described above) and the amount his employer was currently paying into a pension fund approved under section 165 (a). Testimony given at the hearing indicated that such a provision would be extremely difficult to administer. In the first instance, many employers do not actually know how much they are contributing annually to the credit of their younger employees, especially before the time when the employer's contributions are vested in the employee. Many approved pension plans provide that, if the employee does not continue in the service of the employer until retirement, any contributions made by the employer to the credit of the severing employee shall be distributed by formula to the credit of the continuing employees. Thus it would be very difficult for such an employer to determine—even if required to report annually on something like a W-2 form—the current contribution to the pension fund for a named taxpayer. Furthermore, many taxpayers not employed by taxable employers, such as civil servants, firemen, policemen, school teachers and employees of charitable, religious, and scientific organizations, are covered by pension plans that do not need to be approved under section 165 (a). In the interest of simplicity it seems wise to exclude from this revised draft all persons who are covered by any kind of a pension plan, especially the younger employees, who can expect to be covered after completing a specified number of years of service and those who have actually retired and are now receiving pension payments under such plans. It is true that their exclusion will not produce equity in every case, but excluding them seems imperative. There was even some discussion at the hearing of excluding all employed persons and restricting the provisions of this bill to the 10 million self-employed. The authors of the bill did not go that far, because they realized that there are many employed persons, especially those who are employed for short periods of time, who are not now and who probably never will be covered by employer-employee pension plans. Hence the coverage of this revised bill is a compromise between the extremes of covering every taxpayer and covering only the self-employed.

6. There are thousands, perhaps millions of taxpayers who have extreme fluctuations in income. Some of these and other taxpayers might find it impossible to set aside reasonable amounts necessary to finance retirements unless some carry-over provision were developed. Hence an entirely new section was introduced, section (4), which provides for a carry-over of unused exclusions for a period of not more than five years. No additional exclusions from taxable income made under the Special Rule in the revised bill by those now over 55 years of age can be made under the carry-over provision; that is, the carry-over provision applies only to contributions made under the General Rule, most of which will be made by younger persons. The hardship that this carry-over provision is designed to alleviate is found extensively in the entertainment field and among self-employed

persons engaged in architecture and engineering. This is not to say that there are no other groups that have described their need for this type of provision, which, as noted above, has been carefully limited to a five year period and must function within the limit of $7,500 per year.

7. Last, we shall consider the severe restriction on the distribution of the proceeds of the restricted retirement fund and exactly similar restrictions on the restricted retirement annuity. In simple language, these provisions would prohibit any withdrawal from these savings funds until the taxpayer reached age 65. In the original bill the age was 60; several members of the Committee on Ways and Means indicated a preference for age 65, the retirement age specified in the Social Security Act. This restriction will greatly deter the use of the provisions of this bill and will certainly confine its use to those who sincerely and honestly want to save for old age and for no other reason. Consider a lawyer aged 40 and his wife contemplating setting aside 10% of his earned income under this measure. First, they would have to decide that they did not want to spend the money, as there would be no regular deduction from their monthly pay check by the accounting department of a corporation. Thus the decision to save would have to be made deliberately. Second, they would realize that they could not touch this fund for any of the financial crises that the family might encounter before the husband reached age 65, 25 years hence. Would that seem to you to provide a serious temptation to abuse the privileges provided by this bill? If you will think back over your own lives, you might conclude that you would have preferred paying the income tax currently and then investing the remainder in some fund that you could tap for the ordinary financial crises that your family might encounter, for the ordinary rainy days for which persons have always saved.

Because of the unattractiveness of this investment opportunity, it was provided that, in the event of permanent and total disability continuing for a period of more than three months, the taxpayer would be permitted to make withdrawals from his savings. There are many financial crises in the family other than the disability of the breadwinner, but withdrawals cannot be made in order to meet any of these other types of crises. Time and experience may demonstrate that this restriction is so severe that it will have to be modified to encourage eligible taxpayers to save for old age. It is one of the most important reasons back of the contention that the use of the provisions under this bill will be so limited that the Treasury will have little reason to worry about the loss of current revenue.

There is, of course, the possibility of repealing section 165 (a) in order to provide equity between the persons not covered by the pension plans and those who are covered by pension plans; or, in other words, between those who now enjoy tax deferment privileges in the financing of their pensions and those who do not. But the repeal of section 165 (a) does not seem to merit any serious consideration. The repeal of that section would destroy most of the 16,000 pension plans and darken the future of millions of loyal employees who are confidently expecting pensions under their company pension plans. Such a solution seems cruel. Rather the solution is to provide for the self-employed and those not covered by any kind of pension plan the privileges of tax deferment that persons covered by pension plans enjoy and expect to continue to enjoy.

Our problem is to rectify the error of 1942, the error Congress made in excluding the self-employed from the tax deferment pension benefits of section 165 (a). We have given a cold shoulder to 10,000,000 self-employed persons. Our problem is also to provide reasonable pension opportunities for employed persons not actually covered by pension plans 10 years later. Until we close this gap we shall have failed to complete the new social theory enunciated in 1935 and implemented in 1942. The heart of this new social theory is that pensions are good and that the Federal government should encourage them. All would agree that pensions are good. But many would not want the federal government to encourage pensions. It seems to me, however, that none can disagree that, if the federal government does encourage pensions, it should encourage pensions for all and not merely for those who are employed by employers who have established pension plans.

COUNCIL ON MEDICAL SERVICE

This is the second in a series of studies made by the Committee on Indigent Care of the Council on Medical Service concerning local plans for medical care of the indigent. A general introduction to the series and the first of the Committee's studies (Erie County, New York) appeared in the May 10, 1952, issue of The Journal, *pages 188-191.*

MEDICAL CARE FOR THE INDIGENT IN MADISON, WISCONSIN

This report is a study of medical care available for indigent and medically indigent residents of the city of Madison in Dane County, Wisconsin. Dane County has a population of approximately 165,000 and is primarily a farming and dairying county, with some small industries. Including the 14,000 students at the University of Wisconsin, 100,000 persons, 61% of the county's population, reside in Madison, the state capitol and county seat and the main business center of the county.

Public assistance is provided on a county-wide basis, but each of Dane County's 60 political subdivisions is responsible for general assistance to its own indigent residents. For this reason, a study of welfare medical care in Madison is considered more practical than a survey of the entire county's programs.

Table 1.—*Estimated Average Monthly Coverage, Dane County and Madison, 1951*

	Dane County	Madison
General Assistance (including medically indigent)	377*	230
Public Assistance	3,651	2,227*
Old Age Assistance	2,175	1,327*
Aid to the Blind	66	40*
Aid to Dependent Children	1,350 (600 cases)	823*
Aid to Permanently and Totally Disabled	60	37*

* Estimate, based on percentage of county's population resident in Madison.

ELIGIBLE POPULATION

Residents of Madison are eligible for general assistance medical care when admitted to the welfare department's relief rolls or deemed medically indigent by the department. An average of 230 residents received such aid monthly in 1951.

An estimated 3,651 persons in Dane County received aid monthly from public assistance programs in 1951. In table 1 are given figures on the various types of assistance, with the proportion of the population aided by each program assumed as the same in city and county.

According to the estimate in table 1, approximately 2,457 Madison residents per month, about 2.5% of the city's population, received general or public assistance and were therefore eligible for medical care at public expense in 1951.

ADMINISTRATION

The general assistance program is administered in Madison by the city welfare department, which determines eligibility of both indigent and medically indigent clients. Each client's eligibility is certified monthly. The welfare department is also in charge of planning and authorization of benefits, subject to the approval of the city council, to which the department makes monthly and annual reports. Any appeals by clients are made to Madison's director of welfare.

The public assistance program for all of Dane County is supervised by the Dane County pension department. Eligibility for aid to the blind and aid to the permanently and totally disabled is determined by the state department of public welfare. The county agency determines eligibility for old age assistance and aid to dependent children and is responsible for the planning, authorization, and approval of bills for all public assistance programs. Certification of clients lasts at least a year before a recheck is necessary. The county pension department reports to the county board of supervisors and to the state department of public welfare. Clients may appeal county decisions to the state authorities, who must, by law, provide a "fair hearing." Both programs provide for control of abuses by patients; for example, a patient who changes physicians too frequently can be required to choose a single physician to treat him for the duration of his illness.

Complaints against physicians are referred to the Dane County Medical Society. The society's medical coordinator reviews complaints concerning fees or refusal to accept welfare cases and can, if necessary, refer the complaint to the society's board of censors for further consideration as to disciplinary action.

SERVICES AVAILABLE

Benefits provided by the general and public assistance programs are sufficiently alike to be discussed jointly in this report. All health services are potentially available to anyone on any of the programs and are generally provided on a "free choice" basis. Each person is treated according to his needs, and the extent and course of treatment is usually decided on by the client's physician. Home and office calls are provided by physicians on a free choice basis. Specialists' care, in or out of the hospital, is provided on referral by the attending physician. The patient may be referred to a specialist or to a group clinic of his choice.

Madison does not have a city or county hospital; as a result, hospitalization, when recommended by a physician, is available on a free choice basis. The patient may be admitted to the hospital without prior authorization from welfare agencies, and has free choice of a physician for in-hospital care. Provisions for a physician's first visit and for hospitalization without prior authorization insure that there will be no time lapse between the request and receipt of most services.

Madison has no public clinics or hospital outpatient clinics except some specialized clinics maintained by the health department. There are approximately 15 private group practice units located in various parts of the city to which welfare clients may go if they wish to do so. Welfare patients are treated by physicians in the same way as private patients. Any recommendations for services or appliances made by the attending physician are authorized. Drugs, as prescribed by the attending physician, are provided by private pharmacies and paid for by the authorizing agency. Home nursing and invalid home care are also available when necessary or advisable.

A person who needs funds because of illness and who is not eligible for either general or public assistance may receive help from the Community Chest agency in Madison. This agency will help pay for both medical care and hospitalization. Patients are usually referred to the Chest agency by the hospitals, so that a double check is obtained as to eligibility and need.

PROVIDERS OF SERVICES

The patient may be treated by any one of the approximately 300 physicians in Madison, except the nonpracticing staff members of the University of Wisconsin Medical School. The physician need not be a member of the Dane County Medical Society to treat welfare patients, and no participation agreement is required. The client has free choice of physician, even during hospitalization, and has free choice of specialist if specialized treatment is advised by the attending physician. Fifteen group practice units in Madison also participate in the program.

Madison's health department operates well-baby, immunization, and chest clinics, which are open to anyone, whether or not referred by a physician. The health department clinics perform no therapy; the chest clinic is diagnostic and performs fluoroscopic examinations. The health department also employs a full-time health educator whose services are available to any agency interested.

All hospitals in Dane County may accept welfare clients. The general hospitals in Madison and the number of beds in each are given in the following tabulation.

Madison General Hospital	177
Methodist Hospital	132
St. Mary's Hospital	230
Wisconsin General Hospital	750

Wisconsin General Hospital is primarily a teaching institution, maintained by the state; however, it will accept welfare patients from any county in Wisconsin. Each county is allotted a quota for the care of public patients at the state hospital, and the cost of care for these patients is shared by the state and the county. When the quota is exhausted, the county assumes full financial responsibility for any additional public patients admitted to the hospital. There are two nursing homes in Madison itself and approximately 11 in the rest of the county. Home nursing is provided on a contract basis by the Visiting Nurse Service in Madison.

PAYMENT FOR SERVICES

Payments to private physicians are made according to a fee schedule developed by the Dane County Medical Society and the city welfare department and accepted by the county pension department for the whole county. Fees are approximately 80% of conservative normal fees and cover all phases of surgery and medicine, including in-hospital physician services.

The fee for home calls is $3.00 during the day and $4.00 after 7 p. m., with a $2.00 fee for each additional patient seen during the visit. Office calls are $2.00 each. The physician is paid for visits to a hospitalized patient at a rate of $2.00 per visit for the first 10 days, $1.00 per visit for the next 20 days, and $1.00 per visit for the first 10 days of each succeeding month. Physicians who treat general assistance patients submit their bills to the city welfare department. The department sends the money for payment to the medical society, which pays the physician according to the fee schedule, less 5% for administrative costs.

Physicians providing care under the aid to dependent children program are paid directly by the county pension department. Old age assistance and aid to the blind clients who require medical care receive an increase in their monthly aid checks and are expected to pay the physician out of this amount; however, when the grant is used up, the pension department pays any further medical costs directly to the physician. There is no supplementary medical budget for aid to the permanently and totally disabled. Clients of this program receive their medical services as general assistance patients, and the service is paid for by the city welfare department.

Pharmacies are paid directly for prescriptions filled for all classifications of welfare clients and hospitals are paid by the welfare agencies according to fixed *per diem* rates.

COSTS AND FINANCIAL SUPPORT

The general assistance program is supported by city tax funds. The state and county governments share the medical costs of public assistance; the state provides 35% of these funds, and the county provides the rest. Indigent persons who have lived in Wisconsin less than one year and need medical assistance receive care at the expense of the state department of public welfare.

All appropriations are made annually. Budgets for general assistance must be approved by the city council and those for public assistance by the county board of supervisors. Supplementary budgets may be submitted if the funds allocated are insufficient. The agencies are required by law to supply adequate funds; the city must bond itself, if necessary, rather than curtail its program.

The cost figures given in table 2 are for the first six months of 1951. Official figures for public assistance expenditures are available for Dane County only; however, Madison has 61% of the county's population, and, for the purposes of this study, city expenditures have been assumed to be approximately in proportion to population. As previously noted, medical care for the permanently and totally disabled is provided from general assistance funds.

TABLE 2.—*Costs of Welfare Programs*

	Dane County	Madison
General Assistance	....	$ 22,773.45
Public Assistance	$199,896.82	121,937.06 *
Old Age Assistance	149,250.29	91,012.68 *
Aid to the Blind	2,619.25	1,597.74 *
Aid to Dependent Children	48,027.28	29,296.64 *

* Estimate, based on percentage of county's population resident in Madison.

With the estimated figures as a basis, the total amount expended on welfare medical care in Madison during these six months would be approximately $144,710.51, or almost $300,000.00 a year.

Following are estimates of the average monthly cost of medical care in Madison for each of the four programs and for each eligible client, as well as the monthly cost of some of the major items provided. For the purposes of this tabulation, the 37 clients estimated as eligible for aid to the permanently and totally disabled have been included with those eligible for general assistance.

	G. A.	O. A. A.	A. B.	A. D. C.
Estimated monthly cost.......	$3,795,58	$15,173.78	$266.29	$4,882.77
(Estimated number eligible)..	(267)	(1,327)	(40)	(823)
Cost per eligible person.......	14.22	11.44	6.61	5.93
Physician's care	4.08	2.60	1.38	2.45
Drugs	1.71	1.12	1.53	0.81
Hospitalization	5.37	2.77	0.20	0.91
Nursing home care........	2.49	4.34	2.64	....

The average cost of medical care per eligible client for Madison's entire welfare program during this period, based on the above figures, was about $9.82 per month.

MEDICAL SOCIETY RELATIONSHIP

The medical coordinator of the Dane County Medical Society occupies a key position in the city and county medical aid program. His duties are "to make certain that the patients, the doctors and the various agencies are mutually satisfied with the services and the allowances" agreed to by the medical society and the welfare agencies. The coordinator, who serves without pay, acts on behalf of the medical society in controlling abuses by physicians, reviewing complaints, and helping to settle disputed fees. He serves as liaison officer between the medical profession and the welfare agencies and, if necessary, may refer cases directly to the society's board of censors. Much of the success of the total program is due to the work of this office.

It is largely through the efforts of the Dane County Medical Society and its coordinator that the patient is assured of completely free choice of a physician not only for home and office but also for hospital care. The fee schedules established by the society and the welfare agencies provide for fair reimbursement for all services provided by the physician, with compensation according to the treatment given.

It is noteworthy that treatment can be provided by any physician in the county and that no participation agreement is required. Thus, the choice of physician is completely free, not even limited to society members, although the society has a major part in the program through the fee schedule and the coordinator and is used by the city welfare department to distribute payments to the individual physicians.

SUMMARY

The medical aid program for recipients of general assistance and public assistance in Madison achieves reasonably complete unity and coordination through the office of the medical coordinator of the Dane County Medical Society. The services offered under the two programs are almost identical, and payments to physicians are in accordance with a fee schedule agreed on by the welfare agencies and the medical profession. The clients of any relief program are eligible for home and office care, hospitalization, home nursing, convalescent care, and prescribed drugs. As a result, relief clients have potentially available to them every medical service provided in the county, and, because the services are the same for all relief groups, there is little incentive to "jump" from one program to another in order to obtain more and different types of medical service.

For those who are unable to meet the qualifications of general or public assistance but who need aid to pay for medical care, the Madison Community Chest agency has a program of financial assistance. Thus, any person in Madison who finds it impossible to provide himself or his family with medical care has some program of assistance to which he may turn.

A number of features of the Madison program are especially noteworthy. A completely free choice of physician, with the same physician caring for the patient over a period of time and when hospitalized, is the best assurance of a good quality of medical care to the indigent. The fact that authorization is not required before a patient calls a physician means that there will be no gap between the request for and receipt of services. In addition, physicians are paid for all services, thus placing the indigent on a par with private patients and eliminating any stigma that might be attached to indigency.

The role of the medical society of Dane County is admirable. It not only negotiates agreements between official agencies and physicians and sees to it that the physicians give service to all indigents under the terms agreed on but it polices its own membership. The only weakness here is that the control of abuses by physicians is vested primarily in one man. At the present time this man handles the position extremely well. His honesty and fortitude of character are especially suited for this responsibility. It would seem, however, that since the medical coordinator's job is such an all-important cog in the machinery his authority should be shared by a committee of no less than three, so that, in the event one person is unable to carry on, the program can be assured of continued strength and continuity of policy.

From the client's standpoint the program is certainly successful in providing complete medical care with a minimum of delay and red tape. The main objective of the program is to render adequate medical care to relief clients, and in this there can be little doubt that Madison has succeeded.

BUREAU OF LEGAL MEDICINE AND LEGISLATION

MEDICOLEGAL ABSTRACTS

Devices: Chiropractor Not "Physician" Under Federal Food and Drug Act.—This was a proceeding by the United States for the condemnation of 22 devices known as Halox Therapeutic Generators. The case was heard in the United States district court, southern district, California.

A Halox Therapeutic Generator is a device for the electrolysis of a sodium chloride solution. It is housed in a leatherette-covered plywood cabinet, its base being approximately 12 in. by 15 in. and its height approximately 12 in. At the front of the cabinet is a control panel. Inside the cabinet there is a glass jar partly filled with a saturated sodium chloride solution into which carbon electrodes extend, and when the generator is in operation electricity is carried to these electrodes. As a result of the electrolysis of the salt solution, chlorine gas is produced. A small electric fan blows a current of air through the jar and out through a rubber hose. Thus a mixture of air and chlorine gas goes into the tube. This mixture is then administered to patients receiving the "chlorine inhalation therapy" by having the patient hold the rubber tube to his nose and inhale.

Administrative regulations issued pursuant to the Federal Food, Drug, and Cosmetic Act exempt from the labeling requirements devices that are delivered to physicians for use in their professional practice, provided information adequate for the use of such a device by the physicians is readily available. All, save 1, of the 22 devices involved in this suit were shipped to W. G. Keys, who held a valid license as a chiropractic physician from the state of California. Keys maintained several offices in Los Angeles County at which these devices were used by him in his practice. The remaining generator was in the possession of another chiropractor, C. J. Henaghan. At the outset, said the court, it is clear that the generators did not bear adequate directions for use. In fact, they bore no directions for use. This was admitted by the claimant who argued that the devices were exempt from such requirement by virtue of having been shipped or delivered to physicians to be dispensed by physicians in their professional practice. It is at once apparent, continued the court, that the claimant can succeed only if a chiropractor is a "physician" within the meaning of the regulations. The regulations define "physician" as "only those physicians, dentists and veterinarians who are licensed by law to administer or apply such drug or device." Thus the persons to whom the devices were shipped must, first of all, be physicians. Only if they are physicians is it necessary to inquire into the matter of their authority to administer or apply the device.

In its broadest sense, said the court, the term "physician" includes anyone exerting a remedial or salutary influence, as a physician of the soul; however, the court did not believe that the term as used in the regulation was intended in the broad sense. In Webster's New International Dictionary, 2nd ed., 1949, one definition of "physician" is: "A person skilled in physic or the art of healing." In the same volume "chiropractic" is defined as "a system for the practice of adjusting the joints, especially of the spine, by hand, for the curing of disease." Thus a chiropractor is one skilled in the art of healing, in a limited manner, although not one skilled in physic, since this latter term refers to the practice of medicine. In Webster's dictionary a second definition of "physician" is: "one duly authorized to treat diseases, especially by medicines; a doctor of medicine; often distinguished from a surgeon." The chiropractic law of California provides in part: ". . . any licensee under this act who uses . . . the term 'physician' . . . or any other letters, prefixes or suffixes, the use of which would indicate that he or she was practicing a profession for which he held no license from the State of California, . . . shall be guilty of a misdemeanor. . . ." It follows, said the court, that one who is licensed to practice chiropractic in the state of California is not a physician by virtue of such license. The record is devoid of any evidence attributing qualifications as physicians to the chiropractors concerned here.

There is, in addition, a more compelling reason for holding that the seized generators were not exempt from the requirement that they bear adequate directions for use. The exemption regulations define a physician as a physician who is "licensed by law to administer or apply" the drug or device in question. Thus, even if the term "physician" is broad enough to include chiropractors, it includes only those chiropractors who are licensed by law to administer or apply the drug or device. The authority of a licensed chiropractor is defined by law which provides: "One form of certificate shall be issued by the board of chiropractic examiners, which said certificate shall be designated 'License to practice chiropractic,' which license shall authorize the holder thereof to practice chiropractic in the State of California as taught in chiropractic schools or colleges; and also, to use all necessary mechanical, and hygienic and sanitary measures incident to the care of the body, but shall not authorize the practice of medicine, surgery, osteopathy, dentistry or optometry, nor the use of any drug or medicine now or hereafter included in materia medica." The scope of this authority, said the trial court, has not been defined by the supreme court of California, but appellate courts of California have had occasion to consider the question. On the basis of their decisions it is clear that a chiropractor licensed by the state of California is not authorized to treat diseases and ailments by mechanical means. As we have seen, the parties are agreed that the generators under seizure were intended for use in the conditions of arthritis, sinusitis, hay fever, and bronchitis. All these conditions are diseases and as such could not be treated by chiropractors by means of the Halox Therapeutic Generator. It follows that the seized generators were not exempted by the regulations, since they were not shipped to physicians who were licensed by law to administer them.

There is yet a third reason, said the trial court, why the seized devices were not exempted by the administrative regulations. Those regulations require that information adequate for the use of such device by physicians be readily available. By affidavit, Dr. Thienes, M.D., chairman of the department of pharmacology at the University of Southern California Medical School, stated that the use of chlorine gas in an effective antiseptic concentration would be too irritating to be withstood by the average person and that concentrations less than this are ineffective for any purpose. He stated that chlorine gas to be safely inhaled for even a short time requires a concentration of less than 10 ppm. The results of tests made by a chemist employed by the Food and Drug Administration indicated that a constant output of such a safe amount was obtained at only two dial settings. Yet nowhere is it shown that directions were available suggesting such dial settings. Furthermore, even at these settings the chlorine output varied throughout a three hour test period. Thus it is clear, the court concluded, that adequate directions were not available for the use of the generators.

Accordingly, the court held that the 22 Halox Therapeutic Generators must be condemned and disposed of by destruction in accordance with the provisions of the Federal Food, Drug, and Cosmetic Act. *United States v. 22 Devices, More or Less, Halox Therapeutic Generator, 98 F. Supp. 914 (1951).*

MEDICAL MOTION PICTURES

The Use of the Microscope: 16 mm., color, sound, showing time 19 minutes. Prepared by Dr. Merrill J. Shepro, University of Illinois. Produced in 1949 by and procurable on rental ($15) or purchase ($175) from Scientific Film Co., 6804 Windsor Ave., Berwyn, Ill.

This is a textbook type film designed for teaching students the correct and efficient manipulation and care of the microscope. It is composed of the following 12 text units: basic parts of the microscope; optical principles; types of microscopes; light source adjustment; adjustment of optics; slide preparation; correct sitting posture; technique of microscopy; oil immersion technique; cleaning of lenses; care of the microscope; and common mistakes of beginners.

In an effort to produce a textbook type of film in 20 minutes time, certain important phases have been omitted, some are incomplete, and others have been unduly emphasized. Since a great deal of the subject matter does not lend itself to the motion picture medium, most of the information presented is contained in the narration. It would therefore be impossible to digest all the material in one showing.

The following demonstrations are well done: method of determining the source of dirt; operation of field and aperture diaphragms; adjustment of the condenser; and adjustment of the intensity and the illuminated area.

In addition to the general criticisms, the specific point of indicating that a beginner should remove the objective while cleaning it can also be criticized. There is also a lack of clear labeling of parts. The photography is good; but the narration is constant and rapid, and the narrator's voice tends to be somewhat monotonous. The film may be of use in stimulating interest in the use of the microscope for the beginner or as a review for the advanced student.

Transplantation of the Vertical Recti for Lateral Rectus Paralysis: 16 mm., color, silent, showing time 14 minutes. Prepared by Conrad Berens, M.D., and Louis J. Girard, M.D., the Ophthalmological Foundation, New York. Produced in 1951 by the Photography Department of the New York Eye and Ear Infirmary. Procurable on loan or purchase from the Ophthalmological Foundation, Inc., 314 E. 14th St., New York 3.

An operation to provide lateral movement of the eye in the field of operation of a paralyzed lateral rectus muscle is described, and diagnosis of the condition is made by examination and history. The operation consists of recession of the medial rectus about 5 mm., resection of 7 mm. of the lateral rectus, and splitting of the superior and inferior recti tendons from their insertion into the sclera backward 5 to 7 mm. The lateral half of the superior and inferior recti muscles is attached, together with the resected lateral rectus, at the site of the former lateral rectus insertion.

The operation is done in such a manner as to preserve the integrity of Tenon's capsule. The tendons are reattached to the sclera with absorbable sutures, and the conjunctiva is closed with running silk suture. An illustrative patient is a young girl who shows lack of rotation of the left eye in the field of action of the left lateral rectus muscle before operation. Following operation, movement of the eye in this field is adequate and, apparently, conjugate with the right eye.

The film is designed to show operative technique and also to demonstrate the changes in action of the lateral half of the superior and the inferior recti from adduction to abduction. The photography is very well done. The film would be useful in teaching students of ophthalmic surgery and is recommended for residents and ophthalmological societies.

MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A.M.A. Arch. Dermat. and Syphilol., Chicago

65:379-510 (April) 1952

Human Melanogenesis: Tyrosinase Reaction in Pigment Cell Neoplasms, with Particular Reference to Malignant Melanoma: Preliminary Report. T. B. Fitzpatrick.—p. 379.
Stress Studies in Eczema-Asthma-Hay-Fever Diathesis. T. H. Sternberg and M. C. Zimmerman.—p. 392.
Pituitary Adrenocorticotropic Hormone (ACTH) and Cortisone in Diseases of Skin: II. Allergic and Other Dermatoses; Report of 67 Cases. J. G. Hopkins, B. M. Kesten, C. T. Nelson and others.—p. 401.
Acute Forms of Pemphigus. H. E. Michelson.—p. 422.
Chronic Pemphigus Vulgaris. F. E. Senear.—p. 429.
Genetic Aspects of Malignant Melanoma. E. P. Cawley.—p. 440.
Amyloidosis Cutis: Macular Variant. L. L. Palitz and S. Peck.—p. 451.
Localized Acquired Hypertrichosis. A. C. Ressmann and T. Butterworth.—p. 458.
Asterol® Dihydrochloride Therapy of Microsporum Audouini Scalp Infections. W. Snyder.—p. 464.
*Accidental Tattooing of Face Treated by Abrasion with Sandpaper. W. A. Rosenberg.—p. 466.
Linear Unilateral Basal-Cell Nevus with Comedones: Report of Case. R. G. Carney.—p. 471.
Genetic Study of Hereditary Type of Epidermolysis Bullosa Simplex. R. O. Noojin, J. P. Reynolds and W. C. Croom.—p. 477.

Abrasion with Sandpaper in Treatment of Accidental Tattooing.—A truck driver requested treatment for a bluish tattooing of his face, which had been caused about seven months before by an explosion of the gas tank of his truck. There were streaks and discrete bluish pigmented spots and patches on the left side of the face, especially the forehead and the infraorbital region. His co-workers frequently teased him for having a black-and-blue eye. An area about 2.5 cm. in diameter on the left cheek was anesthetized with procaine hydrochloride, and the skin over the area was treated by abrasion with sandpaper, no. 000, until bleeding was profuse. Within 48 hours a crust formed, which fell off in about five days, leaving a smooth pink surface. In about 10 to 14 days the area healed. Similarly involved areas were treated at weekly or biweekly intervals. Two months after the last treatment, examination showed excellent results and no cicatrization.

A.M.A. Arch. Pathology, Chicago

53:281-390 (April) 1952

Arteriosclerosis in Dog: I. Spontaneous Lesions in Aorta and Coronary Arteries. S. Lindsay, I. L. Chaikoff and J. W. Gilmore.—p. 281.
Methylcholanthrene-Induced Tumors in Guinea Pigs with Review of Literature on Tumors Induced with Polycyclic Carcinogenic Hydrocarbons in This Species. W. O. Russell and L. R. Ortega.—p. 301.
Atrophy of Left Lobe of Liver. E. J. Benz, A. H. Baggenstoss and E. E. Wollaeger.—p. 315.
Renal Siderosis in Rabbits Following Injections of Hemoglobin and Organic Iron. D. Bloom, L. H. Westman and J. J. Lalich.—p. 331.
Mobilization of Mucoprotein by Parathyroid Extract. M. B. Engel.—p. 339.
Studies on Hypersensitivity Arteritis in Rabbits: I. Effect of Acute Arteritis on Blood Pressure. II. Observations on Animals Subjected to Numerous Large Injections of Horse Serum. J. L. Orbison.—p. 352.
Study of Death of Irradiated and Nonirradiated Cells by Time-Lapse Cinemicrography. R. Schrek and J. N. Ott Jr.—p. 363.

American Journal of Medical Sciences, Philadelphia

223:349-468 (April) 1952

*Early Diagnosis of Cancer of Pancreas Based on Clinical and Pathological Study of 100 Autopsied Cases. R. K. Brown, V. Moseley, T. D. Pratt and J. H. Pratt.—p. 349.
Use of Dextran in Treatment of Blood Loss and Shock. J. S. Wilson, E. H. Estes Jr., J. T. Doyle and others.—p. 364.
*Streptomycin Therapy in Pulmonary Tuberculosis: Controlled Study. A. Goldman, B. Friedman and L. Sale Jr.—p. 370.
Comparison of Resting Electrocardiogram, Exercise Electrocardiogram, and Ballistocardiogram During Prolonged Periods of Observation. A. Soffer, P. N. G. Yu, M. A. Epstein and E. S. Olsan.—p. 378.
*Clinical Significance of Small Benign Gastric Ulcer, with Note on Benign Ulcer of Greater Curvature and in Absence of Free Hydrochloric Acid. E. D. Palmer.—p. 386.
Familial Nature of Amino-Aciduria of Wilson's Disease (Hepatolenticular Degeneration). L. L. Uzman and B. Hood.—p. 392.
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Plasma Antithrombin Determination: New Test for Cancer of Pancreas Associated with Jaundice. I. Innerfield and A. Angrist.—p. 422.
Survey of Epidemic Typhus Antibody Levels in Bloods of Individuals Born in Eastern and Central Europe and the United States. M. M. Sigel, L. B. Weiss, N. Blumberg and J. C. Doane.—p. 429.
Erythema Nodosum: Survey of Some Recent Literature. H. Beerman.—p. 433.
Industrial Noise: Its Medical, Economic, and Social Aspects. M. S. Fox.—p. 447.

Early Diagnosis of Cancer of Pancreas.—The clinical records of 100 cases of cancer of the pancreas in which necropsy was performed showed that in 55 cases the symptoms or the findings were suggestive of cancer of the pancreas, while in 41 no evidence of pancreatic disease was revealed by the history, physical examination, or laboratory tests at the time of admission to the hospital. In 73 cases necropsy showed that cure was impossible at the time the patient was examined, either because metastases had developed or because the disease had extended locally to such an extent as to be inoperable. The diagnosis of cancer of the pancreas must be made early if surgical intervention is to offer any hope of cure. If pain in any part of the abdomen, either paroxysmal or constant, has developed recently, there has been an unaccountable loss of weight, and the results of physical examination and gastrointestinal roentgen ray studies are negative, a probable diagnosis of cancer of the pancreas is justified. In early cases of cancer of the body and tail there is no diminution of the pancreatic juice, and hence, if the patient is to be saved, the radical operation must be undertaken when only the symptoms mentioned are present. Bulkiness of the stools indicates absence from the intestine of pancreatic juice, as in no other condition are the stools so massive. Visible fat resembling butter or cream on the surface of the stools or in the surrounding fluid is pathognomonic of pancreatic disease. In order to demonstrate this evidence of pancreatic insufficiency the diet should contain a considerable amount of fat. When limited amounts of fat are fed, there is usually no visible fat on the surface of the stool. The presence of fat, fatty acids or soaps gives the feces a clay color, which is not caused by the absence of bile, as is usually thought. The significant finding on microscopic examination is the presence of large numbers of undigested muscle fibers, which may be studied by feeding the patient daily 100 to 150 gm. of finely chopped beef or ham given raw or lightly broiled. The secretin test will demonstrate deficiency of pancreatic juice. Serum lipase and serum diastase tests, although usually negative in cancer of the pancreas, should be employed whenever possible because, if positive, they are diagnostic of pancreatic disease. Contrary to the commonly accepted view, fever and pronounced leukocytosis are often present in advanced cases of cancer of the pancreas. Liver function tests and the demonstration of occult blood in the stools are misleading and needlessly delay operation.

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Titles marked with an asterisk (*) are abstracted.

Streptomycin Therapy in Pulmonary Tuberculosis.—Of 85 patients with pulmonary tuberculosis, 42 were treated with streptomycin and 43 served as controls. Each patient in the streptomycin group received 0.1 gm. of the drug per 11 lb. (5 kg.) of body weight daily, with a maximum dose of 1.5 gm. The total daily dose was divided equally and given at eight hour intervals for 91 consecutive days. Follow-up observations were made at 3 month intervals for 12 months. The treated group showed greater improvement with respect to increase in body weight, decrease of fever, and decrease in sputum weight. Roentgenograms revealed definite improvement in the streptomycin-treated group. The proportion of sputum conversions was definitely greater in the streptomycin-treated group. The greatest improvement occurred immediately after three months of streptomycin therapy. Resistance appeared in approximately one-half of the treated patients after three months of streptomycin therapy. At the end of one year all available cultures in the treated group showed drug resistance.

Small Benign Gastric Ulcer.—Of 100 patients with small benign gastric ulcers measuring no more than 1 cm. in diameter, 20 also had a duodenal ulcer and 1 had peptic ulceration in four locations, including the upper gastrointestinal tract. Five patients had two or three gastric ulcers. Achlorhydria was observed in 10 patients, no free acid being demonstrated on at least two occasions following histamine stimulation plus a 12 hour nocturnal continuous drainage. Of nine patients with ulcers of the greater curvature, six had ulcers in the antrum and three in the middle portion. There were two deaths attributable to the lesions, and important complications occurred immediately prior to or during hospitalization in 45, including severe hemorrhage in 30 and perforation in 12. Surgical treatment was required in 21 for reasons other than suspected carcinoma. Small benign ulcers are important clinically because of the danger of serious complications.

American Journal of Psychiatry, New York

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Long-Term Prognosis in Mental Illness: 30-Year Follow-Up of 141 Mental Patients. W. L. Holt Jr. and W. M. Holt.—p. 735.
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American Journal of Surgery, New York

83:489-612 (April) 1952

Proctologic Manifestations of Carcinoma of Prostate. R. J. Jackman and J. R. Anderson.—p. 491.
Oleogranulomas of Rectum: Following Rectal Instillation of Petrolatum or Ointment Containing Petrolatum. D. A. Susnow.—p. 496.
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*Survey of Fires and Explosions in Hospitals of the United States. B. J. Ciliberti and P. M. Wood.—p. 527.
Herniated Lumbar Disc. S. A. Grantham.—p. 531.
Antithrombin Titer in Pancreatic Cyst. I. Innerfield, A. Angrist and J. W. Benjamin.—p. 538.
Plasma Preparation Simplified for Surgical Use. I. M. Tarlov, P. S. Entmacher, R. Day and A. Martin.—p. 544.
New Approach to Problem of Postoperative Pain. A. H. Iason and H. E. Shaftel.—p. 549.

Fires and Explosions in Hospitals.—Investigation of fires and explosions is difficult, since many institutions are reluctant to give information because of legal entanglements and pending court action. However, an effort was made to review as many as possible of such accidents that occurred in hospitals in the 11 year period from 1938 to 1949. This investigation does not include those reported by Greene in 1941. Questionnaires were sent to all hospitals registered with the American Medical Association in 1948. There were 2,285 replies (35.7%) from 6,400 hospitals. In this group 28 fires and 41 explosions in operating rooms were reported. The commonest cause was static electricity (32 cases), while suction pressure machines and cautery apparatus were the second and third commonest causes. Since static electricity caused 32 of 41 explosions, these 32 cases are analyzed. The operating room had no conductive flooring in 24 (75%) of the cases. There was no grounding in 17 (59.3%) cases. In the light of recommended safe practices for operating rooms, these are extremely high percentages. Sparks from a mask as it was removed, woolen blankets, woolen trousers, rubber-soled shoes, silk slips, sponge rubber cushion on anesthetist's stool, and nonconductive linoleum flooring caused some of the explosions. Explosions due to static electricity occurred most frequently during the last and first quarters of the year. It is estimated that about eight explosions per year due to static electricity occur in hospitals in the United States. Conductive flooring, grounding, and proper clothing are still not used in many hospitals, and this situation should be corrected.

Bulletin New York Academy of Medicine, New York

28:197-276 (April) 1952

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Treatment of Bacterial Infections of Heart and Pericardium. T. H. Hunter.—p. 213.
Observations on Rehabilitation of the Neurologically Handicapped. E. B. Schlesinger.—p. 229.
Group Therapy with Epileptic Children and Their Mothers. Z. de Fries and S. Browder.—p. 235.
Medical Management of Acute and Chronic Arterial Occlusion. A. W. Duryee.—p. 241.
*Surgical Therapy of Acute and Chronic Arterial Occlusion. J. W. Lord Jr.—p. 259.

Surgical Therapy of Arterial Occlusion.—The four cases of acute arterial occlusion may be classified as embolic, thrombotic, traumatic, and spasmodic. In the embolic cases, an embolectomy is generally indicated in patients under 50, if they are seen within 10 hours of onset, if the artery obstructed is the aorta, iliac or femoral artery, and if the general condition of the patient is such that an operation is tolerable. Embolectomy is generally contraindicated for emboli of the upper extremity and of the lower extremity from the popliteal artery down. The use of heparin broadens the indications for embolectomy as far as the age and time factors are concerned. The ideal management of the patient with an acute thrombotic occlusion of a main artery is still conservative and nonoperative. For acute arterial occlusion due to trauma, the best period for repair is between two and twelve hours after the accident. Methods available for repair are direct suture, autogenous vein graft, or homologous arterial graft. The spasmodic type of acute arterial occlusion, seen most frequently with massive venous thrombosis of the lower extremity, is handled best by Veal's method of vigorous passive and active exercise of the leg, with elevation. The surgical therapy of chronic arterial occlusion should be limited to selected cases after failure of nonoperative measures. Only sympathectomy seems to have real therapeutic value in properly selected cases. The surgical procedures discussed by the author are (1) the so-called revascularization of the leg by production of a three-limbed arteriovenous fistula; (2) the operation of "scraping" the thrombosed artery; and (3) the resection of a relatively short segment of the thrombosed artery, followed by insertion of an autogenous vein graft to restore blood flow through the main arterial channel. Finally the author describes a new surgical treatment of occlusion of the subclavian or axillary artery that is due to disorders of the shoulder girdle. The operative procedure consists of total subperiosteal resection of the clavicle followed by removal of the periosteum, division of the scalenus anticus muscle, and mobilization of the subclavian artery. Good early results were obtained with this method in eight patients with shoulder girdle syndromes of hyperabduction, scalenus anticus, and costoclavicular, or cervical rib syndromes that had resulted in temporary occlusion or permanent thrombosis of the artery.

California Medicine, San Francisco

76:255-314 (April) 1952

Surgical Considerations in Diseases of Pancreas. J. A. Morgan, J. D. Briggs and G. A. Stevens.—p. 255.
The Don'ts of Spinal Anesthesia. B. M. Anderson.—p. 261.
Oxygen Therapy. J. B. Dillon.—p. 263.
Less Commonly Recognized Clinical Features of Amebiasis. M. J. Goldman.—p. 266.
Planned Resection of Regional Lymph Nodes in Pneumonectomy for Carcinoma. J. A. Weinberg.—p. 270.
Pulmonary Resection in Tuberculosis. D. J. Dugan.—p. 273.
Hirschsprung's Disease and Congenital Megacolon. E. E. Leon and R. G. Karshner.—p. 276.
Incidence of Splenic Metastasis of Carcinoma. H. L. Abrams.—p. 281.
Trichophyton Tonsurans (Crateriforme) Infection of Scalp. H. Price and D. R. Taylor.—p. 283.
Carcinoma of Ampulla of Vater: Whipple Operation and 10-Year Arrest in Three Cases. L. C. Bennett.—p. 289.
*Culdoscopy. S. T. Lee.—p. 294.

Culdoscopy.—The culdoscope is an instrument for visualization of the female pelvis through the cul-de-sac. It was designed by Decker and Cherry, when attempts to visualize the pelvis through the peritoneoscope were unsatisfactory because of intervening bowel and difficulty in isolating the pelvic organs. A special trocar and cannula are necessary to perforate the cul-de-sac for introduction of the culdoscope. The importance of performing the procedure in the knee-chest position to utilize the negative pressure produced to draw air into the abdomen and thus push the bowel out of the pelvis has been repeatedly stressed by Decker. On the basis of his own experience with culdoscopy in 45 cases, Lee concludes that cul-de-sac puncture for introduction of a culdoscope is easily made with the patient in the knee-chest position. The pelvic organs can be viewed clearly. In all cases in which laparotomy was done after the examination, the culdoscopic diagnosis was confirmed. In no case in which tubal pregnancy was present was the diagnosis missed in culdoscopic examination. Patients were only slightly uncomfortable after the examination. No pelvic peritonitis resulted in any patient, and no pregnant patient aborted as a result of the procedure.

Delaware State Medical Journal, Wilmington

24:59-82 (March) 1952

Diagnosis and Treatment of Coronary Artery Disease by the General Practitioner. A. H. Clagett Jr.—p. 59.
Role of the General Practitioner in Diagnosis, Treatment of Fractures of Hip. J. H. Smith II.—p. 63.
Delayed Splenic Rupture Following Trauma. R. Kerdasha.—p. 64.
Kidney Changes in Gout. H. W. Keschner.—p. 67.
Rheumatoid Spondylitis: Report of 30 Cases. R. M. Myerson.—p. 69.
Clinical Electroencephalography. S. G. Rogg.—p. 74.

Proc. Soc. Exper. Biol. & Med., Utica, N. Y.

79:339-554 (March) 1952. ***Partial Index***

Aureomycin, Chloromycetin and Terramycin in Treatment of Acute Viral Hepatitis. J. W. Colbert Jr., L. Bungards and M. Knowlton. —p. 339.
Sarcomas Induced in Rodents by Imbedding Various Plastic Films. B. S. Oppenheimer, E. T. Oppenheimer and A. P. Stout.—p. 366.
Virus (Herpes Simplex, Vaccinia) Studies in Embryonated Eggs with Radioactive Phosphorus. H. Blank, B. Kaneda and O. Liu.—p. 404.
Poliomyelitis Virus in Blood of Orally Infected Monkeys and Chimpanzees. D. M. Horstmann.—p. 417.
Technic for Determination of Adrenocorticotrophin in Blood. K. L. Sydnor and G. Sayers.—p. 432.
*Pulmonary Infarction from Cardiac Catheterization. H. E. J. Houssay, F. W. Haynes and L. Dexter.—p. 444.
*Virulence Enhancing Activities of Aureomycin on Candida Albicans. E. Seligmann.—p. 481.
Protective Role of Pyridoxin Against Toxic Effects of P^{32}. C. Artom, W. E. Cornatzer and G. T. Harrell Jr.—p. 494.
Effect of Antibiotics on Development of Resistance to Hemolytic Streptococcus Infection. R. S. Speck and E. Jawetz.—p. 510.
Effects of Fluoride on Developing Bones in Tissue Culture. G. H. Paff and M. J. Boyd.—p. 518.
Coccidioides Immitis Spherule Antigen in Complement Fixation Test for Experimental Coccidioidomycosis. R. A. Vogel and N. F. Conant. —p. 544.

Pulmonary Infarction Following Cardiac Catheterization.—The major complications of cardiac catheterization are cardiac arrhythmias, which develop largely as a result of introduction of the catheter into the right auricle and ventricle, and thrombosis at the venotomy site that sometimes propagates centrally. To measure the pulmonary "capillary" pressure, Houssay and associates introduced the catheter by the usual fashion into the pulmonary artery and then advanced it further until it was wedged into one of the distal branches of this vessel. In the first 100 cases in which this procedure was employed, no untoward reactions occurred, but in March, 1950, the first of a series of pulmonary infarctions occurred in a patient with mitral stenosis and congestive heart failure. At first, these infarctions occurred only sporadically, but later they occurred with increasing frequency. Between March and November of 1950, only four such infarctions occurred; between November, 1950, and May, 1951, pulmonary infarcts were produced in 15 of 27 patients with mitral stenosis. In no instance did death occur, but many of the patients had a stormy course for several days. The pulmonary infarction occurred from 2½ to 79 hours following wedging of a cardiac catheter into a branch of the pulmonary artery. In other patients without pulmonary congestion, pulmonary infarction did not occur. When the catheters were sterilized by autoclaving instead of by soaking in mercuric oxycyanide, pulmonary infarction did not occur in 20 consecutive patients with either severe mitral stenosis or congestive heart failure. Thus there seems to be no doubt that chemical sterilization of the catheters was responsible for the infarction.

Enhanced Virulence of Candida Albicans by Aureomycin.—The author describes studies of the mechanism of the stimulation of growth and increase in pathogenicity of fungi sometimes observed in patients treated with antibiotics. The strain of Candida employed proved to be nonpathogenic for mice on intraperitoneal injection of large amounts of cells (320 million) in saline. The aureomycin dose of 2 mg. intraperitoneally was also nontoxic. However, when Candida cells were suspended in aureomycin solution (0.1 ml. of cells in 2.4 ml. of aureomycin) and the mixture injected intraperitoneally, 16 of 17 mice died in one experiment and 28 of the 32 died in another. A similar though less pronounced "drug-fungus" effect was observed when aureomycin was given from 24 hours before to 4 hours after Candida inoculation. Aureomycin given 8 to 24 hours after infection was ineffective. The virulence-enhancing activity of aureomycin was destroyed by standing at room temperature or by boiling. No indication of toxin production or biological alteration of Candida was found in vitro. No direct growth-stimulating effect by aureomycin on Candida was observed in cultures. The action in vivo was one of lowering the animal's resistance.

Psychiatric Quarterly, Utica, N. Y.

26:1-176 (Jan.) 1952. **Partial Index**

Interaction of Physiological and Psychological Processes in Adaptation. R. A. Cleghorn.—p. 1.
Free Association, Silence and Multiple Function of Speech. J. A. M. Meerloo.—p. 21.
*Mental Changes Experimentally Produced by L. S. D. (*d-Lysergic Acid* Diethylamide Tartrate). H. J. DeShon, M. Rinkel and H. C. Solomon. —p. 33.
Prefrontal Leukotomy: Clinical Survey of 100 Cases Given Active Retraining Program in a Mental Hospital. M. Klotz, W. P. Ritchie and B. C. Schiele.—p. 54.
Therapeutic Relaxation: Follow-Up Results in Casualties of War. E. Schmidhofer.—p. 73.
Somatic Organization of the Schizophrenic. S. T. Michael.—p. 78.

Production of Mental Changes by *d*-Lysergic Acid Diethylamide Tartrate.—The chemical *d*-lysergic acid diethylamide tartrate (L. S. D.) belongs to the ergonovine group of ergot alkaloids. This paper describes the mental changes produced by *d*-lysergic acid diethylamide tartrate in 17 experiments on 15 volunteers. Besides such subjective symptoms as change in appetite, poor co-ordination, and nausea, changes were observed in thinking and speech, emotion, mood, affect, sensory and time perception, and behavior; there were also morbid ideas and sensory experiences and neurological signs. The reaction occurred in three phases lasting a total of from 12 to 16 hours; in some subjects a fourth phase, lasting one to several days, was seen. The reaction showed some aspects of an exogenous toxic picture. Even more impressive, however, was the simulation of schizophrenic manifestations. Simple schizophrenic and turmoil states were common. Transient paranoid trends were frequent. Schizophrenic symptoms such as negativism, ambivalence, blocking, and unexplained smiling were observed. Schizoaffective and manic-like states were also encountered. Catharsis was experienced by only one subject. The reaction was not specific, and the extent to which it was dependent on the basic personality of the subject was not determined. The clinical effects imply involvement of the highest centers of the central nervous system and perhaps of lower levels of the nervous system as well. Further study of this drug is indicated because of the remarkable simulation of schizophrenic and affective psychoses on oral administration of a very small amount of this substance.

South Carolina Medical Assn. Journal, Florence

48:57-82 (March) 1952

Tetanus Neonatorum—Report of Case Treated with Mephenesin. R. J Kahaly and J. R. Harvin.—p. 57.
Dissecting Aneurysm of Aorta: Report of Case. R. S. Pollitzer.—p. 59.
Carcinoma of Cervix Uteri. L. L. Hester Jr.—p. 61.

48:83-116 (April) 1952

Emotional Aspects of Pregnancy. K. G. Lawrence.—p. 83.
Basic Facts Concerning Suicide. O. R. Yost.—p. 86.
The Physician and the Atomic Bomb. V. P. Bond, M. C. Fishler and W. H. Sullivan.—p. 93.

Southwestern Medicine, El Paso, Texas

33:117-156 (April) 1952

Role of Dental Pathology in Head and Neck Complaints. M. P. Spearman.—p. 134.
*The "Best" Method of Prostatectomy. H. M. Gibson Jr.—p. 138.

The "Best" Method of Prostatectomy.—Four surgical approaches to the prostate, the suprapubic, transurethral, perineal and retropubic, are described. The disadvantages, results, and special indications for each method are considered. It is apparent that there is no "best" method of prostatectomy. In general, any one of the methods properly carried out will give satisfactory results in most cases. The method selected by the individual surgeon will depend on his training, inclinations, equipment, and hospital facilities. However, in certain cases, the favorite operation of a surgeon will not give the best results; when this happens, he must be able to adapt the one best suited for the patient. It is no longer sufficient for the urological surgeon or the general surgeon who does prostatectomies to know and perform only one method. In order to give the maximum benefit to the patient, he must learn all four methods.

Surgery, St. Louis

31:487-646 (April) 1952

Aortography in Upper Gastrointestinal Hemorrhage: Experimental Study. J. A. Dolphin and B. Williams.—p. 487.
Treatment of Experimental Acute Pancreatitis with Banthine. W. W. Shingleton, W. G. Anlyan and A. K. David Jr.—p. 490.
Serum Amylase Levels in Experimental Pancreatitis. J. R. Thistlethwaite and R. P. Hill.—p. 495.
Renogastroepiploic Anastomosis in Treatment of Portal Hypertension. R. de Britto, O. Vaz, P. Abdalla and R. da Silva.—p. 502.
Experimental Study of Protein and Fat Assimilation After Total Gastrectomy. T. C. Everson.—p. 511.
Effects of Partial Gastrectomy on Canine External Pancreatic Secretion. D. Annis and G. A. Hallenbeck.—p. 517.
*Suppurative Pelvic Thrombophlebitis: IV. Results. Study of 70 Patients Treated by Ligation of Inferior Vena Cava and Ovarian Vessels. C. G. Collins, R. O. Norton, E. W. Nelson and others.—p. 528.
Intussusception in Adults: Report of 15 Cases. C. H. Brown and A. G. Michels.—p. 538.
Conservative Treatment of Acute Form of Sigmoid Volvulus. N. H. Isaacson and D. B. Greer.—p. 544.
*Carcinoma of Thyroid and Nontoxic Nodular Goiter. E. J. Cerise, S. Randall and A. Ochsner.—p. 552.
Suggested Terminology for Ligaments of Groin; Their Clinical and Surgical Application in Repair of Hernias. C. C. Burton.—p. 562.
Squamous-Cell Carcinoma of Floor of the Mouth. W. L. Mattick, D. J. Meehan and J. P. Haberlin.—p. 575.

Ligation of Inferior Vena Cava for Pelvic Thrombophlebitis.—Follow-up studies are reported on 70 patients who had undergone ligation of the inferior vena cava and ovarian vessels for suppurative pelvic thrombophlebitis between Feb. 2, 1942, and January, 1950. Ligation was performed only if the patients had failed to respond to medical therapy or showed evidence of pulmonary infarction and had severe pelvic infection. Sixty-two of the 70 patients survived. All patients who died within one year of operation were considered operative failures, because the authors feel that ligation accomplishes nothing if the patient dies of distant septic metastasis, even though the vena cava and ovarian vessels are successfully ligated. Four of the eight patients who died were operated on from 9 to 21 days following admission to the hospital, and the authors feel that in these death did not result from delay in operating. Varicosities of the abdominal wall, flank, thigh, or legs did not develop in any of the patients. One woman had varicosities of the legs prior to operation, but they were not made worse by ligation of the inferior vena cava and ovarian vessels. Forty-eight of the patients had no edema. Eight patients noticed occasional mild swelling of the feet after they had been up and about for many hours. Six patients noted some swelling of the ankle and lower half of the leg to about the calf except when at complete bed rest. None of these patients were incapacitated. The vascular status of some of the patients was evaluated, and provided evidence that there was adequate compensation for the ligation of the vena cava. Subsequent surgical procedures required by some of the patients disclosed that the operative hazard was not increased. Menstrual or sexual function was not altered by ligation of the inferior vena cava and ovarian vessels. The authors conclude that ligation of the inferior vena cava and ovarian vessels is indicated in patients with suppurative pelvic thrombophlebitis who fail to respond to medical therapy. Ligation can be done at the same time as other surgical interventions.

Carcinoma of Thyroid and Nontoxic Nodular Goiter.—At the Ochsner Clinic 51 proved cases of carcinoma of the thyroid were compared with 529 cases of nontoxic nodular goiter. The incidence of carcinoma discovered at operation was 19.8% in glands with single nodules, and 12.8% in glands with multiple nodules. In the cases of carcinoma, the average interval between discovery of the tumor and institution of definitive treatment was 5.7 years and between the discovery of the tumor and the time the thyroid was first examined by a physician was 2.8 years. The diagnosis of carcinoma was not made clinically in 63.4% of the cases and was not made at operation in 39% of the cases. In the nine cases of "lateral aberrant thyroid" in which the homolateral lobe of the thyroid was removed, the primary carcinoma was found in the lobe in each instance. The admittedly short term follow-up revealed that about 60% of the patients in the present series are apparently well, and about 17% are dead as a result of the neoplasm. The surgical treatment for carcinoma of the thyroid at the Ochsner Clinic is individualized, but generally hemithyroidectomy with removal of the isthmus is performed on patients whose tumor is limited to the thyroid. If the lesion has spread to the cervical nodes, or locally to muscles, or if there is evidence of local recurrence, radical neck dissection is performed. Patients with massive involvement are best treated by removal of as much tissue as possible, decompression of the trachea, tracheotomy, and postoperative roentgen therapy. The authors stress the following points: 1. Carcinoma of the thyroid cannot be diagnosed early by present clinical means. 2. The incidence of carcinoma in both single and multiple nodular goiters is sufficiently high to warrant prophylactic thyroidectomy for all nodular goiters unless a definite surgical contraindication exists. 3. "Lateral aberrant thyroids" are in reality metastases in cervical lymph nodes and should be so treated.

Western J. Surg., Obst. & Gynecology, Portland, Ore.

60:49-92 (Feb.) 1952

Postmenopausal Vaginal Bleeding. E. A. Schumann and R. W. Hyatt. —p. 49.
Modified Vein Stripper. D. K. Crystal and V. K. Hillman.—p. 52.
Bacteriology of Urine in Late Pregnancy and at Delivery in Relation to Puerperal Urinary Tract Infection. R. J. Alesbury.—p. 54.
Injuries to Hand from Broken Porcelain Faucet Handles. J. H. Boyes and F. C. Melone.—p. 59.
Sidetracking Operation for Extensive Strictures of Common Duct. H. M. Blegen.—p. 65.
Urethral Diverticulum in the Female. C. P. Gray.—p. 68.
Consideration of Wider Removal of Lymphatics with Combined Abdominoperineal Resection of Rectum. J. M. Beal.—p. 70.
Improved Cervical Cap and Sperm Chamber for Artificial Insemination. E. M. Greenberg.—p. 72.
Indications for Radical Neck Dissection. E. B. Speir.—p. 74.

FOREIGN

Acta Tuberculosea Belgica, Brussels

43:1-76 (Feb.) 1952. **Partial Index**

Early Diagnosis of Coxalgia. E. Delcroix.—p. 5.
Results of Streptomycin Treatment of Osseous Tuberculosis. F. Parisel. —p. 14.
Treatment of Primary Tuberculosis Infection with Para-aminosalicylic Acid. R. de Beule.—p. 23.
*Effect of Streptomycin on Tuberculous Lesions of the Lung. P. Golard. —p. 29.

Effect of Streptomycin on Pulmonary Tuberculous Lesions.—Limitation of pulmonary lesions by fibrous walling-off is a histological reaction characteristic of primary infection. Lung specimens from persons with a severe primary infection treated with streptomycin show a somewhat more pronounced tendency toward walling-off, and the caseous foci are not so pronounced as in untreated patients. These changes are the morphological expression of the body's defensive reaction against infection facilitated by the bacteriostatic effect of the antibiotic. Streptomycin seldom arrests a serious primary infection completely. The best indication for streptomycin treatment occurs in recent miliary infection; signs of anatomical cure have been found in patients who died of other causes after having been treated for miliary tuberculosis. Most of the tubercles show hyperplasia of the lymphocytic elements; structural changes also occur in the central epithelioid cells, forming central lacunae that enlarge as a result of giant cell degeneration. Centripetal lymphocytic proliferation gradually fills the lacunae, making the tubercles predominantly lymphoid. The lymphoid tubercle undergoes hyalinization; the cells accompanying it are resorbed, and only nonspecific fibrous formations are left in the place of the initial lesion. Streptomycin accelerates the natural healing process in chronic miliary lesions, reducing the seriousness of the disease and making it susceptible to other methods of treatment. Streptomycin action on bronchial tuberculous lesions is more rapid and intense than on those in the lungs, and its effects vary with the localization of the lesion in the mucosa or the deeper layers of the tissues. Exudative phenomena are favorably influenced; perifocal reactions decrease and limitation of the lesions is accentuated. Streptomycin, however, has little or no effect on caseous nodules. Parietal cleansing, resorption of exudate, and evacuation of casein are the principal advantages of streptomycin in the treatment of cavities; collapse therapy is usually needed to complete the cure. The rapid and frequent appearance of nonspecific cicatrices, rarely seen in the spontaneous development of tuberculosis, is a characteristic effect of streptomycin therapy.

Annals of Rheumatic Diseases, London

11:1-96 (March) 1952

Peri-Arthritis of Shoulder: Studies of Vegetative Function. E. B. Hansen. —p. 2.
Late Results of Gold Treatment in Rheumatoid Arthritis. N. Egelius, N. G. Hävermark and G. Nyström.—p. 17.
Creatine Metabolism in Arthritis. J. E. Dawson and H. B. Salt.—p. 23.
Subfascial Fat Herniation as Cause of Low Back Pain: Differential Diagnosis and Incidence in 302 Cases of Backache. R. Herz.—p. 30.
*Rheumatoid Arthritis of Menopausal Women Treated with Insulin Hypoglycaemias. Z. Z. Godlowski and H. Paterson.—p. 36.
*Familial Incidence of Rheumatoid Arthritis and Acute Rheumatism in 100 Rheumatoid Arthritics. R. W. Barter.—p. 39.
Observations on Use of Thiouracil in Rheumatoid Arthritis. K. A. J. Järvinen.—p. 47.
Heating Knee Joint. H. M. Whyte and S. R. Reader.—p. 54.
Method of Measuring Sweat from Hands: Some Results in Rheumatoid Arthritis. A. Woodmansey.—p. 57.

Insulin Hypoglycemia in Rheumatoid Arthritis.—Several investigators have observed clinical remissions in patients with rheumatoid arthritis after treatment with insulin hypoglycemia. Godlowski and Paterson report studies on 10 women who had had rheumatoid arthritis for from a few months to 18 years. In all of the women the onset was associated with the beginning of menopausal symptoms, and none had had rheumatic fever or rheumatoid arthritis previously. Before, during, and after treatment the body weight, blood picture, and urinary output of 17-ketosteroids, 11-oxycorticoids, creatinine, and uric acid were determined. Insulin hypoglycemia was induced by giving the fasting patient at 8 a. m. a subcutaneous injection of insulin. The doses varied between 20 and 80 units, but were sufficient to produce the clinical symptoms of hypoglycemia. The hypoglycemic attack was interrupted by means of a glassful of sugared water and a meal rich in carbohydrates. The insulin hypoglycemia was repeated daily for from 14 to 28 days. Nine of the 10 patients showed significant improvement during and for some time after treatment, but all relapsed during the succeeding 10 months, and in some the disorder progressed more rapidly than before treatment. Thus insulin hypoglycemia only temporarily retards the course of this type of rheumatoid arthritis.

Familial Incidence of Rheumatoid Arthritis.—The familial incidence of rheumatoid arthritis and acute rheumatism was compared in 100 patients with rheumatoid arthritis and 100 controls. The incidence of these diseases was 5.3% in the families of patients and 2.3% in the families of controls when grandparents, parents, uncles, aunts, brothers, and sisters were included. The incidences were 11% and 4.6% respectively in the two groups when only parents, brothers, and sisters were included. From these figures it was concluded that approximately one-third of patients with rheumatoid arthritis will have a family history of rheumatoid arthritis or acute rheumatism in two or more members (as many as 7 affected members were recorded in one family of 16); approximately one-third will give a history of one affected member; and the remaining one-third will have no relatives with these diseases. It is suggested that there is a tendency toward development of acute rheumatism or rheumatoid arthritis in certain families. This constitutional tendency is discussed in relation to Selye's "general adaptation syndrome" and Kendall's view that a hypersensitivity state may underlie rheumatoid arthritis. As regards the former, certain persons may have a hereditary weakness in their adaptation mechanism; as regards the latter, it is suggested that certain persons may have a constitutional tendency to react to the effects of the products of an antigen-antibody reaction through the medium of the synovial membranes.

British Journal of Ophthalmology, London

36:113-168 (March) 1952. **Partial Index**

Intra-Ocular Acrylic Lenses: Recent Development in Surgery of Cataract. H. Ridley.—p. 113.
Orbital Implants: Review of Results Obtained at the Moorfields Branch of the Moorfields, Westminster and Central Eye Hospital, London. D. P. Choyce.—p. 123.
Phase-Contrast Microscopy of Animal Vitreous Body. B. A. Bembridge, G. N. C. Crawford and A. Pirie.—p. 131.
Nervous Manifestations in Eales's Disease. M. Jefferson and P. C. P. Cloake.—p. 143.

British Journal of Radiology, London

25:169-224 (April) 1952. **Partial Index**

Evaluation of White-Cell Counting in Radiation Protection. A. C. Chamberlain and E. K. Williams.—p. 169.
SYMPOSIUM ON RADIATION GENETICS
Genetic Aspects of Radiation Risks. A. Howard.—p. 177.
Indirect Effects of X Rays on Chromosomes. G. Bonnier.—p. 180.
Cytological and Cytochemical Approach to Understanding of Radiation Damage in Dividing Cells. A. H. Sparrow, M. J. Moses and R. Steele.—p. 182.
*Limit of Renal Tolerance to X-Rays: Investigation into Renal Damage Occurring Following Treatment of Tumours of Testis by Abdominal Baths. P. B. Kunkler, R. F. Farr and R. W. Luxton.—p. 190.
Radiological Investigation of Stomach Changes Following Loud Auditory Stimulus. H. Jungmann and P. Venning.—p. 202.
Intramuscular Pyelography in Children. K. E. Hodge.—p. 209.

Limit of Renal Tolerance to X-Rays.—After orchiectomy has been done for seminoma of the testis, the region of potential metastases includes the aortic iliac and inguinal lymph nodes and the associated lymphatics. In prophylactic treatment of this region x-rays are commonly employed in a dose of from 2,000 to 3,000 r in three to six weeks. If the kidney is included in the field of irradiation, renal damage may occur. This report is based on a study of over 30 cases. Such damage may be unrecognized unless it is specifically looked for. There is usually a latent period of several months before the onset of symptoms, which in the cases here reported averaged eight months. The first complaints were dyspnea on exertion, headache, swelling of the ankles, and lassitude. On examination pallor was noted. Edema was usually present, but varied in extent and severity. The blood pressure was increased in all cases at some time. In some patients the retinas showed hemorrhages, exudates, and papilledema, and the clinical picture then suggested malignant hypertension. Cardiac enlargement, pulmonary edema, and hepatic enlargement due to congestion were all found in the more advanced cases. Severe dyspnea was a troublesome symptom. Anemia usually progressed rapidly. All patients had albuminuria, and in the urine of some granular and hyaline casts were found. Some of the patients died within a few months of the onset of symptoms, death being due to chronic uremia or to left ventricular failure. However, considerable improvement was possible even in severe cases following judicious blood transfusions and prolonged rest. Those who survived the initial more acute symptoms had signs of chronic nephritis, such as albuminuria, hypertension, anemia, increased blood urea levels, and impaired renal function. The various techniques that were employed for the prophylactic roentgen irradiation of these patients had been described previously, but in this paper an account is given of the arrangement of the fields and the basis on which the dosage is calculated. The survival rates in 93 cases and the incidence of renal failure are correlated with the distribution of the dose through the kidneys. In patients in whom renal failure developed, one entire kidney or both kidneys had received a dose of 2,000 r or over in five weeks. The "safer" techniques were characterized by smaller doses to the upper poles of both kidneys. The risk of renal failure may best be minimized by insuring that about one-third of the volume of the kidneys is outside the irradiated fields or is irradiated with as small a dose as possible. Renal function and location should be determined before roentgen treatment of the abdomen is undertaken.

British Journal of Venereal Diseases, London

28:1-48 (March) 1952

Interpretation of Serum Reactions in Late Syphilis. C. P. Heywood. —p. 3.
Note on Patterns of Quantitative Serological Tests in Late Syphilis. A. Redmond, C. S. Nicol and R. A. Shooter.—p. 13.
Comparative Serum Testing with Cardiolipin and Crude Heart Extract Wassermann Antigens. I. N. O. Price and A. E. Wilkinson.—p. 16.
Two Cases of Gummatous Ulceration Treated with Terramycin. R. R. Willcox.—p. 20.
Occurence of Neisseria Other Than Gonococcus in Genital Tract. A. E. Wilkinson.—p. 24.

Canadian Medical Association Journal, Montreal

66:309-418 (April) 1952

Acute Pulmonary Oedema. L. C. Steeves.—p. 309.
*Cortisone in Asthma. H. S. Mitchell and G. Cameron.—p. 313.
Early Ambulation Following Abortion. M. M. Davis.—p. 317.
Further Study of Blood Cholesterol in Schizophrenia. G. Nadeau and G. H. Larue.—p. 320.
Syphilitic Cardiovascular Disease. C. L. Hunt.—p. 324.
Common Pitfalls in Medical Practice. G. A. Copping.—p. 329.
Presence of Q Fever Complement Fixing Antibodies in Sera of Inhabitants of the Province of Quebec. V. Pavilanis, P. Lépine and N. Morisset.—p. 333.
Islet Cell Tumours of Pancreas. A. O. Whipple.—p. 334.
Therapeutic Evaluation of Cortisone Applied Topically in Ophthalmic Diseases. J. F. Minnes.—p. 342.
Observations on Physiological Effects of Cortisone and ACTH. H. S. Robinson, R. A. Palmer, A. W. Bagnall and H. W. McIntosh.—p. 347.
*Further Experiences with Para-Aminosalicylic Acid. H. M. Ross.—p. 353.
Management of Erythroblastosis Fetalis. C. E. Snelling, W. L. Donohue and M. McKee.—p. 356.
Investigation of Histoaminolytic Activity of Serum in Pregnancy. W. H. Allemang.—p. 359.
Tuberculous Empyema Complicating Artificial Pneumothorax. H. J. A. Cranz.—p. 363.
Glucose Metabolism in Anxiety States. L. J. Cera, T. J. Matas and F. D. White.—p. 366.
ACTH in Aspiration of Peanuts. M. K. MacGougan and S. B. Thorson. —p. 371.

Cortisone in Asthma.—Mitchell and Cameron studied 21 patients with intrinsic bronchial asthma who had been given courses of cortisone or cortisone and corticotropin combined. The duration of asthma varied from a few months to 49 years; four patients had had attacks for less than two years. The severity of the asthma varied from case to case, but in most cases it was of the severe intractable type, with decreasing response to bronchodilators. Seventeen of the patients were given cortisone by the intramuscular or oral route; two were given corticotropin for the first two days in conjunction with cortisone therapy; one was given corticotropin after several days of cortisone therapy while the cortisone dosage was gradually decreased, and one was given corticotropin after an initial unsatisfactory trial of cortisone. The authors gained the impression that cortisone has a definite role in treatment of patients with intrinsic bronchial asthma whose emphysema is not severe enough to cause irreversible changes in their lung fields. It may be a lifesaving measure in patients in status asthmaticus who do not respond to bronchodilators; this is demonstrated in the three cases. The response to the drug is more or less unpredictable. Many more cases should be studied before its value in asthma can be assessed. Cortisone was found to be just as effective orally as intramuscularly. Oral doses of 200 mg. for one day, and then 100 mg. daily for several days thereafter were sufficient. Relapse usually occurred from one week to three months after cortisone had been discontinued. Maintenance doses have to be continued. Cortisone should only be tried in patients in whom all other forms of medication have failed. Side-effects were not frequent. One patient has been taking cortisone for 10 months without ill effect.

Experiences with Para-Aminosalicylic Acid.—The combined use of *p*-aminosalicylic acid orally and streptomycin intramuscularly in patients with pulmonary tuberculosis has become standard practice. In this study one group of 221 patients received only 1 gm. of streptomycin daily for from 60 to 90 days. Organisms persisted in 154 of this number, and in 67% the bacilli showed increased resistance. In a second group 58 patients received 1 gm. of streptomycin and 2 gm. of *p*-aminosalicylic acid daily for the same period. Orangisms persisted in 30 patients, and 64% of these after cessation of therapy had organisms with increased resistance to streptomycin. A third group of 146 patients received 1 gm of streptomycin and 10 gm. of *p*-aminosalicylic acid daily for 60 to 90 days. In 72 cases organisms persisted and 18% of these patients had organisms with increased streptomycin resistance. Thus it would seem that a daily dose of 10 gm. of *p*-aminosalicylic acid rather than 2 gm. along with 1 gm. per day of streptomycin caused a marked reduction in the number of streptomycin-resistant strains of tubercle bacilli.

Clinica Pediatrica, Bologna

34:1-70 (Jan.) 1952

Studies on Action of Antireticular Cytotoxic Serum in Immature and Dystrophic Patients. G. Brogi and M. Verrotti.—p. 1.
Electrocardiograms in Acute Phase of Poliomyelitis. L. Salmi and M. Malossi.—p. 17.
Effectiveness of Early Introduction of Structural Compounds with Lipotropic Activity in Newborn with Extensive Skin Manifestations of Exudative Diathesis. M. Orlandelli.—p. 29.
*Spinal Block During Streptomycin Therapy of Tuberculous Meningitis. P. Nicolaj.—p. 39.

Spinal Block in Streptomycin Therapy of Tuberculous Meningitis.—A severe complication that may occur during streptomycin therapy of tuberculous meningitis is spinal block. This may occur when the streptomycin dosage is inadequate or it is given only intramuscularly thus inhibiting but not stopping the process, whose slow development leads to spinal block. It may be attributable to local irritation resulting from excess streptomycin administered intrathecally, predisposition of the patient to this complication, or a general allergic reaction to the antibiotic. In patients who were never treated with the drug spinal block may arise either because the process develops slowly, or because the meningeal exudate concentrates around the first part of the spinal cord, obstructing the circulation of the fluid. The author reports on 12 patients, in three of whom spinal block developed before treatment was instituted. In four it resulted from inadequate streptomycin therapy, in four from local irritation by the antibiotic, and in one as a part of a general allergic reaction to the antibiotic after recovery from meningitis. It was more frequent in adults than in children, and as a rule, symptoms were severer in patients who had not received streptomycin or only inadequate doses of it. The spinal fluid in all patients was xanthochromic and showed excess albumin. Six patients died, including all four in whom streptomycin therapy had been *inadequate. It is suggested that the prognosis is more favorable in patients who are not treated with streptomycin before the onset of the spinal block and in those in whom the block was caused by local irritation from the antibiotic. If therapy, which must be individualized, is adequate, the spinal block may recede completely, as in some of the author's patients, with complete restoration of all the functions that had been impaired.*

Lancet, London

1:679-728 (April 5) 1952

Factors Which Influence Value of Radiological Investigation. J. F. Brailsford.—p. 679.
*Self-Demand Feeding in a Maternity Unit. R. S. Illingworth, D. G. H. Stone, G. H. Jowett and J. F. Scott.—p. 683.
Sickle-Cell Anomaly as Sign of Mediterranean Anaemia. J. Caminopetros. —p. 687.
Hyperthyroidism and Peptic Ulcer. A. M. Gill and F. W. Walton. —p. 693.
Familial Cataract and Mental Deficiency. B. H. Kirman.—p. 694.
Ornithosis: Isolation of Virus from a Case. P. R. Duncan, A. E. Thomas and J. O'H. Tobin.—p. 696.
Estimation of Formaldehydogenic Steroids. A. A. Henly and M. Potter. —p. 697.
Pituitary Tuberculoma. L. C. Oliver.—p. 698.

Self-Demand Feeding in a Maternity Unit.—A flexible feeding schedule favors the development and maintenance of successful lactation. Babies have been shown to demand very frequent feedings from the fifth to the eighth day of life; when they are kept waiting on a rigid schedule they are apt to become tired and to swallow air as a result of crying, making suckling difficult. The relation of the feeding schedule to the establishment of lactation was studied by feeding one group of 106 babies on a rigid schedule and another group of 131 on a self-demand schedule, beginning on the third day, and comparing the two groups in regard to weight gain, incidence of overdistention of the breasts in the mothers, and incidence of breast-feeding on discharge and at the age of one month. All babies were fed at six hour intervals during the first two days for fear that more frequent feeding might cause soreness of the nipples. Then babies in the demand group were fed when they wanted it, day or night; the others were fed every four hours by day and once at night (6 feedings in 24 hours). Demands from babies in the demand group were most frequent between the fourth and seventh days, 36 babies demanding 8 feedings or more on the fifth day, and 12 demanding 9 or more. Babies fed on demand gained weight more rapidly than the others; by the ninth day 49.1% had regained their birth weight as compared with 36.1% of those on the rigid schedule. Test feedings, showing a positive correlation between the weight gain and the amount of milk taken, indicated that babies fed on demand empty the breast better than those on a rigid schedule. Overdistention of the breast and soreness of the nipples was more than twice as frequent among mothers of babies on a rigid schedule as among mothers of babies in the demand group. The number of fully breast-fed babies in the rigid group declined from 88.1% at the time of discharge to 64.5% at the age of 1 month. Comparable figures for the demand group were 94.4% on discharge and 80.3% at 1 month. Self-demand breast-feeding, by causing better emptying of the breast and less frequent soreness of the nipples, favors lactation.

Nederlandsch Tijdschrift v. Geneeskunde, Amsterdam

96:593-660 (March 15) 1952. **Partial Index**

*Decreasing Mortality from Whooping Cough in Nursing Infants. J. E. Minkenhof and M. W. van der Kooi.—p. 598.
Treatment of Severe Uremia by Intestinal Dialysis and Feeding Through a Cardiac Catheter. G. Bossinga and E. E. Twiss.—p. 604.
*Copper in Treatment of Hypochromic Anemia Refractory to Iron. S. I. de Vries.—p. 611.

Decreasing Mortality from Whooping Cough in Nursing Infants.—Minkenhof and van der Kooi gained the impression that during the last 50 years infant mortality from whooping cough has decreased not only as the result of more spontaneous recoveries but also because of more effective treatment of secondary pneumonia. Furthermore chloramphenicol and aureomycin do not influence uncomplicated whooping cough favorably. On the contrary, they found that in some cases these drugs render other bacteria, such as those causing pyoderma, resistant to all chemotherapeutic and antibiotic drugs. However, secondary bronchopneumonia that has proved resistant to sulfonamides, penicillin, and streptomycin responds favorably to these drugs. Investigating the deaths among 249 infants with whooping cough who had not been immunized against this disease, the authors found that while in 1946 there were 22 fatalities among 56 cases, there were none among 40 in 1951. Secondary pneumonia was treated with sulfonamides or dihydrostreptomycin. Aureomycin was used and proved effective in only two resistant cases. Since 1948, infants with whooping cough have been given dihydrostreptomycin in order to prevent pneumonia.

Copper in Treatment of Hypochromic Anemia.—De Vries reviews the literature on the use of copper in the treatment of anemia. On the basis of his own observations on eight patients, of which four are reported in detail, he is convinced that, in patients with hypochromic anemia who are refractory to treatment with iron and in whom the copper content of the serum is low, treatment with copper will have a therapeutic effect. It is assumed that copper has a catalytic effect in these cases.

Practitioner, London

168:313-448 (April) 1952. **Partial Index**

Cardiac Emergencies in General Practice. C. J. Gavey.—p. 317.
Respiratory Emergencies. N. L. Rusby.—p. 325.
Abdominal Emergencies. R. M. Walker.—p. 331.
Paediatric Emergencies. R. W. B. Ellis.—p. 338.
Obstetrical Emergencies. H. H. Evers.—p. 347.
Neurological Emergencies. E. C. O. Jewesbury.—p. 357.
Psychiatric Emergencies in General Practice. E. W. Anderson.—p. 363.

Ugeskrift for Laeger, Copenhagen

114:351-384 (March 20) 1952

*Simmond's Syndrome Treated with Desoxycorticosterone Acetate, Testosterone Propionate and Thyroidin and with ACTH. H. Petersen. —p. 351.
*Vitamin B_{12} Treatment of Megalocytic and Macrocytic Anemia. J. S. Baastrup.—p. 356.

Treatment of Simmond's Syndrome.—Simmond's syndrome must be differentiated from myxedema, Addison's disease, and anorexia nervosa; myxedema is the most frequent mistaken diagnosis. In the first case reported no treatment was attempted because of the unfavorable prognosis. In one of the three patients treated with desoxycorticosterone acetate (percorten®), testosterone propionate (perandren®) and iodothyrin ("thyroidin") the result was particularly good and in another case the results were good from the somatic viewpoint, but the psychic changes were not affected. During this treatment attention must be directed to such symptoms of overdosage as increase in weight, increase in blood pressure, edema, and signs of cardiac insufficiency. In the cases described slight signs of overdosage were seen in the form of crural edema, increased weight, and increased blood pressure. *In the third case in which the combined treatment was used unsuccessfully, treatment with corticotropin (ACTH) had had no effect.* The commonest causal factors in Simmond's syndrome were represented, craniopharyngeoma in one case, grave postpartum hemorrhage in two cases; the etiology in the remaining case is unknown. Petersen's impression is that possibly psychic changes cannot be influenced favorably by treatment.

Vitamin B_{12} Treatment of Megalocytic and Macrocytic Anemia.—Seven cases of pernicious and related anemia were treated by intramuscular injection of vitamin B_{12}. In one case of pernicious anemia, one case resembling pernicious anemia, and two cases of macrocytic anemia complete remission resulted, while in two cases of pernicious anemia and one case resembling pernicious anemia the hemoglobin level and the erythrocyte count rose only to slightly subnormal values. Since injection treatment with vitamin B_{12} is agreeable for the patient and since in some cases complete remission follows and in other cases the treatment at least to begin with is as effective as treatment with liver and stomach preparations, it seems to Baastrup reasonable at present *first to try treatment with vitamin B_{12} in megalocytic and macrocytic anemia; if full restitution is not attained, liver and/or stomach preparations can be given, perhaps with the addition of vitamin B_{12}.*

BOOK REVIEWS

The Metabolic Response to Surgery. By Francis D. Moore, M.D., Moseley Professor of Surgery, Harvard Medical School, Boston, and Margaret R. Ball, A.B. Publication number 132, American Lecture Series, monograph in American Lectures in Surgery. Edited by Michael E. De Bakey, M.D., Professor of Surgery, Chairman of Department of Surgery, Baylor University College of Medicine, Houston, and R. Glen Spurling, M.D., Clinical Professor of Surgery, University of Louisville, Louisville, Ky. Cloth. $7.50. Pp. 156, with 48 metabolic diagrams by Mildred B. Codding, A.B., M.A., Surgical Artist, Department of Surgery, Harvard Medical School, Peter Bent Brigham Hospital, Boston. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto, 2B, 1952.

This book describes studies carried out on metabolic balances in order to determine the biologic responses to surgery. The studies deal with patients undergoing surgery who had been treated by ordinary methods. A uniform method of charting is presented, and, although an attempt has been made to present it simply, it has become cumbersome and confusing as can be noted in figure 31 on page 83. Charting of this type, although accurate and conclusive, must be simplified if it is to be of practical value, particularly to the general practitioner or the general surgeon.

Great detail was gone into in the preparation of this material, and the authors utilized a "team" of 35 co-workers. Careful studies were made on total intake and total output of selected chemical substances in order to cast light on biologic processes. The collection of output presented special problems. When dressings were stained by exudate or gastrointestinal contents, they were either soaked in distilled water or put through a small washing machine with distilled water, and this water was analyzed for nitrogen and electrolytes. Operative blood loss was measured calorimetrically, utilizing acid hematin on sponge rinsings; this was checked by total nitrogen.

This book is not easy to read. The need for profound study detracts from its practicability; however, it is a sincere study and may be of reference value as further studies dealing with these metabolic responses are carried out. The two most appealing sections are those dealing with facts and corollaries and surgical diet and parenteral supplements.

Child Psychiatric Techniques: Diagnostic and Therapeutic Approach to Normal and Abnormal Development through Patterned, Expressive, and Group Behavior. By Lauretta Bender, B.S., M.A., M.D., Professor of Clinical Psychiatry, New York University College of Medicine, New York. Cloth. $8.50. Pp. 335, with 55 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto 2B, 1952.

The author of this volume is well known for her work on the psychology of perception and her "visual motor Gestalt" test, which is used extensively by psychologists. In this book Dr. Bender describes the technique she uses in treating psychiatric conditions of childhood. The work is based on the results obtained in the reexamination of thousands of problem children at the psychiatric division of the Bellevue Hospital. Some of these children were first examined when they were very young and were reexamined during adolescence. Parts of the book were published previously but are now unified. Some of the work was done with other authors, notably the late Paul Schilder.

The book will be of great value to psychiatrists, psychologists, and play therapists, but it will also be of interest to the general physician. Some chapters will be of special value to pediatricians, notably the ones on form as a principle in the play of children, the drawing of a man by children with chronic encephalitis, the art of high grade mental defective children, and group activities on a children's ward. Although the book is technical, the author's style is simple. The illustrations, many of which are freehand, add to the value of the book.

American Universities and Colleges. Mary Irwin, Editor, American Council on Education. Sixth edition. Cloth. $10. Pp. 1105. American Council on Education, 1785 Massachusetts Ave., N.W., Washington 6, D. C., 1952.

American Junior Colleges. Edited by Jesse P. Bogue, Executive Secretary, American Association of Junior Colleges. Third edition. Cloth. $7.50. Pp. 604, American Council on Education, 1785 Massachusetts Ave., N.W., Washington 6, D. C., 1952.

"American Universities and Colleges" contains full descriptive data on 904 accredited universities and colleges in the United States, Alaska, Hawaii, and Puerto Rico. The new feature in this edition is the arrangement of institutions by states, with an institutional index instead of the former alphabetical listing by name only.

Part 1 of the book is devoted to professional education. Discussion on medical education by Dr. D. G. Anderson, Secretary of the American Medical Association Council on Medical Education and Hospitals, is concluded by a list of accredited medical schools. Dentistry, pharmacy, nursing, and veterinary medicine are adequately discussed.

The companion book, "American Junior Colleges," contains descriptions of 575 accredited junior colleges in the United States. This list is also arranged by states, with data on R. O. T. C. units and other features. These two publications are indispensable reference tools in any library, college, or office concerned with higher education.

The United States Public Health Service 1789-1950. By Ralph Chester Williams, M.D., Assistant Surgeon General, United States Public Health Service, Washington, D. C. Cloth. $7.50. Pp. 890, with illustrations. Commissioned Officers Association of United States Public Health Service, Box 5874, Bethesda, Md., 1951.

This excellent history of the United States Public Health Service is well written, carefully documented, and impressive in its scope. The author is a regular career officer of the service, with broad experience in many different departments of the service. The presentation is effective, the typography good, and the photographic material of unusual historical interest.

The dramatic role of the Public Health Service in protecting the American people from communicable disease introduced from other countries is particularly well told. The innumerable other health activities, curative, educational and preventive, are also described at length in an interesting and stimulating fashion. The United States Public Health Service emerges from this history as a noble career service, dedicated to preserving the health of the people within the framework of the policies adopted by Congress and singularly free from the political storms that have afflicted many other federal agencies. If the author fails to note and record the occasional policy errors and internal dissensions that must have occurred some time in the long history of the United States Public Health Service, he may be forgiven, for the total story is true, an exciting, fruitful history of physicians devoted to public service in the finest tradition of medicine.

Atlas of Genito-Urinary Surgery. By Philip R. Roen, M.D., F.A.C.S., Instructor in Urology, New York Post-Graduate Medical School, New York. Introduction by Clarence G. Bandler. Cloth. $8. Pp. 325, with illustrations by Charles Stern. Appleton-Century-Crofts, Inc., 35 W. 32nd St., New York 1, 1951.

As indicated by the title, the author has compiled, with the aid of an excellent artist, a series of line drawings illustrating most of the common operations performed on the genitourinary system. The text, which describes each operation, is brief and direct, often being less than half a page in length, and is placed on the page opposite the illustration to which it refers. There is an illustration on every other page of the book. The book was planned for the resident, the young urologist, and the general surgeon who is occasionally required to perform a urologic operation. Only standard surgical procedures are discussed, although, in some cases, multiple procedures are illustrated for a given type of operation. Details of surgical technique are not

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The reviews here published have been prepared by competent authorities and do not represent the opinions of any official bodies unless specifically stated.

included, but the principles and successive steps of a given operation are outlined.

The book is divided into nine sections dealing respectively with general principles of urologic surgery, operations on the kidney, adrenal gland, ureter, bladder, prostate, scrotum and testicles, and urethra and penis. An index is included. The author draws from his experience in learning, teaching, and practicing urology for his discussion of the essentials of standard urologic operations that he has found useful and practical. The material is discussed in an easily understandable form, with clear illustrations and a minimum amount of text. The book is neither a textbook for the medical student nor a reference book, as no bibliographic references are included, and descriptions of many less common procedures are omitted, but it should be a useful manual for those not too familiar with urologic surgery. Although not everyone would agree with certain practices suggested by the author, such as the use of oxidized cellulose gauze imbedded in the renal parenchyma and the use of carbolic acid in surgical treatment of solitary renal cyst, his opinions, in general, coincide with current urologic practice. Indications for and relative evaluation of various surgical procedures are not included. The book is beautifully and completely illustrated and provides a quickly available source of information pertaining to urologic surgery.

Symposium über die Beeinflussung des reaktiven Geschehens durch Hypophyse und Nebennierenrinde. Veranstaltet von der Schweizerischen Akademie der medizinischen Wissenschaften 30. September bis 2. Oktober 1951 in Zürich. [In German, English and French.] Sonderausgabe von Fasc. 1/2, Vol. 8 des Bulletin der schweizerischen Akademie der medizinischen Wissenschaften. Paper. 16 Swiss francs. Pp. 228, with 75 illustrations. Benno Schwabe & Co., Klosterberg 27, Basel 10; imported by Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1952.

This monograph consists of a series of 20 papers on the effects of cortisone and corticotropin (ACTH); they were read at a meeting of the Swiss Academy of Medicine at Zürich in September, 1951. The first 12 papers deal with tissue reactions, the next 2 with hemorrhagic states, and the last 7 with allergic reactions and their influence. They are written in English or German, depending on the nationality of the author, and there are brief discussions by participants at the symposium at the end of each paper. Almost all the material is based on the authors' own work, which by now has been published through the usual channels. Those interested in the experimental field will find this collection valuable, particularly the criticisms and comments of the discussants. Like similar collections that have appeared elsewhere, the work throws light on the fundamental mechanism of action of corticotropin and cortisone. The papers deal with such diverse topics as the effect of cortisone on trichophytosis in guinea pigs (Sonck and Miescher), the relation of the endocrine system to tissue reactions in tuberculosis (Rössler), the effect of cortisone on the tuberculin reaction (Ballabro and Sala), the Arthus phenomenon (Uehlinger), its site of action (Studer), and its relation to anaphylaxis (Dougherty). Of more clinical interest are papers dealing with corticotropin and cortisone therapy of asthma (Rose), ulcerative colitis (Weissbecker), and eczema (Miescher).

Today's Science and You. By Lynn Poole, Producer of Johns Hopkins TV Science Review. Cloth. $2.75. Pp. 208, with illustrations by Jeanne Bendick. McGraw-Hill Book Company, Inc., 330 W. 42nd St., New York 18; Aldwych House, Aldwych, London, W.C.2, 1952.

Many of the discussions in this small volume seem to offer little in the way of helpful education. This may be because the discussion does not relate the various scientific advances closely enough to what the average man knows. For example, it will not profit a man much to know that there is a device capable of seeing 100,000 colors, that the perfect fly repellent has yet to be developed, or that different shaped knobs on the automobile panel can simplify car operation. These and other scientific findings reported are interesting but open to so many arguments that their value may be questioned. Nevertheless, there is a considerable amount of interesting material. Anyone who has watched the Johns Hopkins Science Review on television will agree that many extremely stimulating programs have been presented. Perhaps the lesson from this is that science teaching can be effective only when it is given in the real-life pattern for which television is unique.

The Diagnosis and Treatment of Intrathoracic New Growths. By Maurice Davidson, M.A., D.M., B.Ch., Consulting Physician to Brompton Hospital for Consumption and Diseases of Chest, London. With chapter on Radiotherapy by David W. Smithers, M.D., M.R.C.P., D.M.R., Professor of Radiotherapy, University of London, and chapter on Operative Treatment by Oswald S. Tubbs, M.A., M.B., B.Chir., Thoracic Surgeon to St. Bartholomew's Hospital, London. Cloth. $8.75. Pp. 260, with 170 illustrations. Oxford University Press, 114 Fifth Ave., New York 11; Amen House, Warwick Sq., London, E.C.4, 1951.

As a rule, when a senior British physician produces a volume on a clinical subject, one can expect a clear, readable, and balanced presentation. This monograph is no exception. The author brings his extensive clinical experience to bear on a topic that is of immense practical importance today. The point of view is that of the general physician who, encountering tumors of the chest in his practice, is seeking reliable information in regard to the present methods of treating them. The material is discussed according to pathological entity. Each tumor or class of tumors is discussed according to its morbid anatomy, clinical diagnosis, and treatment. This is not, however, merely a handbook of differential diagnosis or a manual of diagnostic tests but a careful description of pathological processes supported by well-documented and pertinent clinical examples.

A large portion of the book is devoted to carcinoma of the bronchus. The discussion is that of the intelligent physician's point of view toward surgery of the lung. Surgical treatment is measured against radiation therapy and chemotherapy. The latter are discussed at length together with excellent practical advice for the treatment of the "untreatable" patient.

The chapter on mesoblastic growths provides an account of pleural endothelioma and related tumors. The confusing aspects of this problem are well illustrated by specific examples. Separate chapters on the techniques of radiation therapy, by Smithers, and standard thoracic surgical procedures, by Tubbs, complete the volume.

This book can be highly recommended as an intelligent correlation of fundamental pathology and wide clinical experience. It should be of great service both to the general practitioner and his expert consultant. It should provide excellent background material for medical students and residents and should find a place in any well-equipped hospital library. The text is delightfully written, and the illustrations are superb.

Fractures and Joint Injuries. Volume I. By Sir Reginald Watson-Jones, B.Sc., M.Ch.Orth., F.R.C.S., Orthopædic Surgeon to His Majesty the King, London. Fourth edition. Cloth. $22 per set. Pp. 443, with 709 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1952.

In every era of medicine, certain select treatises have profoundly influenced medical education and practice. Sir William Osler's "Textbook of Medicine" is an outstanding example. In the field of traumatic surgery, Sir Reginald Watson-Jones' "Fractures and Joint Injuries," the first volume of the fourth edition of which has recently been published, is another.

The first edition of this important textbook was published in 1940, at the beginning of the great conflict that was shortly to engulf mankind. It described clearly and concisely the principles underlying the treatment of fractures and joint injuries, and it can be said that, perhaps more than any other surgical text of our time, it became the "bible" of surgeons in every land, confronted as they were then by a host of complicated orthopedic injuries. It raised the standards of treatment incalculably, and untold thousands owe life, limb, and happiness to its author. Since 1940, successive editions have been published, each in its turn revised and rewritten to incorporate the experience gained by Sir Reginald in his unique vantage point as civilian consultant in orthopedic surgery to the Royal Air Force and more recently as director of the orthopedic and accident service at the London Hospital.

The fourth edition has been carefully revised to include recent advances in the treatment of fractures and joint injuries, and many new illustrations have been added. The physiology and pathology of bone repair are carefully and wisely discussed, and the all-pervading principles of treatment stressed. In chapter 6, there is a valuable discussion of vascular injuries, immersion foot, frostbite, and the crush syndrome. In the discussion of nerve injuries in chapter 7, the Seddon and Cohen classification of nerve injuries is retained, but perhaps a more venturesome policy of earlier nerve exploration and repair would seem justified with the present advantages of chemotherapeutic control of infection. The section on clinical and radiographic diagnosis is of great value and should be read and reread by all practicing surgeons.

A wise and balanced conservatism governs the author's advice on the operative reduction of certain fractures, and, in the present volume, the great value of intramedullary nailing of certain fractures, notably of the femur, is commented on. The chapter on reactions of bone to metal is a most welcome addition to the present edition. It stresses that the use of plates, screws, and nails are merely methods of internal suture, which, in fact, introduce foreign bodies not unlike sequestra. This is an important observation that may well temper our present and perhaps premature advocacy of prosthesis in the treatment of avascular necrosis of the head of the femur and osteoarthritis of the hip joint.

A new and valuable addition is the chapter on shock, stress, and the adaptation syndrome, the invariable complications of all major injuries. The discussion and advice tendered is sound and practical. The section on open fractures and joint injuries has been greatly enhanced in value by important sections on the chemotherapeutic control of infection, the early closure of wounds by secondary suture or skin grafting, and the role of plastic surgery in fracture treatment. Few procedures are more important, and, until recently, more neglected. The last chapters of the book deal with fatigue and stress fractures, birth fractures, and pathological fractures. They are of immense value, and perhaps nowhere else has so much recondite information been so clearly assembled.

The plates and illustrations are excellent throughout, demonstrating clearly the author's discussion. The style is clear, straightforward, and to the point. The type is easily read and the paper used of excellent quality. In addition, the book is of manageable proportions. This book is a fine achievement, worthy of its distinguished author and of British surgery.

Lehrbuch der Röntgendiagnostik. 5. und 6. Lieferungen: Innere Organe. Von H. R. Schinz, W. E. Baensch, E. Friedl, und E. Uehlinger. Nebst Beiträgen von E. Brandenberger et al. Fifth edition. Paper. 84 marks; 98 marks. Pp. 1921-2356, with 522 illustrations; 2357-2884, with 507 illustrations. Georg Thieme, Diemershaldenstrasse 47, (14a) Stuttgart O; agents for U. S. A., Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1951; 1952.

For the review of sections 1 to 4 (*J. A. M. A.* 144:885 [Nov. 4] 1950; 146:1542 [Aug. 18] 1951) this reviewer had access to a privately owned copy of the fourth edition, but the edition available at his own institution and the one generally available in the United States is the third and, in the present review of sections 5 and 6, comparisons, of necessity, are between the third edition published in 1932 and this fifth edition published nearly 20 years later.

These two sections contain 962 richly illustrated pages dealing with the thorax in health and disease and will be followed by still more material on the cardiovascular system as a part of section 7. The space devoted to the normal thorax has been doubled and now includes a consideration of microfilming, a detailed description of the technique of bronchography with water soluble contrast media, and a consideration of normal lung architecture as seen in bronchograms.

The pages dealing with nontuberculous diseases of the trachea, bronchi, and lungs have been expanded from 91 in the third edition to 354 in the fifth. Particularly noteworthy are the passages on congenital anomalies of the bronchi and the appearance of the bronchial tree in various types of bronchitis. Aspirated foreign bodies, pulmonary neoplasm, and the various pneumonias are adequately discussed and illustrated, and in addition to the so-called dust diseases the authors include a description of the findings following aspiration of aqueous and nonaqueous fluids and the inhalation of gases. For the mycotic pulmonary diseases the authors have turned largely to the American literature. Lung cysts, lung abscesses, and parasitic diseases of the lung are discussed and well illustrated. The story of pulmonary tuberculosis, which was told in 72 pages in 1932, takes up 113 pages in the new edition. One hundred and eighty pages are devoted to diseases of the pleura, diaphragm, and mediastinum, and these are followed by 27 pages of tables and sketches summarizing the criteria for the differential diagnosis of pulmonary disease.

The final 215 pages of section 6 are given over to the roentgen findings in the normal and diseased heart, pericardium, and great vessels, and this same subject will be continued as part of section 7, which is to follow. In 1932, 116 pages were considered adequate for this subject, but radiology has made great strides in 20 years. Much of the added material is concerned with angiography, a subject that was barely mentioned in 1932.

It is hard to conceive of a textbook of radiology more complete or more dependable than this magnificent fifth edition, which reflects so well the enormous development that has occurred in clinical radiology within two decades. In 1932, three authors plus six collaborators sufficed, but today four authors and 13 collaborators are required. The authors remain authors, however, and not mere editors, so that one finds a completeness, unity, and minimum of repetition that is apt to be lacking in a compilation by a group of individually competent contributors.

Einführung in die Röntgenologie: Ein Lehrbuch für Ärzte und Studierende. Von G. F. Haenisch, a. o. Prof. Dr. med., Chefarzt i.R. am allgem. Krankenhaus Barmbeck, Hamburg, und H. Holthusen, o.ö. Prof. Dr. med., Chefarzt am allgemeinen Krankenhaus St. Georg, Hamburg. Mit einem physikalisch-technischen Beitrag. Von A. Liechti. Fifth edition by W. Fehr. Cloth. 60 marks. Pp. 521, with 371 illustrations. Georg Thieme, Diemershaldenstrasse 47, (14a) Stuttgart O; agents for U. S. A., Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1951.

The chief authors of this extensive introduction to roentgenology are well known to Americans. This edition is greatly expanded beyond the first edition of 1933. There are 44 pages devoted to physical and technical background, and the section on diagnosis, by Dr. Haenisch, is 240 pages long. The second half of the book, by Holthusen, is a masterly exposition of the field of roentgen and radium therapy. Radioactive isotopes are fully described, and growth disturbances following gamma irradiation are discussed and illustrated. This is an area that has been neglected in American literature. Delayed or late damage to the skin and internal organs receives extensive attention as does irradiation sickness. The field of superficial and deep therapy is covered with a staccato brevity and yet a thoroughness that is characteristic of Holthusen as Americans know him. For a textbook on an introduction to roentgenology, Haenisch does not seem to have spared an area of useful exposition and illustration. One cannot escape the recollection of the story of the German who, in a prize competition on the elephant, offered three volumes entitled, an introduction to the study of the elephant. The authors have compressed their contribution into one volume, apparently without missing any single important segment of roentgenology. The quality of paper, the contrast in the lithographs, and the quality of the printing surpass the average, comparable, American textbook.

Contractor's Final Report on Artificial Arm and Leg Research and Development Covering the Period January 2, 1945, to December 31, 1950. Submitted by Northrop Aircraft, Inc. Contract number VAM-23062. National Research Council Committee on Artificial Limbs Advisory to Veterans Administration and the Office, Surgeon General, United States Army. This report compiled and written by Gilbert M. Motis. Cloth. Pp. 358, with illustrations. Northrop Aircraft, Inc., Northrop Field, Hawthorne, Calif., 1951.

This report is a detailed technical description of the application of modern engineering principles and materials to the problems of increasing the functional efficiency of the prosthesis and its attachment to the amputee. The design of many devices by Northrop engineers was facilitated by basic data derived from time and motion studies of arm and leg improvement made by the University of California at Los Angeles and the University of California (Berkeley). This work will serve as a valuable reference book to those concerned with the manufacture and fitting of prosthetic units and will be a stimulus for further research.

There is an interesting chapter on x-ray cinefluorographic technique and a short appendix on an experimental infant prosthesis. Northrop has produced a good upper extremity prosthesis that satisfies all basic maker's requirements and standard prerequisites of light weight, durability, comfort, and ease of operation. There are numerous detailed drawings and photographs of the prosthetic units, their component parts, and of the attachment of these units to the amputee. This represents a distinct advance in the mechanism of artificial limbs, and the book may be read with interest by those who are interested in the problems of amputees.

Clinical Applications of Recreational Therapy. By John Eisele Davis, Sc.D., Chief, Corrective Therapy, Veterans Administration, Washington, D. C. *Publication number 123, American Lecture Series, monograph in American Lectures in Physical Medicine.* Edited by W. A. Selle, Ph.D., Professor of Biophysics, University of California Medical School, Los Angeles. Cloth. $3.75. Pp. 118, with 21 illustrations. Charles C Thomas, *Publisher,* 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto 2B, 1952.

The author of this monograph attempts to supply a basic philosophy for the employment of "free play, exercise, and activity" in rehabilitation, especially from the viewpoint of psychodynamics. The 44 sections are distinct essays that usually take some previous publication as a text. Such topics as the psychological appeal of free play and recreational techniques based on concepts of milieu therapy are discussed.

This book is mainly concerned with the rehabilitation of patients institutionalized for psychiatric purposes. The number of these patients is increasing. The effects of institutional life on their initiative can be deadly, and many are not interested in doing anything that would be useful to others or to themselves. Even so, some readers will feel that this book unduly emphasizes recreational as distinguished from occupational therapy. The meticulous, expensive forms of recreational therapy suggested need not only be rationalized on the basis of theory but must also be defended on the basis of more practical considerations. With respect to the latter, the book leaves something to be desired. It must, however, be commended, as it is well written and attractively illustrated.

Fertility in Men and Women: The How and Why of Having Children. By James Alan Rosen, M.D., Attending Obstetrician and Gynecologist, Lincoln Hospital, New York. Edited with introduction for Health Series by Harry Swartz, M.D. Cloth. $3. Pp. 177. Coward-McCann, Inc., 210 Madison Ave., New York, 1952.

This little book is the first of a projected health series, under the general editorship of Dr. Harry Swartz. This volume is prepared for the lay reader, particularly those couples who have infertility problems. The author discusses the anatomy and physiology of the male and female reproductive apparatus and the techniques and methods used in diagnosing the contributory defects. There are chapters on treatment to alleviate the complaint and an attempt at evaluation of artificial insemination as a mode of treatment in infertility problems. The information offered is technically sound and should be clear to the lay reader, but the chapters lack the style and clarity of other recent books offered to the laity. The author includes a chapter of typical case histories, which might have some usefulness but suffer from lack of professional detachment and objectivity. The format of the book is attractive. The two illustrations are crudely designed. A useful glossary of technical terms is appended.

Handbook of Basic Microtechnique. By Peter Gray, Ph.D., D.I.C., Head, Department of Biological Sciences, University of Pittsburgh, Pittsburgh. Cloth. $3. Pp. 141, with 57 illustrations. Blakiston Company (Division of Doubleday & Company, Inc), 1012 Walnut St., Philadelphia 5, 1952.

This is intended as a useful handbook for the student or technician faced with the problem of preparing animal or plant material for microscopic examination, and supplements Gray's "Handbook of Basic Microtechnique," a larger reference work now in preparation. It is an inexpensive book for quick consultation on microtechnical procedures. Part 1, devoted to materials and processes of slide making, has chapters on materials and equipment, fixation and fixatives, stains and staining, dehydrating and cleaning, mounts and mountants, making whole mounts, making smears, making sections, and cleaning, labeling, and storing slides. Part 2, devoted to specific examples of slide making, has 15 short chapters on the preparation of whole mounts, bacterial stains, and certain histological sections. This handbook meets its objectives in full and is recommended to students or technicians engaged in histological procedures.

Symposium on Geographical Pathology and Demography of Cancer. Council for Coordination of International Congresses of Medical Sciences under auspices of World Health Organization and United Nations Educational, Scientific and Cultural Organization, session held at Regent's Park College, Oxford, England, July 29 to August 5, 1950. Chairman: Prof. J. H. Maisin. Editor: Dr. J. Clemmesen. Cloth. Pp. 152, with illustrations. The Council, 19, Ave., Kléber, Paris 16^e, [n.d.].

This volume contains the papers read at a symposium held in Oxford, England, in 1950, under the chairmanship of Prof. J. H. Maisin and sponsored by the World Health Organization (WHO) and the United Nations Educational, Scientific, and Cultural Organization (UNESCO). Several papers are devoted to statistical variations in cancer incidence between different countries or within a single country. A paper by R. Doll on the etiology of cancer of the lung, especially the correlation between incidence among tobacco smokers and nonsmokers is a careful statement of current English studies. Other papers analyze cancer incidence in India, Indonesia, and North Africa in relation to nutrition and food habits, environment, and other disease states. Dr. Symeonidis from the National Cancer Institute introduces interesting observations on the relationship of poststarvation gynecomastia in Greece to hormonal imbalance and its implication as a factor in breast cancer. The administrative aspects of geographic pathology as now being developed at the National Cancer Institute are described by H. L. Stewart. Cancer demography is emerging as a definite field, and the contributors to this symposium have indicated the scope and range of investigations now developing in this field in a most effective manner. Discussions by members of the conference are included for each chapter. Each paper has an abstract in both French and English. The volume is well printed, and the graphs and charts are clear and legible. The microphotographs are poor.

Elsevier's Encyclopædia of Organic Chemistry. Founded by E. Josephy and F. Radt. Edited by F. Radt. Series III, Carboisocyclic Condensed Compounds. Volume 14, Supplement: Tetracyclic and Higher-Cyclic Compounds Except Steroids and Triterpenes. [Cumulative index]. Cloth. $88; $77 to subscribers to series III; $66 to subscribers to complete work. Pp. 938 S; 113. Elsevier Press, Inc., 402 Lovett Blvd., Houston 6, Texas; 118 Spuistraat, Amsterdam, Netherlands, 1951.

Volume 14 in the regular series of this work covered the literature on tetracyclic and higher cyclic compounds up to and including 1936. The growth of studies on these compounds has been so rapid since that time that the editors have published a supplement that brings the literature up to 1946. The amount of work done on these substances in that 10 year period has increased to the extent that it is necessary to publish the supplement in two volumes. The first of these two volumes is now available and covers all tetra and higher cyclic compounds with the exception of the steroids and triterpenes, which will appear in the second volume.

The subject index includes references to both the main volume and the supplement, with a compilation of all substances that fall into this chemical class under such index headings as carcinogenic compounds, estrogenic compounds, and naturally occurring compounds in coal tar, minerals, and plants. Such features as a summary table of the carcinogenic activities of 1,2-benzanthracene derivatives is included. The physiological properties of many interesting parent compounds and their more important derivatives are discussed. Among those discussed in the present supplement are fluoranthene, naphthacene, benzanthracene, dimethyl-benzanthracene, chrysene, benzophenanthrene, pyrene, dibenzofluorene, cholanthrene, 20-methyl-cholanthrene, dibenzanthracene, benzopyrene, and hypericin.

Contractura isquémica de Volkmann. Por F. Bastos Mora. Cloth. Pp. 119, with 44 illustrations. Publicaciones médicas José Janés, editor, Muntaner 316, Barcelona, 1951.

This is a detailed, well-written monograph that covers the subject thoroughly. The author has evidently drawn from extensive experience with this condition. The pathogenesis and pathology of Volkmann's contracture are discussed in detail. The complete clinical picture from the time of initial injury to the final tendon transplant is discussed clearly, concisely, and adequately. Alternative methods of surgical reconstruction are also discussed. The illustrations are satisfactory and the bibliography good. This work will prove exceedingly useful to those interested in Volkmann's contracture.

QUERIES AND MINOR NOTES

ALLERGY AND DUODENAL ULCER

To the Editor:—*Is allergy still recognized as a cause of duodenal ulcer? Can food allergy aggravate an existing duodenal ulcer?*

M.D., Ohio.

Answer.—A discussion of allergic factors in the pathogenesis of duodenal ulcer by Andresen and Grossman may be found in the book entitled "Peptic Ulcer" (edited by Sandweiss, D., Philadelphia, W. B. Saunders Company, 1951). It is clear, as the result of a wide variety of experimental studies, that allergic reactions can be produced in the mucous membrane of the stomach or the duodenum very much as they can be produced in the skin. For the most part, however, they heal readily and do not give rise to chronic, progressive ulcers resembling those seen in man or those produced in experimental animals either by the Mann-Williamson procedure or by the transplantation of the antrum of the stomach into the colon by the method of L. R. Dragstedt and his associates. Andresen believes that an allergic response to some of the products arising in the body as a result of foci of infection may produce typical ulcers and may also cause their recurrence. He suggests that the beneficial effects of removal of foci of infection may in part be due to these factors. Most of the experimental and clinical evidence at present available indicates that duodenal and gastrojejunal ulcers are due to the corrosive properties of the gastric content that are greater than normal because of hypersecretion of gastric juice in the fasting stomach as well as after the ingestion of food. This hypersecretion is thought by Dragstedt to be of nervous origin, and this is the basis for the vagotomy operation.

CEPHALIN FLOCCULATION TEST IN HEPATITIS

To the Editor:—*Is information available on the length of time a cephalin flocculation test remains positive after a patient is clinically free from hepatitis?*

F. A. Nause, M.D., Sheboygan, Wis.

Answer.—It is difficult to give a categorical answer to this question without knowing the technique of the cephalin cholesterol flocculation test performed. As a general rule, the test becomes negative at or about the time the serum bilirubin returns to a normal figure. This was the general experience with volunteers and has been confirmed many times in studies on the naturally occurring form of the disease. In a recent study m the army hepatitis center in Germany, only 10% of 667 tients studied had a positive cephalin cholesterol flocculation st by the end of the fourth week of the disease when the 48-hour flocculation test was used. When the 24-hour flocculation test was used, the percentage of positive tests at the end of the fourth week was more than twice as high. By the end of the third month of the disease, only about 2% of the 48-hour flocculation tests were still positive (Neefe, J. R.; Gambescia, J. M.; Gardner, H. T., and Knowlton, M.: Comparison of the Thymol, Cephalin-Cholesterol Flocculation and Colloidal Red Tests in Acute Viral Hepatitis, *Am. J. Med.* 8:601 [May] 1950).

Sborov and Keller have recently presented figures on patients with the mildest form of the disease: 74% of patients with serum bilirubins of less than 5 mg. per 100 cc. had negative cephalin flocculation tests by the third week of the disease. In patients who had serum bilirubins at a higher level a much higher incidence of positive tests was obtained. In general, one ay say that a patient who has a positive cephalin cholesterol occulation test, which persists beyond the third month from the onset of jaundice, deserves investigation by other methods to be certain that subacute or chronic hepatitis is not developing.

LUMBAR MENINGOMYELOCELE

To the Editor:—*I repaired a lumbar meningomyelocele on a 1-day-old infant. The surgical procedure was uneventful, and the child has done reasonably well. Two weeks later, a neurosurgeon who examined the child said the repair should not have been done, because it was unnecessary and the removal of a sac from which fluid might be absorbed would cause hydrocephalus. We have had, in our hospital, about six of these infants in the last two years. None of them were treated surgically, and hydrocephalus developed in all of them. It is difficult for me to understand how absorption of fluid from the spinal canal (arachnoid) could, in any way, affect an internal hydrocephalus and why repair of a meningomyelocele would have anything to do with the development of hydrocephalus. What is the proper treatment of a lumbar meningomyelocele without skin covering?*

M.D., Texas.

Answer.—The selection of patients with lumbar meningomyelocele who can be surgically treated must of necessity be on an individual basis. The surgeon should elect to operate only on those patients whom he believes he can help. The fact that it is surgically possible to repair the defect on the back is not, in itself, sufficient indication for operation. Children whose legs are paralyzed, who have a loss of sensation in the sacral distribution, who have a paralysis of bowel and bladder, or in whom a hydrocephalus is developing, are not suitable candidates for surgical repair of the deformity.

There is no good evidence that removal of the meningocele sac is responsible for the development of hydrocephalus. On the contrary, the study of a large number of cases has shown that hydrocephalus develops as frequently if the meningocele is not operated on. This is not surprising, because pathological studies demonstrate that the abnormality responsible for the hydrocephalus lies in the head and not in the spinal canal. It is true that the idea that the sac absorbs cerebrospinal fluid has been advanced, but it has not been generally accepted nor is it in accord with the facts.

If a congenital deformity of this nature is suitable for surgical repair, the redundant skin and meninges should be excised, the dura mater should be closed tightly, and fascial flaps should be reflected from the paravertebral muscles on both sides and imbricated over the defect in the spinal canal. The skin is then closed. No attempt should be made to close the bony defect.

LIVER BIOPSY

To the Editor:—*Please tell me the technique used for obtaining a needle biopsy specimen of the liver.*

J. C. Eakle, M.D., Sutton, W. Va.

Answer.—Liver biopsy can be performed to advantage with the "vim"-Silverman needle, a 14-gage needle having a hypodermic bevel tip. It contains an inner needle that is split longitudinally and has a cutting edge. After preparation of the skin and a preliminary dose of morphine sulfate, a small nick is made in the skin, usually at the right midclavicular line, just below the costal margin. The outer needle is inserted with the inner needle withdrawn, so that its point is above the bevel of the outer needle. The inner needle is then moved forward into the substance of the liver.

As it advances, it cuts out a small cylinder of liver tissue, about 2 mm. in width. The outer needle is then advanced over the inner needle, thus trapping the specimen, closing the prongs of the inner needle, and separating the innermost portion of the tissue to be removed. Both needles are then withdrawn simultaneously. The small specimen of tissue obtained, usually measuring 2 cm. by 2 mm., is, as a rule, adequate for a study of liver histology.

Most operators confine their biopsies to patients who have hepatomegaly and in whom the subcostal approach is possible;

The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

however, many who are more experienced do not hesitate to obtain specimens through the ninth intercostal space in the posterior axillary line. The risk of the procedure is relatively small, provided one avoids the danger of bleeding. Bleeding, coagulation, and prothrombin times should be determined routinely on all patients for whom liver biopsy is contemplated. If there is any evidence of there being an inflammatory process within the abdomen or at the right lung base, the procedure is contraindicated. About 5% of patients have some pain postoperatively; this usually disappears within a few hours. There is an excellent account of the various instruments and techniques used in an article by Hoffbauer (*J. A. M. A.* **134**:666 [June 21] 1947).

MILD CONVULSIVE SEIZURES IN INFANT

To the Editor:—*A primipara delivered a child easily following an uneventful pregnancy. An episiotomy was performed, but forceps were not used. The child at birth weighed 9 lb., 12 oz. (4,422.52 gm.) and seemed to be normal. About two and one-half months after birth, mild convulsive seizures developed in the child. During bottle-feeding or when he is lying at ease in the crib, he suddenly has clonic contractions of the arms and legs, with inconstant horizontal nystagmus. The first attack lasted about 30 seconds; subsequent ones have been less severe. After the first two attacks he cried loudly and long and then quieted down and was apparently normal. During the seizures he turns pale and appears to lose consciousness to varying degrees. During the first two attacks, the baby seemed to have colic. Recently, he has had a mild upper respiratory tract infection and had the last few attacks with the onset of the infection. The child has been examined by several pediatricians, all of whom gave the opinion that he is normal. An electroencephalogram was made by a neurologist, who reported normal results. The child eats well. I would appreciate a discussion of the etiology, treatment, and prognosis of this condition.*

Edgar E. Struve, M.D., Fullerton, Calif.

Answer.—With the normal birth history and normal results of physical examination, it seems reasonable to rule out birth injury as a cause for these attacks. Infantile tetany could be excluded as a cause by the rarity of its occurrence before the age of three months and by the lack of a history of rickets or of an abnormal condition observed during physical examination. The existence of a normal blood calcium level would eliminate the possibility of infantile tetany, as would normal reactions to electric stimulation. Parathyroid tetany is a rare condition caused by hypofunction of the parathyroid. When noted in infancy, it occurs in the period immediately after birth and is characterized by a low blood serum calcium level. Tetany may also be associated with alkalosis caused by vomiting and loss of acid gastric secretions, by hyperventilation, by the administration of salts that form insoluble calcium compounds, and by nephritis with chronic renal insufficiency, in which the level of inorganic phosphates of the blood serum rises and the level of blood serum calcium falls. In the case in question it is assumed that the results of urinalysis are normal and that no nephritis is present. These conditions include most of those that have a physical etiological basis and, from the data furnished, can probably be quite easily excluded from possibility.

Some functional conditions may give rise to the symptoms shown by this infant. Occasionally, an infant shows unusual susceptibility to many kinds of stimuli, and has prolonged spells of crying and muscle spasms causing flexion of the arms and legs and clenching of the hands. He is easily startled, frequently thrashes about, and may evidence breath holding and pallor; such a baby is termed a hypertonic infant. In the past, these babies were thought to suffer from vagotonia and were often treated with atropine. The exact pathogenesis of this condition is not clear, although heredity probably is an important factor. These infants respond favorably to hospitalization or the substitution for their care of a calm nurse for easily agitated parents. Mild sedation with phenobarbital sodium for several weeks is often helpful. The prognosis is generally good: the symptoms usually subside completely, and permanent emotional instability is not a necessary aftermath of this condition.

HYDROCEPHALUS AND SPINA BIFIDA

To the Editor:—*A child, who had mild hydrocephalus and severe spina bifida but was apparently normal otherwise, weighing 6 lb. and 15 oz. (3.15 kg.), died five days after birth. This was the mother's third pregnancy and her first son. Two other children are normal. The mother is one of three children, all girls, the first of whom had a congenital defect of the spine, described by the grandparents as typical of mild spina bifida. The mother also had two half-sisters, neither of whom had any congenital defects. There is no history of congenital defects in her husband's family. Are hydrocephalus and spina bifida definitely hereditary? If so, what were the chances of this child being affected, since two others were normal? What are the chances of a fourth or fifth child being normal? Could the mother's health during the third pregnancy have influenced the appearance of the defects? (She suffered from hypothyroidism, fairly well controlled by thyroid extract, 4 grains [0.25 gm.] daily, mild hypoglycemia, and moderately severe secondary anemia.) Does the sex of the child influence the likelihood of congenital defects of this type?*

M.D., Georgia.

Answer.—Ordinarily, neither hydrocephalus nor spina bifida is definitely hereditary. Spina bifida, with visible deformity on the back, such as a meningocele or a meningomyelocele, occurs in about one out of every thousand births. Spina bifida occulta, which is usually asymptomatic and often without external manifestation, occurs in about 5% of the population. This is in addition to spina bifida of the lowest sacral segments, which occurs in practically everyone. Occasionally, instances of congenital defects in the spinal column, associated with deformities of the nervous system, have shown a definite hereditary or familial tendency. In the present instance, the history, as given, is not sufficiently detailed to permit one to judge whether there is a familial tendency present here. The nature of the congenital defect said to have been present is not stated nor are we certain of the presence of congenital defects in some members of this family. In view of the inadequate information on these points, positive statements beyond those given above are not possible. The cause of the congenital deformities of the spine in persons in which they appear is unknown. It is conceivable that the mother's health could be a factor. Whatever the cause, it must be effective within the first month of intrauterine life. After that date, the development of the nervous system has proceeded to such a point that this type of deformity would not be possible. There is little evidence that sex of the child influences this type of congenital deformity. The matter is fully discussed in P. C. Bucy's "Spina Bifida and Associated Malformations" (in Practice of Pediatrics, edited by J. Brennemann, Hagerstown, Md., W. F. Prior Company, Inc., 1937, vol. 4, chap. 15.)

TREATMENT OF PAIN IN ARTHRITIS

To the Editor:—*Two products in tablet form have come to my attention for the treatment of rheumatoid arthritis. One contains p-aminobenzoic acid and the other, sodium gentisate. I am interested in knowing which drug has proven more effective in alleviating the pain of rheumatoid arthritis.*

Francis M. Walker, M.D., Rochester, N. Y.

Answer.—Sodium gentisate was reported in 1948 to be as effective as salicylates in relieving articular pain in rheumatoid arthritis; however, it possesses no special advantages over salicylate preparations, except that it may be tolerated in a patient who is unable to take either aspirin or sodium salicylate. *p*-Aminobenzoic acid, in common with other benzoates, exerts some analgesic action, although inferior to that gained from salicylates. About five years ago, its use in conjunction with sodium salicylate was studied. The conclusion was drawn that the blood salicylate level was thus increased, chiefly by means of decreasing the excretion of free salicylate. Determinations made subsequently have failed to confirm these findings, although a clinical study has shown that an enhanced analgesic effect may be obtained by the addition of *p*-aminobenzoic acid to oral doses of sodium salicylate. In the experience of many who treat rheumatoid arthritis, aspirin is more consistently effective than sodium salicylate in relieving pain and stiffness and is therefore the drug chosen. Sodium salicylate may be substituted for patients who show gastric intolerance to aspirin in any form.

EFFECTS OF DIAGNOSTIC RADIATION

To the Editor:—*In several cases of apparently highly radiosensitive tumors, after routine initial diagnostic roentgenologic work-up (chest x-ray examination, gastrointestinal series, or urological radiographs), the hematological picture has shown an increase in hemoglobin, red blood cells, and thrombocytes, without any medication or change in food intake. Can it be that in the initial phase of the disease, the small dose of roentgen radiation thus incurred and directed unknowingly at the suspected cancer was effective in causing secondary hematopoiesis by inhibiting growth of the mass and metastases or in stimulating primary hematopoiesis?*

C. D. J. Generales, M.D., New York.

Answer.—It is likely that the amount of radiation received in certain diagnostic procedures can produce recession of highly sensitive malignant tumors, but it does not seem possible that the blood changes described could be due to a primary or direct effect on the hematopoietic system. The cells of the hematopoietic system are highly sensitive to radiation, but the effect of the latter is to produce destructive, rather than productive, changes. Neither does it seem probable that a destructive effect on a primary tumor or its metastases could produce the stimulative effect described. It seems more likely that the improved blood picture in the observed cases is either coincidental or due to improvement in the patient's general health.

ESTROGENS AND CANCER OF THE UTERUS

To the Editor:—*A 49-year-old patient with adenocarcinoma of the uterus and metastasis was elsewhere put on estrogen because of nervousness and other symptoms following bilateral ovariectomy. I am treating her with deep x-ray therapy, but I am not following the doctor's instructions about giving her estrogen, as I am a little skeptical, regardless of her menopausal symptoms. Is there definite proof that the administration of estrogen under these circumstances is a hazard?*

M.D., Alabama.

Answer.—There is no definite evidence that estrogens are carcinogenic. In spite of this inconclusive evidence, it is the opinion of a number of gynecologists that it would be better to withhold the hormone in patients who have carcinoma or who have had it. Cancer of the endometrium is not found frequently in the castrate, and Dockerty reports that prolonged, unopposed estrogen stimulation may be a factor in the production of such endometrial changes. Therefore, the usual menopausal symptoms in a woman of this age would best be controlled either with sedation or with the male hormone, testosterone.

PHARMACOLOGY OF MAGNESIUM SULFATE

To the Editor:—*Please summarize the pharmacodynamics of magnesium sulfate as related to its use in the treatment of toxemias of pregnancy. What, if any, are its toxic effects on the body? In three instances we observed gross hematuria after intramuscular injection of 10 to 20 cc. of 50% magnesium sulfate, and we are reasonably sure that bleeding did not occur from an indwelling catheter present at the time. Is it possible for a chemical nephritis to have occurred?*

W. R. Fallon, M.D., Chicago.

Answer.—Magnesium sulfate is used in the treatment of preeclampsia and eclampsia because of its sedative and vasodilator effects. These pharmacologic actions favor a decrease in the blood pressure and allay the convulsive seizures. The efficacy of the drug as a central nervous system depressant and as a vasodilator has been substantiated by experimental as well as clinical evidence. It is considered the most efficient drug for allaying the convulsions of tetanus. It has been used extensively in the treatment of hypertension and uremia because of its vasodilating effects. Haury has shown that the intravenous injection of magnesium sulfate produced marked dilatation of the splanchnic organs and the kidneys.

Magnesium sulfate is a respiratory depressant, and, when used in any dosage, it is prudent to have an antidote readily available in the form of a 10% solution of calcium chloride or calcium gluconate. Calcium salts should be administered slowly if the patient has been digitalized. Hematuria following the administration of magnesium sulfate has not been reported. The amount administered to this patient is not excessive, for as much as 35 gm. has been given to a patient during the first 24 hours of eclampsia without undue effects.

THE SCLERA IN CAROTENOSIS

To the Editor:—*Can hypercarotenemia produce yellowish pigmentation of the sclera as well as of the skin? If so, which appears first and why? Is there any relationship to jaundice?*

Max S. Lake, M.D., Salina, Kan.

Answer.—The yellow pigmentation of the skin known as xanthosis, or carotenosis, is caused by an excess of carotene in the blood and is most marked in the palms, soles, and nasolabial folds. In contrast to jaundice, the scleras usually remain clear. The differential diagnosis of xanthosis and jaundice depends on the history and other findings, a knowledge of the patient's diet, the concentration of bilirubin in the serum, the icterus index and the lipochrome index. When with yellow pigmentation of the skin a low value for the serum bilirubin and a high icterus index are present, the condition must be xanthosis; if the value for the serum bilirubin is high and the lipochrome index is low, the abnormal coloration is due to jaundice. If both the concentration of serum bilirubin and the lipochrome index are elevated, xanthosis and jaundice are associated.

REFERENCE:

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TREATMENT OF SMALL AREA OF DRY GANGRENE ON ARM

To the Editor:—*Advice is requested regarding the treatment of of an area of dry gangrene. The area is situated on the volar, medial aspect of the left upper forearm and is about 1 in. (2.54 cm.) by 1½ in. (3.81 cm.) in size. This condition developed after the paravenous injection of a small amount of 10% calcium levulinate. The black, dry, necrotic crust is sharply demarcated from the unaffected skin, which does not show any inflammatory reaction. On palpation, the underlying tissues and possibly the muscle appear to be affected by the necrotic wedge. There is no pain, but use of the left forearm is somewhat impaired.*

M.D., South Dakota.

Answer.—This necrotic lesion should be treated conservatively. In most instances, the necrotic area will slough off, and healing will take place spontaneously. If, after several months, this does not occur, surgical excision may be necessary, and granulation will then probably fill in the defect.

ACTIVITY AFTER OPERATION FOR UTERINE PROLAPSE

To the Editor:—*After a plastic operation for complete prolapse of the uterus, how soon can the patient be out of bed? How soon can she do heavy work, and how soon can she have sexual intercourse?*

M.D., Central America.

Answer.—The pattern of postoperative care has changed markedly in recent years. There is no reason why the patient cannot be allowed out of bed to the bathroom or to use a bedside commode the day after surgery. Early ambulation is desirable, because many of the patients are elderly and early activity decreases the hazards of thrombosis and infarction. Ambulation should be restricted during the first week. Heavy work should be postponed for at least eight weeks. Sexual intercourse is permissible six or eight weeks after surgery if the healing has been satisfactory. The administration of an estrogen, such as 0.5 mg. of diethylstilbestrol, each night for three or four months after surgery favors healing and the restoration of a normal vagina in the woman in the postmenopausal period.

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RECENT ADVANCES IN THE MEDICAL TREATMENT OF CIRRHOSIS OF THE LIVER

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and
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The incidence of cirrhosis of the liver is increasing. Whereas previously it was seen mostly with alcoholism or malnutrition or as a complication of cholecystitis and cholelithiasis, physicians today are confronted with additional causative factors. The widespread epidemic hepatitis of World War II produced a sporadic inoculation of persons hitherto never exposed to the virus of infectious hepatitis. The etiological relationship of acute infectious hepatitis to cirrhosis of the liver is favored by most authors.[1]

Sulfonamides and other hepatotoxic agents have taken their toll of the liver. Aureomycin has been rather extensively used in the treatment of acute and chronic hepatitis. Recently Lepper [2] reported that, among 14 patients given large doses of aureomycin for serious infections, both by mouth and intravenously, 7 showed clinical evidence of liver dysfunction. Furthermore, necropsy in five and biopsy in one of these cases showed pathological changes in the liver cells; however, the doses of aureomycin used are now considered to be excessive.

Finally, the establishment of blood banks and the use of nonirradiated plasma have resulted in serum homologous hepatitis in some patients and have made cirrhosis of the liver a disease of serious import. No longer can one regard blood transfusions as an innocuous procedure unless the medical history of the donor is known.

GENERAL MEASURES

Certain general measures are applicable in the treatment of all types of liver disease. The recognition of the cause of the liver disease is necessary for effective therapy, and removal of the causative factors assumes a major role whenever they can be determined. In the treatment of jaundice one is constantly confronted by the immediate question of whether one is dealing with a medical or a surgical problem. It may not be amiss at this time to emphasize the importance of cholecystectomy and common duct exploration when indicated as a therapeutic measure in the treatment of liver disease. It is well established that cholecystitis usually produces hepatitis, the degree depending on the resistance of the liver on the one hand and the severity of the inflammatory process in the biliary tract on the other. We are firmly convinced that cholecystectomy should be done whenever cholelithiasis is found, in order to prevent liver damage. Certainly the morbidity and mortality of operative intervention can be markedly decreased if such operations are done before liver damage supervenes or when the liver damage has been improved by the therapeutic measures outlined above. This matter should be seriously considered unless there is a definite contraindication for operative intervention. In our experience contraindications become fewer when a careful evaluation of a surgical risk is made and scientific management is substituted for the haphazard surgical procedures previously employed.

It is preferable that the treatment be carried out in a hospital, where repeated studies of the progress of the disease may be made. When the liver damage is more than minor, it is best also to keep the patient in bed. The use of drugs should be avoided as much as possible, and it should be kept in mind that even the commonly used barbiturates and sulfonamides can impair the function of the normal liver and constitute an added strain on a damaged liver.

Constipation also is an added strain and should be treated with mild laxatives. Bile acids are preferable to bile salts because they produce choleresis and therefore decrease the viscosity of bile. Dehydrocholic acid may be used, 3 to 5 grains (0.20 to 0.30 gm.) three times a day. It not only is a laxative but helps to reduce intrahepatic bile capillary pressure. Furthermore, if the constipation is interdependent on overplay of the sympathetic nervous system and inactivity of the parasympathetic nervous system, parasympathomimetic drugs, like neostigmine (prostigmin®) and benzpyrinium (stigmonene®) bromide, stimulate the gastrointestinal tract, so that perfectly

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normal bowel movements are possible without irritation of the lining of the bowel. If one prefers other preparations, however, saline laxatives, effervescent sodium phosphate, or magnesium citrate are much better for the patient than the harsher magnesium sulfate or similar drugs, which, we feel, irritate the intestinal tract too much.

DEXTROSE THERAPY

Intravenous administration of dextrose is one of the most powerful therapeutic measures in the physician's armamentarium for the treatment of liver disease. It is all-important to maintain the glycogen content of the liver cell. It is possible that hyperglycemia inhibits neoglycogenesis and results in needed rest to the enzyme systems responsible, thus contributing to faster recovery. For this, several measures are suggested. If ascites is present, one should start with concentrations of 50% dextrose in volumes of 100 to 200 cc. daily, continuing until the output of fluids exceeds the intake. After this, administration of 300 to 400 cc. of aqueous 25% dextrose solution should be continued until there is another demonstrable increase in the output of fluid, when one may use 10% aqueous dextrose solutions in volumes of 3,000 to 4,000 cc. daily. If at any time during this regimen the output of fluids is less than the intake, one may have to resort as before to use of the more concentrated solutions to restore the fluid balance.

A difference of opinion exists, however, concerning the advantages of intravenous dextrose therapy if the patient can take the "necessary" dextrose or carbohydrate by mouth.[3] The "necessary" amount of carbohydrate is that amount sufficient to raise the blood sugar to a level that will suppress hepatic sugar output. However, while the normal liver will respond to the usual postprandial hyperglycemia, associated with a high carbohydrate intake, the "irritable" liver in patients with acute toxemia may require a much higher blood sugar concentration to inhibit gluconeogenesis. That this is so is seen in the prompt curtailment by the acutely poisoned liver of its output of sugar when large doses of dextrose are given intravenously, while small doses have little or no effect.[4] Furthermore, as Cori and Cori [5] have pointed out, for the normal liver "the blood sugar concentration and not the amount of glucose administered must be regarded as important for the rate of its glycogen deposition." Consequently, when an attempt is made to protect a damaged liver by means of glycogen deposition therein, the blood sugar concentration may have to be raised to a level impossible to obtain by ingestion of carbohydrates. In such cases, the intravenous infusion of dextrose is essential. The fact that extreme hyperglycemia so produced may result in glycosuria should not deter one from such vigorous therapy. Wakim and Mann [6] have shown that regeneration of hepatic tissue after injury begins in islands of normal cells that are supplied mainly by arterial blood, while the cells in areas supplied only by portal vein blood may be too badly injured to regenerate. If the cells from which regeneration is to originate are to receive the full benefit of high dextrose concentration so that they can begin to restore liver parenchyma, the hyperglycemia following intravenous therapy is desirable.

Because of the glycosuria that may result from intravenous dextrose therapy, some physicians favor the routine use of insulin with the sugar. However, unless the patient has diabetes, the indiscriminate use of insulin may defeat the purpose of dextrose administration. Soskin, Allweiss, and Cohn [7] have shown that, in the presence of sufficient insulin to maintain a normal constant blood sugar level, no additional insulin is necessary to obtain a normal hepatic response to administered sugar. The injection of insulin into the nondiabetic person can, therefore, produce no additional hepatic effect, but it does cause increased storage of glycogen in the muscles. This peripheral effect causes a fall in blood sugar, which in turn stimulates the liver to pour out more sugar. Thus, the liver is deprived of glycogen rather than the reverse. Maintaining the hyperglycemia in spite of the administered insulin will prevent this reversal in effect but will not be more beneficial than the administration of the same amount of sugar without insulin. Soskin, Allweiss, and Mirsky [8] have shown that the use of insulin with dextrose, as compared with the use of dextrose alone, in the treatment of toxic, nondiabetic animals shortens life. After prolonged treatment by intravenous dextrose injections designed to suppress the sugar-producing mechanism of the liver, that organ requires time to recover from the inhibition of dextrose formation, so that hypoglycemia may appear one to three hours after cessation of the infusion.[9] This should be anticipated and treated with small doses of carbohydrate by mouth or intravenously if necessary. Many patients with atrophic cirrhosis and associated ascites have been definitely relieved by the daily parenteral injection of hypertonic dextrose solution, which not only is a factor in liver repair but acts as a diuretic agent. However, in patients with prolonged, overwhelming changes of the liver, this treatment may not produce as striking results as in those with early disease.

USE OF AMINO ACIDS

If the patient is unable to eat a full diet, it may be well to administer infusions of amino acids in doses adequate to maintain the normal nitrogen balance. Eckhardt and associates [10] and Lewis and co-workers [11] have questioned that hydrolyzed proteins or amino acids can be utilized normally in the presence of liver damage that may impair the deamination capacity. Eckhardt and associates [10] demonstrated that 500 to 1,000 cc. of 10% amino acid solution administered intravenously each day over a period of one-half to one and one-half hours was metabolized and excreted by patients with diseased liver in a manner differing little from normal. It was further demonstrated that, when this solution was the only source of nitrogen and lipotropic substance, clinical improvement occurred. Lewis and associates [11] and Fagin and Zinn [12] have reported similar results. A possible slight decrease in urea formation and a slight decrease in the rate of amino acid disappearance was reported in cirrhotic patients. Amino acids administered intravenously rapidly are not well tolerated either by cirrhotic or by normal persons. Evidence suggests that reactions are no more frequent in one group than in the other. Frequent side-effects include flushing, nausea, and vomiting.

VITAMIN THERAPY

The role of vitamin therapy of chronic liver disease is not entirely clear at this time. While it has become almost routine to include supplementary administration of vitamin B complex in the management of cirrhosis of the liver, definite evidence for vitamin B deficiency in cirrhosis per se has not been demonstrated.[13] Such vitamin therapy may be expected to be of value only when specific deficiency exists. Moreover, certain theoretical considerations suggest possible damage from excessive use of vitamins. For example, the administration of nicotinic acid in large doses may be harmful in the presence of fatty liver (Chalmers and Davidson [14] and Handler and Dubin [15]). A portion of this vitamin is excreted as the methyl derivative. In animal experiments this has been shown to hasten the development of cirrhosis when a deficient diet was given. A possible antilipotropic effect has been implied.[15] McHenry [16] has suggested that thiamine hydrochloride therapy under certain circumstances may promote fat infiltration in the liver. Morgan [17] has deduced from experimental work that increasing the intake of nicotinic acid of persons with multiple vitamin deficiencies may precipitate conditions worse than the subacute deficiency state produced by an inadequately balanced diet. György [18] has pointed out that it is important to include adequate vitamin B complex in the diet but that imbalance among elements of the B group should be avoided. The deduction is that massive use of any one element of the vitamin B complex is likely to be of little benefit when no specific deficiency exists and may be harmful. Cautious and specific use is therefore necessary. The role of vitamin B_{12} and its possible interrelation with folic acid, choline, and methionine have not been sufficiently explored to permit definitive comment at this time. Vitamin C administration is of value in protecting against some hepatotoxins and is used therapeutically to protect some of the enzyme systems and improve resistance of the liver cells. Vitamin A deficiency is common in liver disease because of impaired absorption resulting from bile deficiency, from impaired conversion of carotene to vitamin A, and from fixation of vitamin A by areas of damaged liver parenchyma. Vitamin D absorption is also defective because of biliary deficiency.

To prevent these deficiencies and for possible protection against loss of glycogen from the liver, supplementary doses of these vitamins should be given to the patient. Dried yeast in divided doses, amounting to 30 to 50 gm. per day, or liquid concentrates of yeast are to be used when tolerated, especially in cases of cirrhosis. Citrus fruit juices, 250 to 360 cc. daily, should be used as a source of vitamin C. Vitamins A and D in doses of 5,000 and 1,000 I. U., respectively, given once or twice daily, are of definite value. The vitamin B furnished by the dried yeast may be supplemented parenterally with thiamine hydrochloride doses of 10 to 20 mg. and nicotinamide in doses of 50 to 100 mg., especially in cases of severe liver damage, when the patient should also receive liver extract orally, 30 cc. three times daily, or 3 to 5 cc. of crude extract parenterally, two or three times per week.

Associated with the decreased flow of bile into the intestine in jaundice is a deficient absorption of vitamin K; when the liver is damaged, there is also a decreased production of prothrombin independent of the absorption of vitamin K. Hypoprothrombinemia results from inadequate absorption or use of vitamin K and is responsible for much of the hemorrhagic tendency that is common in patients with liver disease. Capillary damage and formation of abnormal blood clots are also important factors in this hemorrhagic tendency; however, and it should be recognized that hemorrhage is not always associated with hypoprothrombinemia. Aggeler and Lucia,[19] nevertheless, suggested that it is advisable to administer vitamin K to all patients with liver disease or jaundice, regardless of the prothrombin or bleeding time. It is most important to prepare such patients well for operations; a dose of 10 mg. of vitamin K given parenterally on at least four days before operation and four days after operation is suggested as a good routine practice. Usually a dose of 5 to 10 mg. of vitamin K given parenterally or 5 to 10 mg. orally will bring the prothrombin time to a maximum in 24 hours, and the effect will persist for four days. When liver damage is so severe that there is little response to vitamin K therapy, an infusion of 500 cc. of undiluted plasma will usually correct the hypoprothrombinemia to well beyond the danger point (60% of normal).

Evidence by Meyer and Howard [20] showed that the oral administration of acetylsalicylate and sodium salicylate to human beings in daily doses of 20 to 80 grains (1.2 to 5.3 gm.) consistently produced hypoprothrombinemia and hypocoagulability of the blood. This may be prevented by administration of vitamin K with the salicylate. Hemorrhagic manifestations with acute rheumatic fever may, in some cases, be due to the administration of large doses of salicylates.

Similarly, Shapiro and associates [21] reported that in man, as in the rat, salicylate in adequate doses induces prothrombinopenia, which can be prevented by administration of vitamin K if liver function is adequate. Cirrhosis of the liver and preexisting hypoprothrombinemia augment the effect of salicylate. Acetylsalicylate appears to be more potent than sodium salicylate. The salicylates act, apparently, in the same way as bishydroxycoumarin (dicumarol®) but less effectively.

On the basis of this evidence, one might sound a note of warning as to the indiscriminate use of salicylates in diseases of the liver, particularly those associated with jaundice.

The subject of the so-called lipotropic agents, particularly choline and methionine, is still controversial. Perhaps at least part of the controversy surrounding the use of choline and its allies might be solved, as Best [22] has suggested, if lesions due to choline deficiency were distinguished from those due to other aspects of deranged liver function. If one agrees that the fatty liver is the primary evidence of choline deficiency, it should respond to appropriate therapy. So far as fatty changes occur in cirrhosis of the liver, particularly in the early stages of the process, the experimental evidence of Best and the suggestive clinical results of Morrison,[23] Steigmann,[24] Beams,[25] Beams and Endicott,[26] and Russakoff and Blumberg [27] gave support to the effectiveness of choline and methionine. In spite of the results reported by Stevens and associates,[28] it remains a crucial question whether these agents are more effective than a diet of high protein and vitamin content. One well-controlled study [29] imputed to choline and methionine a definite protein anabolic effect beyond that produced by diets adequate in protein and vitamins in patients with chronic liver damage. The authors specifically pointed to an action of choline and methionine besides the lipotropic action. Fat accumulations were not present in biopsy specimens. Other authors have denied any protein anabolic action of choline and methionine. In a recent study of some interest Cornatzer and Cayer [30] advocated that patients be selected for choline therapy on the basis of an increase of phospholipid synthesis in response to a single large (10 gm.) dose of choline. Their conclusion was based on the finding (with radioactive phosphorus techniques) that in normal patients such medication does not alter phospholipid synthesis, while in cirrhotic subjects a significant increase can be demonstrated. In summary, choline and methionine seem to be of value when fatty infiltration exists as shown by biopsy or by clinical hepatic enlargement. These agents are of possible value in chronic liver disease because of a protein anabolic effect. However, this action is controversial, and even a lipotropic action beyond that found in a high protein diet has yet to be demonstrated beyond doubt. The best indication for the use of choline is a doubt as to the adequacy of protein intake in the presence of a fatty liver. Harmful effects of both cystine and methionine given in large doses have been reported,[29] and caution in their use is therefore advisable.

CIRRHOSIS ASSOCIATED WITH OTHER DISEASE

Fatty metamorphosis of the liver, associated with diabetes mellitus, has been treated with lipocaic. Evidence supporting this treatment is to be found in the observation of Fisher [31] and Allan and co-workers [32] that depancreatized dogs treated with sufficient insulin to control the diabetic symptoms show signs of hepatic insufficiency, become insulin sensitive and die in a state of hypoglycemia within one to eight months. Postmortem studies revealed a marked fatty infiltration and degeneration of the liver as the principal or sole abnormality. By the addition of raw pancreas to the diet, Allan and his co-workers were able to prevent such hepatic changes and to permit survival for a long time. Dragstedt, Van Prohaska, and Harms [33] prepared a fat-free alcoholic extract of beef pancreas (lipocaic) that, when fed to depancreatized dogs, proved as effective as raw pancreas, lecithin, or choline. They expressed the belief that it is a "fat-metabolizing hormone" concerned in the normal transport and utilization of fat. Dragstedt [34] administered lipocaic to patients manifesting diabetes mellitus in association with heptomegaly and noted a decrease in the size of the liver. Similar observations were made by Grayzel and Radwin [35] in three cases of juvenile diabetes mellitus treated with a pancreatic extract prepared according to the method of Dragstedt and his associates. Snell and Comfort [36] also reported a rapid decrease in the size of the liver and an improvement in the hepatic function during the first 12 days of lipocaic therapy. Rosenberg [37] observed recovery after treatment with lipocaic of a patient with fatty metamorphosis of the liver associated with diabetes mellitus. Biopsies were made before and after treatment, and results were correlated with those of liver function and dextrose tolerance tests, cholesterol determinations, and clinical course. The changes observed represented the clinical counterpart of the findings noted in experimental animals.

Crude aqueous liver extract, in addition to its lipotropic activity, seems to aid in detoxication and is definitely a part of the therapeutic regimen in cases of severe liver diseases. Its activity depends partially on its content of xanthine and partially on other, unknown, factors.

Another method of administering liver extract is the intravenous use of the water-soluble preparation. This has been advocated [13] for anorexic patients, in whom it has been claimed that it enhances the appetite and thus improves caloric intake. Ralli and associates [38] have reported a prominent increase in the five year survival rate of a group of patients treated by intravenous administration of 45 to 120 cc. of liver extract weekly, compared with a control group treated by diet, vitamin supplements, and liver extract intramuscularly.

Choline therapy in doses of 3 to 4 gm. per day may be of value when evidence of fatty infiltration can be demonstrated by biopsy of the liver or is seen clinically as enlargement of the liver.

An important symptom in liver disease that requires special attention is anemia, due in part to failure of absorption of iron because of the formation of iron soaps. Administration of iron salts, therefore, may not be successful in correcting hypochromic anemia caused by liver disease until the normal bile flow is restored. The macrocytic anemia seen with the severer liver damage is treated parenterally with liver extract but does not respond as well to this therapy as does pernicious anemia until the process causing the liver damage becomes quiescent.

Calcium deficiency is another defect in mineral metabolism associated with liver disease. This deficiency is caused by the lack of bile, resulting in impaired absorption of calcium and of vitamin D, and possibly by an excessive requirement for calcium when hepatotoxins are present. Calcium diminishes the hepatotoxic action of carbon tetrachloride and of certain other toxins. However, the experimental evidence for calcium therapy is controversial; that it can do no harm is evident; one may use it because occasionally a patient may be benefited.

For some time we have advocated the use of potassium iodide in cases of jaundice, because it seemed that the bilirubinemia disappears more rapidly after the use of this salt in doses of 10 to 20 grains (0.65 to 1.3 gm.) three times a day. If there is a syphilitic factor, the indication for the use of iodides is definite. The availability of penicillin for the treatment of visceral syphilis would naturally obviate treatment with arsenical or other heavy metal preparations in the presence of liver disease.

CORTICOTROPIN AND CORTISONE THERAPY

As in virtually all diseases in which definitive therapy is lacking, the effects of corticotropin (ACTH) therapy and cortisone therapy on cirrhosis of the liver have been explored. Aterman [39] has reported that hepatic fibrosis produced in experimental animals by administration of carbon tetrachloride has been inhibited by cortisone simultaneously administered. A preliminary report of the use of cortisone in one case of biliary cirrhosis and one of serum hepatitis revealed no significant effect.[40] In a series of seven patients treated with corticotropin in daily doses of 50 to 100 mg. 6 to 28 days, no definite improvement was noted. There was suggestive improvement in albumin synthesis and a fall of bilirubin level in four young women with cirrhosis of unknown origin. Patients with Laennec's cirrhosis showed no improvement.[41] Serious complications occurred during corticotropin therapy. In three patients portal thrombosis developed, and in one a fatal esophageal hemorrhage occurred.[42] On the basis of this preliminary work, the use of corticotropin seems to be contraindicated in cases of advanced liver cirrhosis.

RETENTION OF FLUID AND SALT

The crucial factors leading to retention of fluid with ascites and edema in hepatic cirrhosis are not known. However, it seems that among the interrelated factors are portal hypertension, increased sodium retention, decreased colloid osmotic pressure of the blood, and a possible increase in antidiuretic substance. Certainly reasonable doubt has been cast on the Starling principle that the volume of the extracellular extravascular fluid is dependent entirely on a balance between capillary hydrostatic filtration pressure and the colloid osmotic pressure of the blood.[43]

Although this factor plays a role, other factors also are involved. In patients with cirrhosis of the liver, accumulation of fluid and diuresis have occurred without alteration of the total protein and albumin levels.[44] Proof is lacking that albumin influences any aspect of cirrhosis other than fluid balance. Salt-poor human albumin has received extensive clinical trial for control of ascites and edema over the past few years.[45] Janeway and his associates [46] were the first to administer salt-poor albumin intravenously in the treatment of cirrhosis. Sufficient experience has accumulated in the literature to permit certain inferences at this time as to what may be expected from intravenous administration of salt-poor human albumin. The response in apparently similar cases is variable. In most series immediate and effective diuresis with loss of ascites and edema occurred in some cases. In others, response was slow over a period of weeks or months. In still others there was no benefit whatever. One group of workers [47] showed by electrophoretic studies a pronounced and immediate rise in albumin levels and a fall in globulin. After cessation of therapy this improvement gradually declined. In some reported instances,[48] elevations in serum albumin persisted for as long as three months after treatment. Maintenance doses of 20 to 100 gm. of salt-poor human albumin per week have been advocated.[43] The usual therapeutic dose has been 100 cc. of a 25% solution given intravenously in about 45 minutes one or two times a day. The duration of therapy depends on the response.

There is evidence [45] that a considerable part of administered serum albumin enters the ascitic fluid, establishing an equilibrium between the vascular and ascitic compartments at a higher albumin level. Albumin has been found more effective when the disease is not so advanced as to require paracentesis. However, when ascitic fluid exists in large amounts, it is economical of administered albumin to employ paracentesis prior to giving the intravenous medication. It is advisable also to restrict salt intake moderately to decrease the rate of ascites formation and thereby further conserve the serum albumin.[48] Salt-poor albumin has been most effective in treatment of the nutritional type of cirrhosis.[49] There are certain serious complications of intravenous albumin therapy (Post,[45] Watson and Greenberg,[50] Havens and Bluemle,[51] and Faloon and associates [49]). These include febrile reactions, hemorrhage from esophageal varices, epistaxis, formation of pleural effusion, and acute pulmonary edema. All these complications have been frequent enough to command caution, particularly in patients with known heart disease who have had previous episodes of hematemesis. Intravenous use of salt-poor human albumin is no substitute for nutritional and other recognized therapeutic measures for cirrhosis of the liver. However, when judiciously used in conjunction with other treatment, it may prove to be a valuable adjuvant in the therapy of decompensated hepatic cirrhosis with ascites and edema. In selected instances it may be of crucial benefit in enabling a patient to survive a critical period. Patients with cirrhosis of the liver and ascites with normal salt intake excrete minimal amounts of sodium in the urine in the order of 1 mEq. per day (Faloon and associates,[52] Farnsworth,[53] and Goodyer and co-workers [54]). Such patients may have decreased sodium content in the sweat and saliva also, indicating a possible adrenal mechanism in the sodium retention of cirrhosis with ascites (Bongiovanni and Eisenmenger [42] and Eisenmenger and associates [55]). Salt loads given to cirrhotic persons with a normal glomerular filtration rate and renal blood flow revealed impairment of excretion, suggesting a possible defect in the renal tubular mechanism.[54] However, the actual mechanism of the salt retention in cases of cirrhosis is not known at this time.

In view of the marked salt retention accompanying cirrhosis with ascites and edema, salt restriction techniques have been widely used in recent years. Salt deprivation has been attempted by dietary deletion and the use of sodium ion exchange resins. Layne and Schemm,[56] Kark and associates,[57] Lowe and Overy,[58] and Eisenmenger and associates [59] have claimed diuresis and a decrease in the rate of ascites formation as a result of diets low in sodium.

Such diets must be individualized for each patient. Kark and associates [60] recommended beginning with an intake of 25 mEq. of sodium per day and reducing or increasing this amount according to the response. A sodium intake that leads to diuresis in one patient may be ineffective in another.

Patients on prolonged salt restriction diets must be watched carefully for signs of the so-called low salt syndrome (Schroeder,[61] and Janeway and associates [46]). This is manifested clinically by weakness, lassitude, muscle cramps, nausea, and a drop in pulse pressure. If it is allowed to progress, the urea nitrogen level of the serum rises, and renal failure and death may occur. Serum electrolyte studies should be used to warn of a fall in the serum sodium level and changes in the carbon-dioxide-combining power. The danger of this syndrome is particularly marked in patients with renal disease.[57] However, it may occur in cirrhotic patients with normal renal function.

Paracentesis entails less risk and may be resorted to frequently without greatly interfering with the general condition of the patient. Care must be taken not to withdraw too much fluid at one time or to withdraw it too quickly. Further, peritoneal shock should be prevented by carefully anesthetizing the peritoneum. Hypodermic solutions of atropine and epinephrine should be close at hand in case the patient suffers from the effects of peritoneal shock.

In patients whose salt intake has been restricted, abdominal paracentesis increases the danger of the salt depletion syndrome, presumably owing to the large loss of sodium in the ascitic contents. This sequence has been reported in two cases recently.[62] When a salt depletion syndrome occurs, the most effective treatment in our experience has been the intravenous administration of 5% solution of sodium chloride in an amount calculated to replace the specific sodium deficit in the extracellular compartment. This amount can be easily determined from the body weight and the serum sodium level.

In the enthusiasm for sodium restriction it is important not to compromise the therapeutic, caloric, and protein contents of the diet. Although this is difficult to guard against even when low sodium, high protein foods are used, diets maintaining proper balance can be prepared.[48] To further this end and to achieve a more palatable type of effective low sodium diet, ion exchange resins have been introduced in the treatment of cirrhosis (Goodyer and associates,[54] and McChesney and co-workers [63]). Dock has emphasized the value of cation exchange resins in the control of edematous states. Erwin and associates,[64] Martz and co-workers,[65] and Hay and Wood [66] have found that effective diuresis occurred and subsequent fluid retention was retarded in cirrhotic patients by the use of cation exchange resins. They recommended 45 to 60 gm. of resin daily in doses of 15 gm., so that the diet can contain 1 to 3 gm. of sodium per day. The resin used should be potassium, saturated so that there is no potassium deprivation, which can be a serious complication of cation exchange therapy. Ammonium chloride should not be given when an ammonium cation exchange resin is used, since this would increase the tendency to acidosis.[66] There are, however, limitations to the clinical utility of cation exchange resins, as has been recently stressed by Greenman and co-workers.[67] The in vivo efficiency of these resins is usually 1 mEq. of sodium per gram of resin. Since ordinary diets provide 100 to 300 mEq. of sodium daily, these agents, in the amounts recommended, 60 gm. per day, are obviously insufficient. Furthermore, when the dietary sodium is withheld, the efficiency of the resins decreases markedly. Both hydrogen and ammonium resins produce acidosis in addition to the deviation of sodium to stools, which can be considered a beneficial effect except in subjects with impaired renal function. Resins must be used cautiously in the presence of renal disease. As with any form of salt restriction, the danger of a low salt syndrome is present. The use of cation exchange resins has a limited place in certain instances of hepatic cirrhosis as an alternative to other methods of salt deprivation.

Mercurial diuretics may be used in treatment of liver disease. However, their effectiveness in severe chronic disease tends to be decreased after repeated use. This fact calls into play the other means of diuresis discussed earlier. When mercurial preparations are used in conjunction with salt depletion techniques, manifestations of the salt depletion syndrome must be watched for.

PORTAL HYPERTENSION

In addition to the many problems in the edema and ascites of liver disease just described, there is the mechanical problem of intrahepatic vascular obstruction and consequent portal hypertension. The problem of portal hypertension was classically delineated by Whipple [68] in 1945 and has recently been reviewed in an excellent article by Welch.[69] In 1945 Blakemore and Lord [70] outlined a feasible procedure for surgical treatment of portal hypertension. Since that time Rousselot,[71] Blakemore,[72] Linton,[73] and Gerbode and Holman [74] described the results following direct portacaval and splenorenal anastomosis. Results are better in patients with extrahepatic portal obstructions and in young persons whose liver damage is slight. In instances of cirrhosis, Blakemore [75] has stated that, while no one test of liver function is a valid criterion for the determination of a reasonable operative risk, the operation is hazardous when the serum albumin level is below 3 gm. per 100 cc. or when the sulfobromophthalein retention exceeds 35%. While cirrhosis may be the commonest cause of portal hypertension, there is no necessary correlation between the severity of the cirrhosis and the degree of portal hypertension or its manifestations. Hepatocellular dysfunction may be relatively slight in the face of prominent portal hypertension with its sequelae, hypersplenism, and hemorrhage from engorged esophageal veins. It is important to realize that these two complications of portal hypertension are the chief aspects in which improvement can be expected from operative intervention. Ascites is likely to be decreased, while liver function will not be improved. Sufficient time has not yet elapsed for definitive evaluation of the results of the operation. However, the portal decompressing procedures appear to be helpful in carefully selected cases in which manifestations of portal hypertension are marked in proportion to hepatocellular damage.

PHYSICAL ACTIVITY

Bed rest for patients with active liver disease is a cardinal therapeutic principle. It has recently received certain experimental support. Studies by Bradley [76] and Meyers [77] by venous catheterization and sulfobromophthalein clearance techniques have shown that the hepatic blood flow in cirrhotic patients is decreased or normal. Flow is decreased by standing [76] or by passive tilting of the patient to the upright position.[78] This work may offer theoretical confirmation of the clinical impression that early mobilization of patients convalescing from liver disease is detrimental. It is known that liver cells are particularly sensitive to oxygen deprivation. Decreased partial pressure of oxygen, as in high altitudes or in the presence of anemia, may cause fatty changes.[22a] Meyers [77] has shown that in the enlarged liver the hepatic arteriovenous oxygen difference varies inversely with the flow under fasting and resting conditions to give normal splanchnic oxygen consumption. Sometimes, when the liver is small and cirrhotic, the oxygen difference is not great enough to compensate for the markedly decreased hepatic blood flow, and oxygen consumption, therefore, is below normal.[77] It may be inferred that activity would further embarrass such a patient by further reducing the already impaired hepatic blood flow. One might speculate as to the value of increasing the hemoglobin level or perhaps administering oxygen to such a patient.

DIETARY REGULATION

Dietary regulation is an essential part of the treatment of liver disease. An early return of normal eating habits is one of the most important factors in the improvement of liver function after the liver has been damaged. The food should be palatable and served in an appetizing fashion, and feedings should be given at frequent intervals. Nothing is so depressing to the patient as an ill-arranged tray. He has little desire for food, and one should use every psychological effort to encourage an added caloric intake. Ravdin and his associates [79] stated that a caloric intake in excess of basal requirements is most important in protecting the liver and aiding repair of liver damage. A high caloric, highly nutritious diet is the most important single aspect of the long-term management of chronic liver disease. Such a diet should contain at least 3,500 calories. The following diet is suggested:

Foods Allowed

Cereals:	One serving daily of any type cooked or prepared cereal, i. e., enriched cream of wheat,® farina,® oatmeal, and cornflakes. Avoid those containing large amounts of bran.
Bread:	Enriched white, cracked or honey wheat, whole wheat, or rye bread, melba toast, holland rusk, zweibach, or white crackers.
Cereal substitutes:	Rice, noodles, vermicelli, spaghetti, hominy, and macaroni; all to be plainly prepared without the addition of highly seasoned or rich sauces.
Soups:	Lean broths and vegetable milk soups.
Meat:	Two servings daily of very lean meat, i. e., lamb, veal, beef, liver, chicken, or turkey; to be broiled, boiled or roasted without additional fat. Remove all visible fat. No gravies or sauces, pork, or pork products, including bacon. Four-ounce cooked portion equals *minimum serving*; may use more.
Fish:	Lean fresh water fish, i. e., perch, pike, trout, bass, or whitefish; to be broiled, boiled, poached, or baked without additional fat. None to be fried.
Cheese:	Cottage, cream, mild American or Wisconsin brick cheese. No other kinds are to be used.
Eggs:	One or two eggs daily; may be either soft boiled, poached, shirred, or scrambled over water. None to be fried.
Vegetables:	At least three or four servings daily of any type cooked or raw vegetable with the exception of raw onions, radishes, cucumbers, or sauerkraut; all to be prepared without additional fat.
Potatoes:	Mashed, baked, or boiled potatoes. None to be fried.
Fruits:	At least three or four servings daily of any type canned, stewed, or fresh fruits. One fruit should be citrus, i. e., orange or grapefruit or the juice of either.
Desserts:	Fruit as above. Simple puddings, custard, jello,® simple cakes, and wafers.
Beverages:	Three or four glasses of milk daily; may use more. Coffee in moderation, tea, decaffeinized or cereal beverages (sanka,® kaffee hag,® postum®), fruit juices, and water.
Butter:	Three teaspoons or pats per day.
Condiments:	None to be used except salt.
Sweets:	Sugar, jelly, honey, and hard candies as desired.

Foods to Avoid

Fried foods, oils, salad dressings, nuts, and peanut butter; smoked, spiced, or tinned meats or fish; pork and pork products; gravies and sauces; pies, pastries, and chocolate; alcoholic and carbonated beverages; spices and condiments.

Avoid Tobacco

*Sample Menu for Hepatic Regimen**

Breakfast	
Fruit (any type)	1 serving or more
Cereal (any type except bran)	1 serving (½ cup)
Milk	1 glass, 6 oz.
Eggs (prepared any way except fried)	1 or 2
Toast	1 slice or more
Butter	1 teaspoon or pat
Jelly	1 tablespoon
Coffee	
Cream and sugar	
10 a. m.	
High Protein Beverage†	⅓ part of recipe
Luncheon	
Meat, fish, poultry, cheese or eggs	At least a 4-oz. cooked portion of meat, fish, or poultry, or 2 eggs, or large serving of cottage, cream, or American cheese.
Vegetable	1 serving (½ cup)
Salad, vegetable—no dressing	1 average salad
Bread	1 slice or more
Butter	1 teaspoon or pat
Jelly	1 tablespoon
Fruit (any type)	1 serving or more
Milk	1 glass, 6 oz.
3 p. m.	
High Protein Beverage	⅓ part of recipe
Dinner	
Meat, fish or poultry	At least a 4-oz. cooked portion
Potatoes (any way except fried)	1 average serving or more
Vegetable	1 serving (½ cup)
Salad, fruit or vegetable—no dressing	1 average salad
Bread	1 slice or more
Butter	1 teaspoon or pat
Jelly	1 tablespoon
Fruit (any type)	1 serving or more
Milk	1 glass, 6 oz.
Bedtime	
High Protein Beverage	⅓ part of recipe
Crackers	2 double or more

*Hepatic diet with starlac® feeding has the following approximate content: protein, 170; fat, 172; and carbohydrate, 357 (total calories, 3,656). Hepatic diet with protenum® feeding has the following approximate content: protein, 170; fat, 173; and carbohydrate, 353 (total calories, 3,649). Hepatic diet with protelac® feeding has the following approximate content: protein, 168; fat, 172; and carbohydrate, 329 (total calories, 3,536).

†High protein beverage: starlac® (product of the Borden Company), 6 tablespoons; milk, 1½ cups; cream (coffee), 1½ cups; sugar, 7 teaspoons; eggs, 3; and vanilla, 3 teaspoons. Beat eggs, sugar, vanilla, milk, and cream until well blended. Sprinkle starlac® over mixture. Beat until well mixed.

Recent studies by Chalmers and Davidson,[14] Jones and Volweiler,[80] and Patek [81] indicate that an optimal distribution of the caloric content includes protein elements to the amount of 1.5 to 2 gm. per kilogram of body weight per day. This gives some margin of safety over the amount usually allowed, which has been shown to support liver repair (Kimball and Chapple [82] and Kark and associates [57]). Protein is the most effective dietary agent in protecting the liver from damage and in promoting its repair.[81] Indeed, it has been argued [83] that, when dietary protein intake is adequate, little will be gained by supplemental amino acid or lipotropic therapy. This assertion will be alluded to later.

The carbohydrate fraction of the diet should also be as high as possible to supply the bulk of the caloric needs and to exert a protein-sparing influence.

The indictment of fat in the diet of patients with liver disease is not well supported in recent clinical literature (Chalmers and Davidson,[14] Jones and Volwiler,[80] and Patek [81]). While it is true that liver damage in the experimental animal has been produced by diets high in fat,[15] these diets were also deficient in protein elements. When protein intake is adequate, it is questionable whether a diet high in fat leads to liver damage.[84] Convincing clinical studies have shown that a diet of 3,500 calories that provides adequate protein may contain up to 175 gm. of fat and still be effective in the management of decompensated liver cirrhosis. The pertinent point is that fat should not be restricted below the level needed to allow a palatable diet and thus the desired high caloric intake. In general, fats of vegetable or dairy origin are better tolerated than those from meat sources.

HEPATIC FAILURE, COMA, AND JAUNDICE

The patient with severe liver disease is in danger of hepatic failure and coma. Here intensive intravenous use of dextrose, salt-poor human albumin, protein hydrolysates, liver extract, blood transfusions, and plasma in judicious combinations may be lifesaving. Oxygen therapy is necessary with these measures, to combat the hepatotoxic effects of anoxemia. Oxygen is best given in an oxygen tent. The use of nasal catheters is contraindicated, because pressure necrosis from the rubber may result from a possible uremic state, which can form a part of the clinical picture. We have seen severe pharyngitis and erosions of the nose with subsequent bleeding follow the use of rubber tubes in the nose and mouth.

Of the complications that occur in cases of jaundice, acute hepatic insufficiency and hemorrhage are the most important. Jaundice in patients with cirrhosis of the liver may appear as a transitory, almost unnoticed, episode; yet it should be considered a serious sign. Bed rest is imperative and hospitalization is required in cirrhotic patients with jaundice.[65] Finally, the whole therapeutic approach to this problem should be controlled by repeated laboratory investigations, which would indicate the improvement, if any, that has taken place in the liver as the result of the treatment outlined. We cannot urge too strongly the need for a conservative and scientific approach to the treatment of jaundice. One should not be too eager to operate just because of the presence of bilirubinemia, and one should not delay operation in a patient with a definite surgical type of jaundice. There is no substitute for keen clinical judgment with respect to this disease.

After the jaundice has disappeared and definite clinical improvement has been noted in patients disabled by liver disease, ambulatory management should be continued for many months, until results of repeated liver function tests indicate restoration to normal. When this has been brought about, the patient should be warned against exposing himself to the factors that brought about the original condition. In spite of the recent emphasis on dietary deficiency in the genesis of cirrhosis,[9] there is no change in the traditional interdiction of the use of alcohol in the management of the disease.

SUMMARY

The incidence of cirrhosis is increasing. The use of sulfonamides and other hepatotoxic agents and the occurrence of serum homologous hepatitis and epidemic hepatitis of World War II may be partly responsible. Recognition of the cause of liver disease is necessary for effective therapy.

Patients should be at bed rest and preferably hospitalized. The decrease in hepatic blood flow of cirrhotic patients while standing offers theoretical confirmation of the clinical impression that early mobilization of these patients is detrimental. Bed rest is imperative during jaundice in cirrhotic patients. A high caloric, high protein diet is important in the long-term management of chronic liver disease. A diet of 3,500 calories is suggested.

Intravenous infusion of carbohydrates is desirable, in order to hasten the regeneration of liver parenchyma. In patients with atrophic cirrhosis and ascites, the use of hypertonic dextrose solution induces diuresis. Infusions of amino acids may be necessary in order to maintain normal nitrogen balance in patients unable to eat a full diet. Administration of whole blood or plasma may be beneficial when there is marked hypoproteinemia, anemia, or a hemorrhagic tendency.

While it has become almost routine to include supplementary administration of vitamin B complex to cirrhotic patients, definite evidence of deficiency in cirrhotics, per se, has not been demonstrated. Vitamin K should be administered regardless of prothrombin or bleeding time. Vitamin A and D in doses of 25,000 to 50,000 units, given one or two times daily, are of definite value. Choline and methionine seem to be of value when fatty infiltration of the liver exists, although this is controversial, and their action other than that found in a high protein diet has yet to be demonstrated beyond doubt. The use of crude aqueous liver extract seems to aid in detoxication and should be a part of the therapeutic regimen for patients with severe liver disease. Intravenous use of a water-soluble liver extract has been recommended for anorexic patients. We advocate use of potassium iodide in cases of jaundice. If syphilis is a causative factor, the indication for the iodide is definite.

Although the crucial factors leading to retention of fluid with ascites and edema in cirrhotic patients are not known, the employment of salt restriction and diuretics as well as salt-poor albumin is suggested; however, such use is not without certain dangers.

The value of the administration of cation exchange resins is limited, since the amount recommended, 60 gm. per day, is insufficient and larger doses are impractical. Patients on prolonged salt restriction must be watched carefully for signs of the low salt syndrome. Abdominal paracentesis increases this danger when preceded by severe salt restriction and the use of diuretics. On the basis of preliminary work, corticotropin (ACTH) therapy and cortisone therapy seem to be contraindicated in treatment of severe liver cirrhosis. Surgical treatment of portal hypertension has its best indications in patients with extrahepatic portal obstruction and is hazardous when there is a marked liver insufficiency.

Although the laboratory studies have become very important tools for the diagnosis and management of liver disease, they are more valuable when interpreted in the light of keen clinical observation.

104 S. Michigan Ave.

Workmen's Compensation Laws.—The first real contribution to industrial diseases of the skin was made by Ramazzini in the year 1700. His unexcelled description of the dermatoses of bakers, farmers, silk-workers, washer-women and others has won him the appellation of "father of industrial hygiene."

Appreciation of the importance of industrial diseases has been recent in this country, and compensation laws were not enacted until well into the 20th century. The Federal Government made the first provision for compensation of injured employees in 1908. In 1911 the state of Wisconsin established the first Workmen's Compensation Law. Since then all states have enacted legislation to deal with industrial injuries. Frank C. Combes, M.D., Dermatology Problems in Establishment of Workmen's Compensation Claims, *Compensation Medicine,* March-May, 1952.

TECHNIQUES AND INTERPRETATION OF ROUTINE BLOOD CULTURES

OBSERVATIONS IN FIVE THOUSAND CONSECUTIVE PATIENTS

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Blood cultures are often essential in the diagnosis and prognosis of disease. Frequently, however, the results obtained are difficult to interpret and correlate with the clinical state of the patient. Limitations and confusions resulting may be attributable to (1) contamination due to faulty technique in the drawing of blood or in laboratory procedures, (2) preconceived ideas of bacterial "nonpathogenicity," and (3) variations in techniques of blood culture.

Bacteria can be recovered from the blood stream during physiological states and under all pathological conditions from minimal local infections to septicemias. The significance of an organism recovered from the blood stream lies in its meeting certain basic criteria. This study was made in an attempt to correlate the clinical status and the laboratory findings in patients who have had blood drawn for cultures. Certain criteria to facilitate this correlation were established.

Arnold[1] experimentally produced frank bacteremia in dogs by introducing a highly acid chyme into the small intestine from the stomach. This resulted in increased permeability of the wall of the small intestine to bacteria. Arnold further demonstrated that certain physiological alterations in the body, such as adaptations to extremes of temperature, whether artificially induced or from disease, may result in bacteremia. Cirrincione and Francona,[2] also working on dogs, were able to confirm Arnold's findings.

Reith and Squier[3] reported a study of 293 subjects, of whom 278 were male, who had blood drawn for culture at random intervals while they were performing their usual daily occupations. All were subjected to a thorough physical examination, including dental x-rays. Bacterial growth resulted in 113 of the 293 cases. In 48 the bacteria were considered frank contaminants, while in 65 they were considered as being in the blood at the time of culture. An increase in positive blood cultures was noted during the months when upper respiratory tract infections were common, suggesting that they may, in part, be responsible for the rise.

Kulka[4] obtained positive blood cultures from apparently healthy women during menstruation, and Kobak,[5] using umbilical vessel blood cultures, confirmed the physiological entry of bacteria into the blood stream. The presence of bacteria in the blood during apparent health has resulted in studies on possible foci and primary sites from which the organisms invade the blood. Horder[6] as early as 1908 observed the "undoubted fact that the infective agent in most cases of subacute bacterial endocarditis was derived from the mouth." Libman[7] in 1918 reported that alpha streptococci were "constantly invading the blood from foci in the upper respiratory tract and oral cavities, even during periods of inactivity of the foci." Lewis and Grant[8] concluded that invasion of the blood stream is a common event in patients with diseased heart valves. They further state that such invasions are encountered in the great majority of cases without mishap and that an "almost physiological entry" of organisms into the blood stream of the average person constantly occurs. King,[9] in observations on 2,233 consecutive blood cultures, felt that "casual bacteremias" occurred rarely but, nevertheless, did occur.

Minimal trauma as a cause of bacteremia was demonstrated by Murray and Moosnick,[10] who obtained positive blood cultures from patients with chronic dental disease after they had chewed paraffin for one-half hour. Round and Kirkpatrick,[11] Burket and Burn,[12] and Okell and Elliott[13] all demonstrated bacteremia following intraoral manipulations. Hirsh[14] and his associates, using control studies, demonstrated the prophylactic value of penicillin in controlling such bacteremias. McEntegart and Porterfield[15] concluded that bacteremia following oral manipulations was related to the degree of trauma rather than to the intensity of oral sepsis.

Trauma, in the form of operative procedures on or about foci of infection, has repeatedly been observed to be followed by bacteremia. Seifert[16] reported that, 10 minutes after drainage of a variety of abscesses, positive blood cultures with organisms identical to those in the

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abscesses were obtained in 45% of his cases. Richards[17] demonstrated bacteremia following vigorous massage of foci of infection in 15% of 376 cases. Bacteremia, as one of the factors in radiation sickness, has been described by Warren[18] and Lawrence and Tennant.[19] Miller[20] and his associates have experimentally corroborated this observation.

Laboratory technique for blood cultures was different in every report reviewed. Extremes vary from single inoculation of one laboratory medium to fractionation of blood samples with inoculation of multiple mediums under both aerobic and anaerobic conditions. Skin preparation varied from simple alcohol swabbing to surgical preparation of the skin.

Variations in techniques, as well as differences in evaluation and clinical correlation of results of blood cultures, made an analysis of common factors in all references cited difficult. These differences, associated with the failure on the part of many workers to recognize the presence of bacteremia during various physiological conditions, emphasized the need for uniform methods of technique and interpretation of blood cultures.

Organisms recovered from blood cultures were used as the foundation for determining the correlation of bacteremia and the clinical state of the patient in this study. Bacteria whose pathogenic status is controversial, e. g., coagulase-negative Staphylococcus albus, diphtheroids, and Bacteroides, were given special study. The criteria established for interrelation of blood cultures reported here include (1) a consistent technique of sample drawing, (2) uniform methods of bacterial identification, (3) repeated cultures in series, and (4) compensation for complicating effects of chemotherapy and antibiotic therapy.

METHOD AND MATERIAL

Blood cultures of 5,000 consecutive patients, made at the Los Angeles County Hospital, Acute Unit, from Jan. 1, 1949, to Jan. 13, 1950, were reviewed. All blood cultures were routine. Clinical charts of all patients having positive blood cultures were reviewed, and bacteriologic-clinical relationships were analyzed and reviewed.

Five to 10 cc. of blood was obtained from the cubital vein through alcohol-prepared skin. The blood was inoculated into 6-oz. (170-gm.) medicine bottles through rubber-lined screw caps with central perforations. Mediums consisted of 100 cc. of DIFCO tryptose and phosphate broth with dextrose. A like amount of bile broth with dextrose was used for Salmonella, and 20 cc. of tryptose citrate broth was used for Brucella. To all mediums 1% paraaminobenzoic acid was added at the time of preparation as advised by Woods.[21] Penicillinase was added to the culture mediums from patients being treated with penicillin. All specimens were incubated at 37 C. Routine cultures were inspected daily the first week and semiweekly the second week. In the absence of growth, cultures were discarded at the end of the second week. Cultures for Brucella were placed in a 10% carbon dioxide environment after 48 hours. Every four to five days cultures were examined by smear. Subcultures were made the same time to tryptose agar slants, which were also incubated under carbon dioxide conditions. In the absence of growth in four weeks they were discarded as negative.

Relationship of Organisms Cultured to Concomitant Clinical States in 665 Patients

Organism	Bacterial Heart Disease	Pulmonary Tract Disease	Post Traumatic Sepsis	Upper Respiratory Tract Disease	Septicemia, Unknown Foci	Infantile Sepsis	Skin, Bone, Joint Disease	Genitourinary Tract Disease	Gastrointestinal Tract Disease	Group Totals A	Group Totals B
Group A *											
Staphylococcus aureus, coagulase positive	6	3	..	6	4	6	4	14	2	45	..
Beta streptococci	1	2	4	1	9	..	..	..	..	17	..
Nonhemolytic microaerophilic streptococcus	2	..	1	..	..	..	..	..	..	3	..
Proteus morgani	..	..	..	..	..	2	1	..	..	3	..
Proteus vulgaris	..	..	1	..	..	..	1	2	1	5	..
Salmonella	..	..	..	..	..	..	..	..	6	6	..
Diplococcus pneumoniae	..	110	..	..	..	..	..	..	..	110	..
Hemophilus influenzae §	..	..	..	..	..	..	..	..	..	6	..
Neisseria intracellularis §	..	..	..	..	..	..	..	..	..	7	..
Group B †											
Bacteroides	..	..	..	..	..	..	..	..	..	..	1
Clostridium welchii	..	..	..	..	..	..	..	..	..	..	1
Bacillus subtilis	..	..	..	..	..	..	..	..	..	..	17
Group A-B ‡											
Alpha streptococci	19	2	3	..	2	1	..	..	..	27	3
Nonhemolytic streptococci	9	..	2	2	..	1	..	..	..	14	4
Staphylococcus albus, coagulase negative	..	..	..	..	..	9	..	..	..	9	246
Diphtheroids	..	..	..	..	..	3	..	..	..	3	51
Escherichia coli	..	..	2	..	..	5	..	31	4	42	7
Pseudomonas aeruginosa	..	..	..	..	1	2	3	..	1	7	1
Aerobacter aerogenes	..	..	..	..	..	1	11	1	..	13	3
Alkaligines fecalis	..	..	..	..	..	4	2	..	..	6	2
Eberthella (nonspecific)	..	..	..	..	..	1	..	1	..	2	1
										325	340

* Positive bacterial clinical correlation. † Absence of bacterial clinical correlation. ‡ Classification dependent on clinical correlation in individual cases. § Organism produced specific associated clinical state.

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RESULTS

Bacterial growth resulted in 665, or 13%, of the 5,000 cases reviewed. Approximately half (325) the organisms recovered in this study demonstrated bacteriologic-clinical correlation. Multiple invasion of the blood was demonstrated in 45 patients. Specific pathogenicity of the remaining half (340) was either indefinite or not thoroughly established.

On the basis of their probable etiological correlation with the diagnosis of the patient, organisms were placed into one of two groups, designated A and B. Bacteria were placed in group A if (1) an accepted etiological relationship between organism and disease existed, as in the case of meningococcemia and pneumococcemia, (2) repeated blood cultures grew organisms in which clinical correlation was probable, (3) disappearance of the organism from the blood paralleled clinical recovery, (4) bacteria recovered from the blood were identical with those recovered from foci of infection, and (5) in case of death, postmortem blood cultures corroborated the antemortem cultures. They were placed in group B if (1) organisms were obtained in only one of repeated blood cultures and in addition a specific etiological relationship was lacking, (2) the organisms, though potentially pathogenic, were not definitely related to the current illness of the patient, and (3) the organisms are accepted as being ubiquitous, e. g., Bacillus subtilis.

The accompanying table lists organisms on the basis of their grouping and their relationships to the clinical states of the patients. Organisms listed as belonging to group A with established pathogenicity need not be discussed in detail, as they fulfil the criteria for inclusion in group A. Organisms showing questionable pathogenicity are responsible for existing confusion and their status is in need of clarification.

COMMENT

The results of blood culture may be definitive in establishing a clinical diagnosis and determining the prognosis in any given case. Past clinical confusion in interpretaton has been dependent on the following factors: (1) unawareness of bacteremic states during apparent health and in association with minimal trauma, (2) preconceived ideas of certain "nonpathogenic" attributes of certain organisms, (3) disregard of special considerations of pediatric bacteriology, (4) lack of correlation with associated diseases in the host, (5) lack of standard technique of blood culture, and (6) failure to compensate for antibiotic and chemotherapeutic agents.

Bacterial invasion of the blood during health, during physiological alterations in the body, and during disease has been amply observed clinically and corroborated experimentally. Organisms recovered from the blood during health and after minimal trauma as reviewed in the introduction were considered to be chiefly and usually "nonpathogenic." The ability of these bacteria to multiply in the blood stream is usually low, and their power to produce a septicemic state is limited. Their presence is transient. These "nonpathogens," however, constitute a distinct threat to patients with chronic disease such as valvular heart disease. Loewe [22] and his associates, MacNeal and Blevins,[23] and McMillan [24] have demonstrated the role of oral alpha streptococci in the production of bacterial endocarditis.

Pediatric bacteriology as applied to blood culture requires special consideration. Organisms effectively overcome by adults can be both morbid and fatal to infants in spite of treatment. All the cases of Staph. albus, coagulase-negative, in group A were in cultures from pediatric patients. Gram-negative bacilli also demonstrated an increased pathogenicity in infants and children having limited immune mechanisms. An excellent opportunity for comparing antemortem and postmortem blood cultures was made possible by the high percentage of autopsies on the pediatric service. Blood culture results could be correlated in 20 of 27 cases. Of these, coliform bacilli were present alone or as part of a multiple invasion in 16 cases in sufficient numbers to indicate blood stream invasion in life. In instances of multiple invasion, so-called nonpathogens have been associated with disease production. Kanof and Kramer [25] reported 24 cases of multiple invasion in children; organisms usually considered spurious invaders were associated etiologically with the death of two.

The role of metabolic diseases and other associated disturbances in physiological processes affect the role of the host in resistance to bacteremia. A high incidence of diabetes and azotemia was observed among patients in whom facultative pathogens produced disease. Any alterations in immune response due to these factors require consideration.

Review of the literature showed a lack of comparable blood culture techniques. No correlation between the more elaborate methods of skin preparation and a decrease in contamination could be established. The time of obtaining the blood specimens for culture bore little relation to the incidence of positive cultures. The frequency in terms of times per day of obtaining blood for culture revealed no percentage difference of positive recovery attributable to the time factor. Multiple or complex mediums showed no increase in positive blood cultures.

Recent therapeutic advances have introduced new complications into blood culture studies. The use of para-aminobenzoic acid and penicillinase when indicated is fundamental.

An analysis of the diseases associated with blood stream invasion in this study emphasizes the importance of the above considerations. Except for B. subtilis, no organism can be considered as an invariable contaminant. Clostridium welchii and Bacteroides, though placed in group B exclusively, have been shown by numerous other studies to be capable of producing septicemia. Nine organisms are listed in group A-B. The so-called nonpathogens, such as Staph. albus and diphtheroids, fulfil the criteria set up for pathogenicity and in certain cases were considered of etiological significance. All organisms exclusive of B. subtilis placed in group B were not considered as contaminants, but rather their presence was accepted as being part of the large group of blood stream invaders during periods of physiological alteration, whatever the cause. Organisms in group B most frequently were associated with an initial positive blood culture followed by repeated negative ones. The failure to grow the organisms repeatedly may be explained by the institution of treatment immediately after the initial blood culture. Low virulence associated with their rapid destruction, along with the possibility of few circulating organisms, also may have contributed to their absence from subsequent blood cultures. Bacteriologic-clinical correlation was uniformly absent in cases in which group B organisms were grown.

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[22] Loewe, L.; Plummer, N.; Niven, C. F., Jr., and Sherman, J. M.: Streptococcus s.b.e. in Subacute Bacterial Endocarditis, J. A. M. A. 130: 257-260 (Feb. 2) 1946.

[23] MacNeal, W. J., and Blevins, A.: Bacteriological Studies in Endocarditis, J. Bact. 49: 603-610 (June) 1945.

[24] McMillan, R. L.: Subacute Bacterial (Streptococcus Viridans) Endocarditis Treated with Penicillin, Am. J. Med. 1: 628-633 (Dec.) 1946.

[25] Kanof, A., and Kramer, B.: Multiple Invasion of the Blood Stream, J. Lab. & Clin. Med. 27: 173-180 (Nov.) 1941.

SUMMARY

A review of blood cultures of 5,000 consecutive patients indicated the need for and resulted in the establishment of certain criteria for blood culture study, bacteriologic interpretation, and clinical correlation. These criteria included factors affecting the organism, the host, and therapeutic regimens.

Blood for culture was obtained through alcohol-prepared skin with no special considerations as to time of day or number of samples per day. Bacterial growth resulted in 665, or 13%, of the 5,000 cases reviewed. In 50% of the cases the organisms were classified in group A, indicating positive bacterial-clinical correlation in that they fulfilled the following criteria: (1) an accepted etiological relationship between organism and disease existed, as in the case of meningococcemia and pneumococcemia; (2) repeated blood cultures resulted in growth in which clinical correlation was probable; (3) disappearance of the organism from the blood paralleled clinical recovery; (4) bacteria recovered from the blood were identical with those recovered from foci of infection; and (5) in case of death, postmortem blood culture resulted in bacterial growth identical to that obtained in antemortem cultures. In the remaining 50% of the cases the organisms were classified in group B and were considered as "nonpathogenic" invaders because they (1) were obtained in only one of repeated blood cultures during the same disease and a specific etiological relationship was lacking; (2) though potentially pathogenic, were clinically unrelated to the current illness of the patient; and (3) are accepted as being invariable external contaminants, such as Bacillus subtilis.

The following organisms fitted into both groups in accordance with their roles in individual specific clinical states and as such may be classified as having pathogenic capabilities: alpha Streptococcus viridans, indifferent Streptococcus, indifferent micro-aerophilic Streptococcus, coagulase-negative Staphylococcus albus, Escherichia coli, Aerobacter aerogenes, Proteus vulgaris, Proteus morganii, Alkaligenes fecalis, Pseudomonas aeruginosa, nonspecific Eberthella, and diphtheroids. The recovery of organisms from blood culture in cases in which clinical-bacteriologic correlation is absent does not necessarily indicate laboratory contamination, but rather the possibility of bacteremia in association with physiological states or periods of physiological alteration, following both minimal and severe trauma, as well as during septicemic diseases.

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FACTORS INFLUENCING RUPTURE OF THE HEART AFTER MYOCARDIAL INFARCTION

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and
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It is the purpose of this paper to review the factors related to the occurrence of rupture or perforation of the heart after myocardial infarction. Rupture of the heart has been recognized for over 300 years, and in recent times it has been the subject of thorough autopsy and clinical evaluation. The following salient facts have become established through previous investigations: 1. Such ruptures rarely occur in patients younger than 50 years of age.[1] 2. Patients who have unlimited activity after infarction occurs apparently have a higher incidence of rupture than those treated with "adequate" rest.[2] 3. Rupture associated with infarction almost universally occurs in the first few weeks following infarction; it rarely if ever occurs in an old scar.[3] 4. The chances of rupture occurring are three times greater in patients in whom hypertension persists after infarction than in those who have normal or subnormal blood pressures.[1a] 5. Myocardial necrosis is apparently a necessary prior condition for the occurrence of rupture.[4]

In an effort to find further information, we reviewed the records of 80 patients who died at the Los Angeles County Hospital between July, 1941, and October, 1951, of perforation of the heart with hemopericardium and cardiac tamponade. During this time there were, in addition, 11 patients who died from the effects of rupture of the interventricular septum following recent myocardial infarction. (Detailed analyses of the records of these 11 patients have not been included in this report.) Completed autopsy statistics are available at this hospital for the period July 1, 1941, to December 31, 1948, inclusive. In that period there were 13,665 autopsies; of these, 1,026 (7.58%) were performed on patients who died after recent myocardial infarction. Of this latter

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Autopsy records were made available by Dr. E. M. Butt of the Department of Pathology, Los Angeles County Hospital.

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group, 73 patients, 7.11% of those with recent infarction and 0.53% of all patients on whom autopsies were done, died of cardiac rupture, either perforation through a free wall of the heart or perforation through the interventricular septum.

RESULTS OF REVIEW

The clinical, electrocardiographic (when available), and pathological records of these 80 patients were reviewed.

Clinical Records.—In both men and women the greatest incidence of rupture occurred in the seventh and eighth decades of life (table 1). None of the patients was less than 50 years of age. The average age of the men was 69.75 years and of the women, 72 years. The sex difference observed was striking: 33 patients (41.25%) were male and 47 (58.75%) were female. Regarding race, 79 were Caucasian and 1 was Negro.

Because of past emphasis on the fact that physical activity in the postinfarction period may be related to the incidence of rupture and because of recent articles relative to early activity in patients with infarction, the time of onset of infarction was carefully estimated from the history obtained in each case. The length of time from the estimated onset to entry into the hospital and the length of life from the time of estimated onset to rupture were calculated as carefully as possible.

The length of time from estimated clinical onset to entry into the hospital was as follows: 16 (20%) patients were in the hospital and in bed within 6 hours; another 9 (11.25%) entered between 7 and 12 hours after onset; 13 (16.25%) entered between 13 and 24 hours after onset. In addition, 11 patients (13.75%) were in the hospital at bed rest at the time of onset of the infarction. The remainder entered from 1 to 21 days after onset, 9 entering on the second day and 6 on the third day. Sixty-four patients (80%), therefore, either were already in the hospital at onset of infarction or entered within 72 hours afterward. Of the group entering later than that, several had been under the care of physicians at home or in private hospitals. Sex differences were apparent in this analysis: 17 men (51.5%) were admitted within 24 hours after onset of infarction, but only 20 women (42.7%) entered during the same period.

The length of life from estimated clinical onset to time of rupture was as follows: 14 (17.5%) patients died in less than 24 hours; 10 (12.5%) died on the second day; and 12 (15%) died on the third day; thus, a total of 36 (45%) patients died within the first 3 days of infarction. Another 27 (33.75%) died between the 4th and 12th days. In 11 instances, length of life after infarction was unknown, and in 6 instances, death occurred from 19 to 30 days after onset of infarction. Sex differences were found here also: 19 men (57.6%) and only 17 women (36.2%) died within the first 3 days.

Blood pressure observations, where available, were similar to those reported in the past.[1a] In cases in which preinfarction pressures were known, they were with few exceptions abnormally high. Following infarction, the pressure dropped to normal levels in a few cases, but the majority reflected either persistent hypertension or a drop to shock pressures.

The relation of digitalis therapy, causing increased myocardial contractility, to rupture of the heart has in the past been the subject of speculation.[1d] Ten (12.5%) of the patients reviewed had been receiving maintenance doses of digitalis, and 4 others were given digitalis in the last 2 to 4 days of life because of evidence of congestive failure. Thus, there is no concrete evidence to be derived from this series that suggests a significant relationship between digitalis administration and myocardial rupture. Similarly, quinidine sulfate, which was administered terminally to only 3 patients, cannot be implicated.

Electrocardiographic Records.—Electrocardiograms made in the recent antemortem period were available for 42 of the patients and for another 5 who died in the two year period 1939 to 1941. Electrocardiograms of 26 patients were made within 24 hours of death, those of 12 within 48 hours, of 4 within 72 hours, and of 5 within 96 hours. Records of four patients were made by the standard limb and multiple unipolar lead technique; records of three others were made by the standard limb leads plus multiple CF leads, and records of the remainder were made by the standard limb leads and lead CF_4.

TABLE 1.—*Distribution of Patients by Age, Sex, and Race*

Sex	Age, Yr.				Total	Average Age	Race	
	50-59	60-69	70-79	80-89			Caucasian	Negro
Men............	4	11	15	3	33	69.75	33	0
Women........	3	16	18	10	47	72	46	1

Nothing was found in tracings made by the standard and unipolar limb leads that could be correlated in any way with the later development of cardiac rupture. At best, these leads indicated only myocardial infarction. Chest lead tracings showed the following characteristics in patients with anterior wall and posterior wall perforation: 1. Anterior wall perforation—two patients in this group had multiple unipolar precordial leads, and in each, two or more positions showed QS waves characteristic of full-thickness myocardial infarction (fig. 1). In 2 subjects with multiple CF leads, QS waves appeared in two or more leads. A single lead, CF_4, was made in 30 patients; 20 of these had only QS complexes, 6 had deep Q waves, and 4 had essentially normal waves.

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1. (*a*) Edmondson, H. A., and Hoxie, H. J.: Hypertension and Cardiac Rupture: Clinical and Pathologic Study of 72 Cases, in 13 of Which Rupture of Interventricular Septum Occurred, Am. Heart J. **24**: 719, 1942. (*b*) Friedman, S., and White, P. D.: Rupture of the Heart in Myocardial Infarction: Experience in a Large General Hospital, Ann. Int. Med. **21**: 778, 1944. (*c*) Zinn, W. J., and Cosby, R. S.: Myocardial Infarction: Statistical Analysis of 679 Autopsy-Proven Cases, Am. J. Med. **8**: 169, 1950. (*d*) Gans, R. H.: Acute Myocardial Infarction with Rupture of the Ventricle, Am. Heart J. **41**: 332, 1951. (*e*) Jetter, W. W., and White, P. D.: Rupture of the Heart in Patients in Mental Institutions, Ann. Int. Med. **21**: 783, 1944. (*f*) Segall, H. N.: Rupture of the Ventricular Myocardium: Report of 4 Cases with Comments on Pathogenesis and on Clinical Significance of Possibility of Cardiac Rupture in Prognosis of Coronary Artery Disease, Am. Heart J. **30**: 39, 1945.

2. Gans,[1d] Jetter and White.[1e]

3. (*a*) Footnote 1 *a, b, d,* and *f.* (*b*) Foster, L.: Spontaneous Rupture of the Heart: Clinical-Pathological Correlation with Presentation of 12 Cases, Quart. Bull., Northwestern Univ. M. School **20**: 209, 1946. (*c*) Krumbhaar, E. B., and Crowell, C.: Spontaneous Rupture of the Heart: Clinicopathologic Study Based on 22 Unpublished Cases and 632 from Literature, Am. J. M. Sc. **170**: 828, 1925. (*d*) Benson, R. L.; Hunter, W. C., and Manlove, C. H.: Spontaneous Rupture of the Heart: Report of 40 Cases in Portland, Oregon, Am. J. Path. **9**: 295, 1933.

4. Edmondson and Hoxie.[1a] Foster.[3b] Krumbhaar.[3c] Benson and others.[3d]

2. *Posterior wall perforation*—2 patients had unipolar precordial leads; in one there was a QS complex in lead V_6 (fig. 2), and in the other all precordial leads were normal. One patient had multiple CF leads, and a deep Q wave was found in leads CF_5 and CF_6. Of 10 patients examined with lead CF_4 only, 1 had a deep Q wave and the other 9 had normal QRS complexes.

Pathological Records.—The problem of pathological changes in the heart occurring after myocardial rupture has been extensively studied by competent pathologists.[5] In brief, the predominant changes apparently are extensive necrosis, undermining, and dissection of the myocardium. We have found no need to review the detailed histopathological condition of the hearts studied in our group, but we have attempted only to define the location of the infarct (table 2) and the location of the rupture (table 3) and then to determine if there is a predilection for any specific part of the heart to rupture.

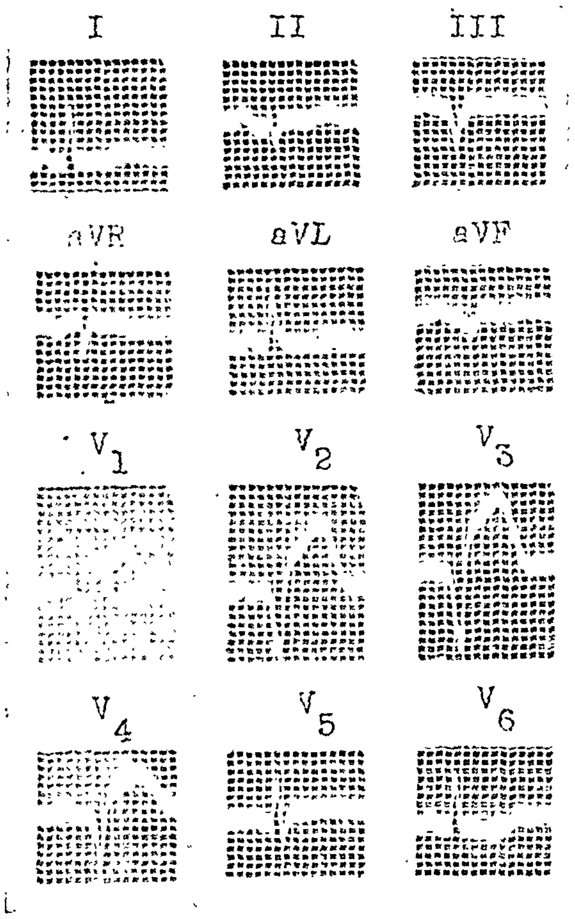

Fig. 1.—Electrocardiogram of patient with anterior myocardial rupture. Note the evidence of full-thickness infarction (QS waves) in leads V_1 through V_4.

In 77 (96.25%) patients, the infarcts were of the left ventricle, and in 3 they were of the right ventricle alone. In 11 (14.3%) of the 77 with infarction of the left ventricle, the infarction had spread to a portion of the right ventricle; in 46 (59.6%) the infarction was anterior, in 28 (36.4%) it was posterior, and in 3 (4%) it was lateral. Detailed subdivision of the locations of infarctions appears in table 2, but it may be noted that of the patients with anterior infarctions, the *septum* was involved in 31 (67.4%), and of those with posterior infarctions, the septum was involved in 17 (60.7%); of the entire group, therefore, the *septum* was involved in 50 patients (62.5%).

The distribution of the locations of ruptures is shown in table 3. Anterior left ventricle perforation occurred in 37 (46.25%) of the patients; in 31 (86.5%) of these, it was parallel to, in, or immediately adjacent to the interventricular septum. Posterior left ventricle perforation occurred in 25 (31.25%); in 16 (64%) of these, it was parallel to, in, or adjacent to the interventricular septum. In 9 the rupture was in the lateral left ventricle. In 7 it was through the right ventricle and was related to the interventricular septum. Multiple anterior perforations occurred in two instances, with specific locations of each perforation not given.

It is apparent, then, that in 54 (67.5%) of the entire group the rupture was parallel to, in, or immediately adjacent to the interventricular septum. It is also apparent that although the septum was actually involved in only 67.4% of those with anterior infarctions, in 86.5% of those with anterior ruptures the ruptures were *intimately* related to that structure.

TABLE 2.—*Location of Infarctions*

Location	No. of Patients	
Left ventricle (total)		77
Anterior (total)		46
Entire anterior wall	5	
Anterior wall and intraventricular septum	31	
Anterior wall and septum	14	
Anterior-apical and septum	12	
Anterior, septum, and right ventricle	4	
Anterior wall and basal septum	1	
Anterior-basal	1	
Anterior-lateral	6	
Anterior-apical	1	
Anterior-apical and lateral	1	
Posterior (total)		28
Entire posterior wall	3	
Posterior wall and intraventricular septum	17	
Posterior-basal and septum	2	
Midposterior wall and septum	2	
Posterior, septum, and right ventricle	7	
Posterior wall and septum, entire length of septum	4	
Posterior-lateral and septum	2	
Posterior-basal and posterior papillary muscle	1	
Midposterior wall	2	
Posterior-apical	2	
Posterior-lateral	3	
Lateral (total)		3
Right ventricle (total)		3
Anterior-apical and intraventricular septum	2	
Apical-lateral	1	
Total		80

In this entire group, the great majority of perforations were dissections, often by devious routes, from endocardium to pericardium. The length of dissection in each instance was variable; not only did blood dissect in a tortuous course through the ventricular wall in many cases, but in several instances the path was through the ventricular wall for several centimeters and then into the septum, parallel to the two septal surfaces, with final perforation into the pericardial cavity from the septum itself. The so-called blowout occurred in only a few instances, and in each of these it was through either the free lateral wall of the left ventricle or through an extensively necrotic anterior wall of the left ventricle.

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[5] (*a*) Edmondson and Hoxie,[1a] Krumbhaar and Crowell,[3c] Benson and others.[3d] (*b*) Snyder, G. A. C.: Spontaneous Double Rupture of the Heart, Arch. Path. **29**: 796 (June) 1940. (*c*) Lodge-Patch, I.: The Ageing of Cardiac Infarcts, and Its Influence on Cardiac Rupture, Brit. Heart J. **13**: 37, 1951.

COMMENT

Comparison of our data with figures obtained at an earlier period at the Los Angeles County Hospital by Edmondson and Hoxie[1a] shows that the increase in the number of patients with myocardial infarctions, about which they commented, has continued since the time of their study. Of all patients on whom autopsies were performed during the years they reviewed, 3.4% had one or more unhealed myocardial infarctions. Over twice that number (7.58%) were found in the same classification in the present study. The number of patients with rupture of the heart, however, was similar in the two series, with 8.3% in their group and 7.11% in ours. The generally accepted incidence in the accumulated literature is about 9%.[1d]

Age, Sex, and Race.—Our figures on age, sex, and race are worthy of further comment. Not one of our group, including the unreported series of 11 patients with interventricular septal perforation, was below the age of 50 years; by far the highest incidence for both sexes occurred between ages 60 and 80 years. On its face, this data apparently is good evidence that the clinician need have little fear of cardiac rupture in patients below the age of 50 years. Regarding sex, women have an incidence of myocardial infarction in various autopsy and clinical studies only one-fourth[6] to one-seventh[7] as high as men. When considered in the light of sex differences, as revealed by an autopsy "population" study by Zinn and Cosby,[1c] the autopsy incidence is about equal for the two sexes. In our group of 80 patients with cardiac rupture, however, 47 (58.75%) were women and only 33 (41.25%) were men. This difference is certainly significant and indicates the greater danger that this complication holds for women than for men. Especially is this so if it is recalled that the peak incidence of myocardial infarction in women occurs at a later age than in men. As far as race is concerned, it has been shown in certain series that the Negro race has a lower incidence of infarction than the Caucasian in a given autopsy population. This is borne out in our series, only 1 of 80 patients being Negro.

TABLE 3.—*Location of Ruptures*

Location		No. of Patients
Anterior left ventricle (total)		37
Parallel to, in, or near intraventricular septum	31	
Anterior-basal	1	
Anterior-lateral	2	
Anterior-apical	1	
Anterior, specific location not stated	2	
Posterior left ventricle (total)		25
Parallel to, in, or near intraventricular septum	16	
Posterior-basal	1	
Posterior-lateral	4	
Posterior-apical	1	
Posterior, specific location not stated	3	
Lateral left ventricle (total)		9
Midlateral	7	
Apical-lateral	2	
Right ventricle (total)		7
Anterior-apical, related to intraventricular septum	5	
Posterior-basal, adjacent to intraventricular septum	2	
Multiple anterior, specific location not stated (total)		2
Total		80

Relation of Activity to Rupture.—Previous studies have shown that in certain instances there is a definite relation between the degree of activity after infarction and myocardial rupture. Because of the specialized classification of the patients considered in at least one such study,[1c] this problem was reevaluated. As has been noted above, 49 (61.25%) of the patients in our series were in the hospital and in bed either at the time of or within 24 hours after onset of infarction. It is difficult to conceive how the course of events in these patients could have been affected by activity. In addition, as mentioned earlier, several of the patients who entered relatively late in the postinfarction period had been in other hospitals or otherwise under the care of private physicians. If it is assumed that all or some of these patients were kept at rest before transfer to the Los Angeles County Hospital, then the available evidence suggests even more strongly that activity does not play an important role in the development of myocardial rupture. There may be exceptions in certain special instances or in cases in which activity has been actually excessive. That there may be a relation between activity and myocardial rupture, however, is possible. This is based on consideration of the fact that while 58.75% of the ruptures occurred in women, only 42.7% of the women entered the hospital during the first 24 hours. On the other hand, while only 41.25% of the perforations occurred in men, 51.5% of the men entered the hospital in the first 24 hours.

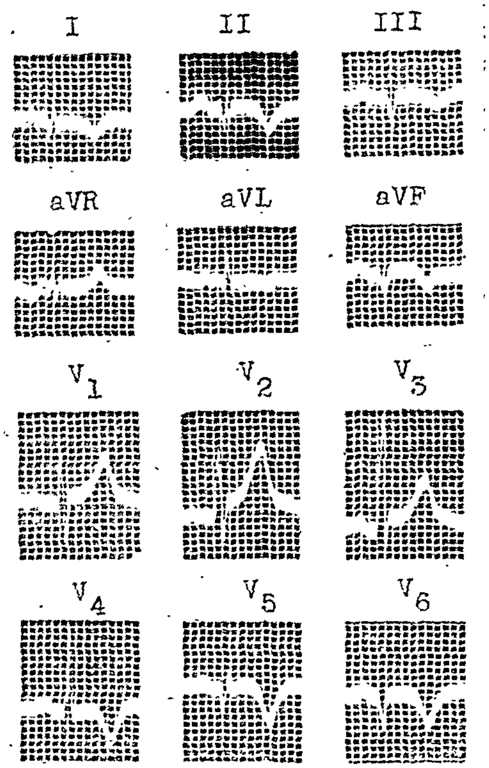

Fig. 2.—Electrocardiogram of patient with posterior myocardial rupture. Note that evidence of full-thickness infarction does not appear until lead V_6.

It has been stated repeatedly that the majority of deaths from myocardial rupture occur from the 2nd to

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6. Clawson, B. J.: Incidence of Types of Heart Disease Among 30,265 Autopsies, with Special Reference to Age and Sex, Am. Heart J. **22**: 607, 1941.

7. Parkinson, J., and Bedford, D. E.: Successive Changes in the Electrocardiogram After Cardiac Infarction (Coronary Thrombosis), Heart **14**: 195, 1928.

the 10th day after infarction. Our figures do not differ greatly from these previously reported values. It is of interest, however, that in 36 (45%) patients rupture occurred within the first 72 hours; in 14 of these, in fact, it occurred within the first 24 hours, and in 10 within the second 24 hours. In one instance, a patient previously well reported for work, suddenly collapsed, and died. Several others were brought to the admitting room of the hospital because of severe chest pain and died at most only a few hours after onset of pain, before more than a cursory examination could be performed. On the other hand, rupture occurred after 19 days in 6 patients. At autopsy these patients had only the infarcts that had caused hospitalization; no evidence of more recent infarction was found. The maximum time after infarction of which we could be reasonably certain was 30 days. It is probably of significance that while approximately equal numbers of men and women died within the first 3 days (19 and 17, respectively), the percentage distribution was 57.6% men and 36.2% women. Men apparently are in greater jeopardy during the immediate postinfarction period.

Electrocardiographic Observations.—Myocardial necrosis is considered essential for development of the rupture, according to pathologists. If presently accepted electrocardiographic concepts are valid, semidirect leads over these areas should record only QS waves. The evidence obtained in this study appears to agree with that concept. In multiple precordial leads made in cases of anterior infarctions, a full-thickness infarct pattern (QS) was obtained at one or more areas. In cases in which a single lead, CF_4, was made, QS complexes were recorded in a majority of instances. In six patients with deep Q waves, lateral or medial displacement of the electrode theoretically would have revealed QS complexes. The ease with which the anterior and lateral portions of the heart may be explored electrocardiographically should enable the clinician to determine with a high degree of accuracy the extent of an infarction in those locations, both in thickness and in surface area involved. In our series, infarctions and ruptures were in anterior or lateral portions of the heart in 66.6% of the patients. In a recent survey of the literature [1d] it was shown that ruptures were anterior in 75% of 80 patients in whom the position of the rupture was reported. It would appear evident, then, that a clue to the possibility of impending rupture (i. e., a full-thickness infarct) may be elicited in two-thirds to three-fourths of the patients who will suffer this catastrophe.

The situation is somewhat different in the case of posterior rupture. Some evidence was obtained from lateral chest leads, but not with the same degree of certainty as in the case of anterior rupture. Esophageal or back leads would be of aid—the former if it were feasible to use these leads in critically ill patients, and the latter if they were in more general use and the criteria of normalcy were more definitely known.

Location of Infarcts and Ruptures.—It is well recognized that the majority of myocardial infarctions occur in the left ventricle. Wartman and Hellerstein [8] reviewed 235 infarcts in 160 patients. They found that the anterior half of the interventricular septum was involved in 54% of the patients who had an infarct of the anterior portion of the left ventricle. The posterior half of the septum was involved in 25% of posterior infarctions. Our series gives a somewhat distorted picture because of the fact that we considered only recent infarctions, although relatively few of the patients had old scars. Nevertheless, as noted earlier, 67.4% of anterior infarctions and 60.7% of posterior infarctions involved the septum. All infarctions in the right or left ventricle that involved the septum constituted 62.5% of the total. While percentage values based on such dissimilar series are difficult to compare, the extreme differences between them show that there tends to be a greater incidence of infarcts involving the septum in myocardial rupture than in those instances in which there is healing or death due to other causes.

Although loosely considered statistics may be fallacious, it is interesting to note that in 54 (67.5%) patients the perforations were intimately related to the septum. There appears to be a definite relationship between the number infarcts involving the septum and the affinity of perforations for the septum. Also, while the infarcts were confined solely to the right ventricle in only three patients, ruptures occurred in the right ventricle in seven. Finally, it is important to realize that few of the "ruptures" were actually ruptures in the sense of an area of heart muscle suddenly blown out of place. Almost all were dissections and perforations. This is generally true, also, of cases reported in the literature. No explanation is immediately apparent for this high percentage of infarcts and perforations that are intimately related to the interventricular septum. No explanation will be attempted here beyond consideration of a few possibilities. First, with the septum relatively fixed between the two ventricles, contraction may result in a tear along a line of "cleavage" between contracting myocardium and nonfunctioning necrotic tissues. Second, the initial tear through which blood later dissects may be related to the various muscle bundles of the heart and the ways in which they may work against or with each other during contraction.

Experimental work reported several years ago showed that intraventricular pressure apparently was not related to the development of rupture.[9] The large number of dissections, the small number of blowouts, and the fact that in 4 patients with both right and left ventricular infarctions the rupture occurred in the relatively low pressure right ventricle all suggest that only infrequently does intraventricular pressure alone play a significant role. Beyond some specific facts gleaned from a survey of autopsies, there is little we can state as to exactly why a given heart will rupture.

SUMMARY

1. Eighty patients who died in the period 1941 to 1951 of perforation of the myocardium with hemopericardium and cardiac tamponade have been reviewed. All of the

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[8] Wartman, W. B., and Hellerstein, H. K.: The Incidence of Heart Disease in 2,000 Consecutive Autopsies, Ann. Int. Med. 28:41, 1948.

[9] Thomas, W. C.: Clinicopathologic and Experimental Observations on the Pathogenesis of Rupture of the Heart Due to Myocardial Damage, Am. J. Path. 23:904, 1947.

patients were over the age of 50 years. Women predominated over men (47 and 33, respectively). Only one of the group was Negro; the remainder were Caucasian.

2. Sixty-four (80%) of the patients were either in the hospital in bed or entered the hospital within the first 72 hours after infarction occurred. A higher percentage of men died within the first 72 hours than women (57.6% of the men and 36.2% of the women). Of all patients, 36 (45%) died within the first 72 hours of infarction.

3. Contrary to previous speculation, there was no evidence that digitalis administration was related to myocardial rupture.

4. Multiple unipolar lead electrocardiograms may give evidence of impending rupture by showing a full-thickness infarct pattern in anterior infarction in patients who otherwise fit the general clinical picture of those reviewed here.

5. The interventricular septum was involved in infarction in a greater number of these patients than could probably be expected in a random sampling of recent infarcts found at autopsy. Of the entire group, 67.5% had ruptures parallel to, in, or immediately adjacent to the interventricular septum.

6. The great majority of ruptures were dissections and perforations; only a few were "blowouts."

7. In general, with the exception that the amount of postinfarction activity these patients underwent did not appear significant, the results of the clinical survey did not differ considerably from the results of previous investigations referred to in the introduction.

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SPEECH REHABILITATION AFTER TOTAL REMOVAL OF LARYNX

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The problems that confront the patient who has undergone a radical laryngeal resection are generally not too well known. To present a total picture of the person who has undergone radical laryngectomy and his problems is one of the aims of this paper. Further, it is our purpose to show that practically all patients, after a total laryngectomy, can acquire a natural form of speech [1] without the use of any artificial aid and that they can resume their former occupations and activities requiring the use of the voice.

The number of patients with cancer of the larynx who have a total laryngectomy is increasing yearly.[2] They number in the thousands. Physicians in many communities in the United States and other countries now have to care for many more of these persons, whose medical needs are many, sometimes unique. The necessity for speech rehabilitation after the operation is now more widely recognized. Formerly it was believed that it was essential only to operate and save a life. Now it is realized that the laryngeal amputation is just the beginning and that the restoration of practical speech to the patient must follow.

After a radical laryngectomy the patient's entire economic and social status abruptly changes. Emotionally, a terrific blow has been sustained. He has been given a diagnosis of cancer; a serious operation and hospitalization follow; and immediately after the operation the patient is entirely speechless. The economic and other handicaps that will follow must be considered, for a radical readjustment of the patient's entire life is necessary. Only those dealing with large numbers of such patients can fill in the unhappy details.[3] Needless to say, the patient's discharge does not constitute the end of the case; it is just the beginning of the rehabilitation program that must follow.

The severest handicap that remains after the operation is the absence of speech. It is regrettable that patients are sometimes discharged without being informed that esophageal speech is a definite possibility. It is important that they be told this before the operation is started in order to prepare them emotionally for the postoperative and convalescent period. Patients are often discharged simply with a sheet of instructions on the use of the artificial voice box. Formerly, patients were dismissed without instructions of any kind in relation to speech therapy; however, this omission is fortunately becoming rarer. Surgeons who remove the larynx now assume the additional obligation of initiating a training program for their patients. The artificial mechanical devices should be reserved for the exceptional patient who cannot master esophageal speech for some special reason.[1] The mechanical larynx, aside from its other deficiencies, produces a tone that does not remotely resemble the human voice.

ALTERED ANATOMY AND PHYSIOLOGY AFTER THE OPERATION

Since esophageal speech depends on the remaining and altered anatomy and physiology, the more important changes following total laryngectomy will now be reviewed. After surgery the patient no longer inhales or exhales air through the nose and mouth. Only the esophagus connects with the mouth and nasopharynx, for the airway has been disconnected. A new orifice has been created in the neck where the tracheal stump has been sutured to the skin. Through this opening air is piped down into the lungs, and this air is sometimes exhaled with a noticeable hissing sound. The patient is now

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1. Levin, N. M.: Teaching the Laryngectomized Patient to Talk (Without Aid of Mechanical Larynx), Arch. Otolaryng. 32:299 (Aug.) 1940; Teaching the Laryngectomized Patient to Talk, Year Book of the Eye, Ear, Nose and Throat, Chicago, The Year Book Publishers, Inc., 1940, p. 592.

2. Martin, H.: Incidence of Total Laryngectomy, Ann. Otol., Rhin. & Laryng. 59:359, 1950; Cancer of the Larynx in Nelson's Loose-Leaf Surgery, 1947 revision, New York, Thomas Nelson & Sons, chap. 5.

3. Levin.[1] Schall, L. A.: Psychology of Laryngectomized Patients, Arch. Otolaryng. 28:581 (Oct.) 1938. Mason, M.: Rehabilitation of Patients Following Surgical Removal of Larynx, J. Laryng. & Otol. 64:759, 1950.

affect future esophageal speech requirements. Further experience in this regard is needed.[5]

The mortality and morbidity rates incidental to a laryngectomy have been reduced to minimal figures. With the help of the antibiotics, sulfonamides, whole blood transfusions, good anesthesia, and nutritional intravenous therapy, the period of hospitalization has been markedly reduced, from 8 to 12 weeks to 10 to 18 days. The total laryngectomy is recommended as the procedure of choice in selected cases of carcinoma of the larynx.[6] Additional radical neck dissection may be required in some cases. Five-year cures occur in 60% of the total number of patients operated on, many of whom live out their normal life spans. By contrast, deep x-ray therapy promises five-year cures in only 10% of patients.[6] Following radiation, there is, of course, no loss of speech.[7] The ideal procedure is the partial laryngectomy (laryngofissure), for it results in five-year cures in 75 to 85% of patients, without any serious impairment of speech.[8] Unfortunately, not too many qualify, for usually when the patient appears for biopsy, the growth has already extended beyond the scope of this operation.

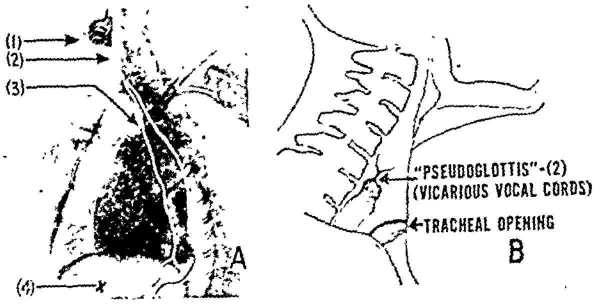

Fig. 3.—*A*, roentgenographic demonstration of esophageal speech mechanism (patient is saying "church"): (1) air-distended pharyngeal pouch; (2) vibrating upper esophageal sphincter, acting as substitute vocal cords (pseudoglottis); (3) air-distended esophagus; (4) gastric air, not ordinarily utilized in this speech mechanism. *B*, schematic representation of *A*, to show substitute vocal cords approximating at exact instant that a sound is produced.

MECHANISM OF ESOPHAGEAL SPEECH

The mechanism on which esophageal speech is based is fundamentally simple.[9] This mechanism utilizes structures that normally remain after the operation or that, if altered, can be trained to assume new functions. A comparison of the mechanisms of normal speech with those found in patients who have undergone total laryngectomy shows that the basic essentials for producing sound in both are the same. In each, a moving column of air from below vibrates closely approximated mucous membrane surfaces, thus producing a sound. The sound is then delivered into the mouth, where it is converted into words and sentences (speech) by the lips, teeth, tongue, and palate (the "molds of speech").

Esophageal speech differs from normal communication mainly in the anatomic structures involved. Normally, air from the lungs passes, on exhalation, up through the narrow aperture of the closely approximated and vibrating vocal cords (the glottis). In esophageal speech, the air pocketed in the upper esophagus passes upward through the closely approximated membranous surfaces of the vibrating cricopharyngeal sphincter (the pseudoglottis) into the hypopharynx. The sound thus produced is converted into speech by the "molds," which remain unaltered after surgery. I accomplished visualization of the substitute vocal cords in action, by radiography, in 1939.[1] It was clearly demonstrated that the folds of the cricopharyngeal sphincter approximated and vibrated at the exact moment that sound was produced and thus closely resembled the action of the true vocal cords on phonation. A column of air escaping through the vibrating sphincter constitutes the mechanism of sound production in esophageal speech. Normally this is also the mechanism that acts in belching. If this column of air can be controlled by prolonging the expulsion at will, the patient can then produce sounds that are converted into esophageal speech.[10]

It is neither a miracle nor a coincidence that air expelled from the upper alimentary tract can be used for producing sounds that can be converted into speech. In some animals and birds, sounds are normally produced by the use of anatomic structures other than the larynx. At times the upper esophageal sphincter must be removed to completely eliminate an extensive malignant lesion. In such cases other tissues may have to take over the function of substitute vocal cords in the hypopharynx. These tissues may be membranous or scar tissue folds, or combinations of these. The base of the tongue against the posterior pharyngeal wall, for example, can also be made to vibrate.

The upper alimentary tract offers a very satisfactory substitute for the respiratory tract in the new speech mechanism. The muscular cricopharyngeal sphincter is strategically situated at the mouth of the esophagus (pharyngoesophageal junction), approximately at the former level of the removed larynx. This muscular ring normally remains contracted and opens reflexly only during swallowing, vomiting, or belching. Since this sphincter also has striated fibers under voluntary control, it can be trained to act as a substitute for the vocal cords. The central nervous system still controls the speech mechanism, even though the larynx has been removed and the lungs are no longer necessary for speech production. The thorax still acts in a normal way, but on an inflated esophagus instead of on the air-filled lungs as in normal speech.[11]

The mechanism of esophageal speech in continuous action can best be described by a composite picture made up of repeated clinical observations on patients who have had total laryngectomy and by fluoroscopic and radiographic evidence.[12] The student begins to talk by closing his mouth, thus forcing air down into the pharynx, which

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5. Levin.[1] Jackson.[4b] Sokolowsky, R. R.: Effect of Extrinsic Laryngeal Muscles on Voice Production, Arch. Otolaryng. **38**:355 (Oct.) 1943. Crowe.[4c] Jackson.[4d] New.[4f]

6. Martin.[2] Jackson.[4b] New.[4f] Ward, G. E., and Hendrick, J. W.: Diagnosis and Treatment of Tumors of the Head and Neck: Not Including the Central Nervous System, Baltimore, the Williams & Wilkins Company, 1950, p. 597.

7. Kaplan, I. I.: Clinical Radiation Therapy, New York, Paul B. Hoeber, Inc., 1949, p. 340.

8. Jackson, C. L.: Voice After Direct Laryngoscopic Operations, Laryngofissure and Laryngectomy, Arch. Otolaryng. **31**:23 (Jan.) 1940.

9. (*a*) Levin.[1] (*b*) Stetson, R. H.: Esophageal Speech for Any Laryngectomized Patient, Arch. Otolaryng. **26**:132 (Aug.) 1937.

10. (*a*) Levin.[2] (*b*) Morrison, W.: Physical Rehabilitation of Laryngectomized Patient, Arch. Otolaryng. **34**:1101 (Dec.) 1941. (*c*) Stetson.[9b]

11. Jackson.[4b]

12. (*a*) Levin.[1] (*b*) Stetson.[9b] (*c*) Lindsay, J. R.; Morgan, R. H., and Wepman, J. M.: Cricopharyngeus Muscle in Esophageal Speech, Laryngoscope **54**:55, 1944.

becomes dilated; then the cricopharyngeal sphincter suddenly opens, permitting the air to go down into the esophagus. All this occurs with amazing rapidity. Immediately after the air is aspirated or swallowed, the esophagus dilates considerably. When the patient begins to make a sound, the air is forced upward, by a rising diaphragm, through the sphincter into the pharynx. The resulting sound is immediately converted into speech. As the patient gains facility by practicing the intake and subsequent expulsion of air, the act becomes much easier and faster. Later it becomes an involuntary reflex entirely. The gastric air pocket is not utilized in the speech mechanism of trained patients, a fact that is easily demonstrated by fluoroscopy or radiography.[13]

METHODS OF INSTRUCTION AND TRAINING

Formal instruction is necessary for most laryngectomy patients to acquire esophageal speech.[14] Occasionally a patient stumbles spontaneously into an excellent method by trial and error; however, learning need not depend on chance but can be directed by using methods that are known to be effective. Before a course of training is initiated, the patient must be examined to determine whether he is a suitable candidate. In the preliminary examination it must be established by fluoroscopy and radiography whether the hypopharyngeal pocket is ample and the upper esophageal sphincter functionally normal. At the same time the status of the respiratory function and the cardiovascular system is ascertained. The nose and throat are examined, and the hypopharynx and the esophageal mouth are inspected with a laryngeal mirror or, if necessary, an endoscope. A search is made for metastases, and the general health of the patient is considered.[1] Hearing must be adequate.

Preliminary orientation and training[15] may begin before the operation, whenever possible. After the operation the patient communicates by writing and is not permitted to attempt speech until the pharynx is completely healed. Whispered speech is ineffective, and it is prohibited because it interferes with the development of esophageal speech. Under ideal conditions, and as soon as the patient's physical condition permits, systematic raining should begin. In addition to those patients who e regularly receiving speech instruction, convalescent atients and those scheduled for surgery may "sit in" during these classes. The psychological benefit for the latter is considerable.

A résumé of training methods used in successful speech rehabilitation[1] follows. 1. Patients are given an outline of the anatomic and physiological changes following the operation. They are taught that they must disassociate the separate functions of respiration through the tracheal orifice in the neck and aerophagia and phonation. Most patients grasp this idea readily. 2. Students begin to practice the aspiration and swallowing of air and its subsequent expulsion. Sipping carbonated liquids or water aids in the sensation of filling the hypopharyngeal pocket, because the effervescence helps in expelling this accumulation. 3. Using this expelled air, patients practice such easy words as church, scram, welsh, and others, and they are pleasantly surprised when these initial efforts result in clearly audible words. With the knowledge that they can now make themselves understood, there is considerable encouragement. 4. One-syllable words can readily be managed with one swallow of air; later, a sequence of syllables can be produced with an equal volume of air. 5. Combinations of various consonants with vowels are practiced, and, after the mastery of short words, words of more than one syllable are attempted. Long words are broken up into their component parts; thus, San Francisco becomes San-Fran-Cis-Co. 6. A series of short words are then combined into simple sentences, with the emphasis on clarity and sharp, clear diction. The intake of air is characterized by labored interruptions, as this is a deliberate, voluntary act. Later, the patient no longer consciously swallows or aspirates the air, as it has now become almost entirely a reflex habit.

Students are thoroughly drilled in each of the preceding steps for an hour daily. At the end of two weeks they can make nearly all of the fundamental sounds, say short words, and combine words into simple sentences. During the next three or four weeks the effort is directed toward polishing the speech and eliminating various defects, such as loud expiratory blowing sounds issuing from the tracheal orifice, gulping noises on swallowing air, unnatural lip movements, poor articulation, and others. Students can now practice three to four hours daily in interrupted periods.

Intelligence, willingness, and the determination to learn are essential requirements for the mastery of esophageal speech. Previous education is a help but not absolutely necessary. Some individual instruction is necessary, particularly in the initial and advanced periods of training. Beginners are much encouraged when they hear trained students talking. At the end of a month of instruction, the trainee usually talks quite well. His words are clear, and he is understood by almost everyone, although a few speech defects may remain. The patient is now ready for the test of talking to his friends and associates. Timid students are encouraged to listen to the new voice on a recording, so that they can appreciate the improvement and progress they have made. The hoarseness that characterizes early speech becomes less noticeable as students acquire greater facility and the voice more flexibility. Women learn to talk with the same ease as men. Persons who have undergone total laryngectomy generally present no unusual problems in training. Some however, have difficulties and peculiarities that require the experienced handling of a laryngologist who is also a teacher. The speech therapist should therefore consult with the surgeon to learn about the postoperative anatomic status of the difficult students. Some of this group have poor speech and show no progress in training, while others are using the mechanical devices unwillingly. These persons, of course, require special consideration.

Those who have accomplished esophageal speech come from all walks of life. We have trained salesmen, laborers, executives, engineers, society matrons, attorneys, housewives, a pharmacist, and others. Laryngectomy patients of all ages can be taught to master esophageal speech. The oldest student was well past 70, and the youngest was 29. It is much easier to train patients if instruction begins shortly after the operation; however, patients have mastered this method after periods of months to years. One man learned to talk after a 22-year interval. Patients using mechanical devices can learn to talk and thus discontinue these artificial aids. The patient past 60 learns without too much difficulty if he has the desire to do so. Those who have a negative attitude or are depressed are at times difficult to train. Failures have been comparatively few, for those who could not acquire the method were either in ill health, depressed, or would not apply themselves sufficiently.

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[13] Levin.[1] Morrison.[10b] Lindsay.[12c]

[14] Levin.[1] Morrison.[10b]

[15] McCall, J. W.: Preoperative Training for Development of Esophageal Voice in Laryngectomized Patients, Ann. Otol., Rhin. & Laryng. 52:364, 1943.

The patient who has mastered esophageal speech is able to talk distinctly in a normal way. The voice is satisfactory for normal speech activities and does not attract unusual attention, though it is somewhat harsh at first. A few patients develop voices so nearly normal that they are not recognized as laryngeal amputees, and their speech improves steadily over a number of years if correct methods are used.

ARTIFICIAL LARYNX AND OTHER MECHANICAL DEVICES

There are two common mechanical speech devices in use today: 1. The reed type of artificial larynx, which pipes air from the tracheal opening into the mouth, after a sound is produced by a vibrating reed. The resulting sound is then converted into a mechanical, artificial speech by the normal speech molds. 2. The electrolarynx, which is essentially a battery driven vibrator over which a diaphragm is stretched. This device is held against the neck. As the diaphragm vibrates, sound waves produced within the pharynx are delivered into the mouth and are articulated into a mechanical and muffled speech. The buzzing sounds resemble an electric razor in action.

Many of the patients who came for speech training were using artificial aids, but none was satisfied with them, even when they could use them successfully. All the patients were anxious to discard these mechanical prostheses, giving as the main objections and reasons that (1) they were conspicuous, attracting unwelcome attention; (2) they made the resulting voice unnatural and unpleasant, with a monotonous quality not resembling the human voice; and (3) they served as a constant reminder of the disability. Some patients who came for training had never used the artificial larynx, not having tried to master the artificial speech technique. Most patients were never informed that it takes as long to master these appliances as it does to acquire the more natural esophageal speech. Once speech is acquired, the aids are dispensed with in most instances. One patient preferred to use the instrument for telephone conversation only; another, a plant foreman, found that the artificial voice box carried well over the noises of machinery. An attorney who had been using the artificial larynx for 12 years was never reconciled to its use. He mastered esophageal speech in eight weeks, though his volume is still low because of the extremely radical neck, dissection procedure required in his case.

The artificial larynx is a poor substitute for a natural voice, and these mechanical devices should be reserved for only those few persons who cannot master esophageal speech for some special reason, such as (1) stenosis of the esophagus after extensive removal of a widespread malignant lesion; (2) resection of the cervical portion of the esophagus; (3) multiple handicaps; for example, laryngectomy plus deafness; (4) suspected recurrence, metastasis, or multiple lesions; for example, a bronchial carcinoma in addition to the laryngeal lesion; and (5) senility or other feebleness.

EMOTIONAL AND PSYCHOLOGICAL CONSIDERATIONS

Emotional disturbances of varying degree occur in nearly all of the patients.[3] While the introverted person suffers more in proportion, even the extrovert is depressed when he finds himself entirely speechless. These reactions are to be expected, because (1) the patient has undergone a major operation after confirmation of a serious diagnosis; (2) on discharge from the hospital he must return to his previous environment, with all of the economic and social problems involved, and where loss of speech is a serious defect; and (3) he has a fear of the recurrence of cancer, open or hidden, but definitely present. Formerly the patient faced the prospect of total laryngectomy with gloomy foreboding, the questions uppermost always being, "Will I survive?" and "What about my speech?" A hopeful answer can be justifiably given because of the five-year (and longer) cures that are on record.[16] Since the postlaryngectomy period requires major emotional and psychological adjustments, the chronically depressed type of person is not ordinarily recommended for this radical procedure without careful consideration.

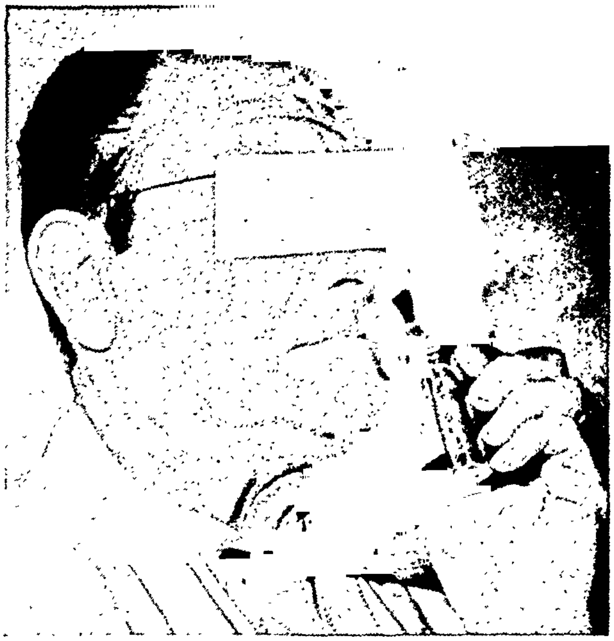

Fig. 4.—Laryngectomy patient using reed-type artificial larynx. Patients find mechanical devices such as this objectionable, because (1) they are conspicuous and attract unwelcome attention; (2) the resulting voice is mechanical and monotonous; and (3) they require the constant use of at least one hand, thus are impractical for bimanual job requirements.

REHABILITATION

Persons who have undergone total laryngectomy are located in many of the communities of the United States and other countries. Physicians who assume the care of these patients are very much interested in their unique and peculiar requirements. They must be supervised, instructed, and checked for a number of years. Certain restrictions must be observed by these patients:[16] (1) They should not engage in occupations in which they will be constantly exposed to dust or irritating fumes and sprays. (2) Obviously they must use precaution while bathing and should keep out of deep water, since this is always an additional hazard. (3) They must guard against aspirating foreign bodies or debris through the vulnerable tracheal orifice. A gauze apron worn over the opening offers considerable protection against dust and wind. (4) Patients who wear trachea tubes must have these cleansed and changed regularly, and the tracheal orifice should be watched for irritation and recurrence of malignant disease.

The rehabilitation program for these patients should start preoperatively,[15] when they are assured that they can talk again. They are much encouraged when they hear others who have mastered esophageal speech, and they should be advised to enroll for a course of speech training promptly. This is by far the most constructive part of this program and the best morale booster. Insight into the total need of these patients came only with my integrated experience acquired in the combined roles of laryngologist, laryngeal surgeon, and speech teacher. My contact with this large group since 1932 convinces me that these patients are optimistic as to their present problems and their future outlook when they have proper aid in adjustment. The majority are able to resume their former occupations or other employment requiring the use of the voice, even over the telephone. Some have addressed groups in public and over the radio. Although several patients have come from neighboring countries for speech therapy, language differences did not present any real problems. Most of these rehabilitated patients have made admirable adjustment to their former lives.

Esophageal speech training has elicited a widespread interest among physicians, medical institutions, and university speech departments. The vocational rehabilitation divisions of several of the southern states have shown an active interest in salvaging these persons. They have adopted very practical programs and have helped to return many speech handicapped persons to gainful employment. One man who came for speech training had won an election as state senator, using an artificial device during the campaign. Even though successful, he was never reconciled to the use of this aid. He learned esophageal speech in about eight weeks, then discarded the aid. Now, intensely interested in the rehabilitation of persons like himself, he has been instrumental in effecting legislation that provides for subsidizing esophageal speech training in his state.

Pioneering work has established esophageal speech on a more solid foundation. Increasing interest is attracting laryngologists and speech pathologists to do more original work in this field, although there is still need for more cooperative effort in the interest of these handicapped persons. In addition to the training facilities now available, there is need for more active teaching programs, both for patients and teachers, in well-located centers.

SUMMARY AND CONCLUSIONS

A total picture of the patient who has undergone radical laryngectomy and his problems is presented. It is shown that nearly all these patients can return to occupations and activities requiring the use of the voice. The more important characteristics of the altered anatomy and physiology following this radical procedure are reviewed. While total loss of speech is most important, there are other marked changes that require consideration. The emotional and psychological disturbances following the operation are also considered.

Esophageal speech depends on certain anatomic structures, the most important of which are the hypopharynx and the upper esophageal sphincter. Laryngeal surgeons make every effort to conserve these and other normal structures that is consistent with adequate removal of the malignant tissue. The mechanism of esophageal speech is described. There is considerable interest in those structures that anatomically and physiologically take over the functions of the resected larynx. A résumé of successful methods of instruction used in esophageal speech training is outlined. The artificial larynx and other mechanical devices are discussed.

A rehabilitation program essential for these patients is outlined. The number of such patients is increasing yearly, because surgical techniques now assure from 50 to 60% of five-year cures (and longer), and many of them attain their normal life expectancy.

Patients can now be reasonably assured of a practical speech development after total laryngeal resection. Mechanical aids, with all of their deficiencies and disadvantages, are unsatisfactory and should be reserved for those few patients who cannot learn a more natural form of speech because of special difficulties. In short, the possibility of vocal loss should no longer deter the physician from considering the total laryngectomy operation as the procedure of choice when indicated. Esophageal speech adequately compensates for the loss of this important function.

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16. Levin.[2] Jackson.[4b] Crowe.[4c] Jackson.[4d] Clerf.[4g] Tucker.[4h]

Cleft Lip.—*In newborn babies with cleft* lip alone or combined with cleft palate, the first treatment of course is concerned with surgical closure of the lip. Regarding early treatment, it should be stated that cleft lip and cleft palate do not constitute a surgical emergency. Frequently, the physician who has delivered a baby with one or both of these anomalies is under the impression that immediate closure of the cleft is imperative to enable the baby to nurse, and that without surgical closure starvation is imminent. This is erroneous. The surest way to kill a baby in a poor condition of nutrition is to operate on *it. No operation should be done until the child begins to gain weight and is otherwise in good physical condition.* By the use of a little ingenuity, feeding can be carried on by medicine dropper, spoon or special feeders. Swallowing is greatly facilitated by holding the baby during feeding in the upright position. Some surgeons, prompted possibly by the natural anxiety of the parents to have the deformity masked as soon as possible, advocate operation on the lip within a few days after birth. We do not subscribe to this, as we feel that accurate coaptation of the cleft edges is more difficult when the parts are so small and that much better end-results are obtained by waiting until the child is six weeks to three months old.—Robert H. Ivy, M.D., Modern Concept of Cleft Lip and Cleft Palate Management, *Plastic and Reconstructive Surgery*, February, 1952.

GENITOURINARY LESIONS IN LEPROSY

WITH SPECIAL REFERENCE TO THE PROBLEM OF ATROPHY OF THE TESTES

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and
Lawrence L. Swan, M.D., New Orleans

Genitourinary lesions specifically related to leprosy (Hansen's disease) are relatively few. Of considerable clinical interest and importance are the frequent leprous orchitis, testicular atrophy, and gynecomastia. A review of urologic literature reveals no reference to involvement of organs of the genitourinary tract by the Mycobacterium leprae. Of the standard urologic textbooks only Young's [1] refers to leprous orchitis.

In the general literature of leprosy only passing reference is made to the genitourinary manifestations. Fox and Knott [2] described cutaneous leprous nodules on the scrotum and penis in 4 of 34 male patients. Serra [3] examined 200 patients with leprosy, describing vulvar lesions in 13%, preputial lesions in 10%, and lesions of the balanic sulcus in 5%. These lesions were especially common in patients with the nodular and mixed types of leprosy, although it is known that macular leprosy of the penis and vulva often mimics primary chancre. One patient presented a nodular lesion at the balanopreputial sulcus following intercourse with a woman known to have leprosy. Inguinal lymphadenopathy developed, and Myco. leprae were found on microscopic examination.

The diagnostician must be particularly wary in many cases, for two surveys conducted by the United States Public Health Service revealed that in presumably nonsyphilitic leprous patients there were 45% to 59% false positive serologic reactions for syphilis.[4] In fact, it has been estimated that approximately half of the patients admitted to the National Leprosarium at Carville, La., have had a diagnosis of syphilis made sometime prior to admission.[5]

In Serra's group of 200 patients with leprosy, testis and epididymal involvement was described in 97% and 29%, respectively. Bacilli have been described in the epididymis and in the rete testis, and Kobayashi [6] has even described bacilli in the seminal ejaculum. In a series of 842 patients with leprosy Baptista [7] described gynecomastia in 72 (8.6%). In another group of 292, gynecomastia was observed in 17 (5.8%).

While the kidney and more rarely the adrenal are frequently involved in a nonspecific type of amyloidosis similar to that seen in other degenerative diseases, no significant renal or adrenal lesion characteristic of leprosy has been described. Nephritis has been a frequent finding. The degree of renal and adrenal amyloidosis is often pronounced, and in large areas of many kidney sections the glomeruli are almost completely obliterated by amyloid. The adrenals are less commonly infiltrated by amyloid. Ureteral, bladder, or prostatic involvement by any process peculiar to leprosy has not been recorded to the best of our knowledge.[8]

It is the problem of leprous orchitis and atrophy that forms the basis for this preliminary report, in which a few of the clinical findings are correlated with the histological changes observed in the testis.

MATERIAL AND METHODS

One hundred seventy-nine male patients of the United States Public Health Service Hospital, Carville, La., were examined. In 154, only the breasts and testes were observed. In 25, more detailed examinations were carried out, including high-span and caliper testis measurements. In addition, notation was made concerning beard growth, voice changes, thyroid cartilage size, ejaculation, impotence, temporal recession of hair, trunk and axillary hair, pubic hair, and external genitalia. When it became apparent that height-span measurements were not indicative of an eunuchoidal skeletal structure, sole to symphysis pubis and symphysis pubis to vertex measurements were not made.

Formal biopsy specimens of the testis were obtained from three patients, two of whom had gynecomastia. Breast material was available from one of those who had previously undergone mammoplasty. Sections of breast were typical of classical gynecomastia, so that further studies with relation to the gynecomastia were not emphasized.

In addition to biopsy sections of the testis, tissue obtained post mortem from 17 patients with lepromatous leprosy was fixed in formaldehyde (formalin®) for study. Microscopic sections of the testes were stained with hematoxylin and eosin and for acid-fast organisms. Selected sections of testes were stained with Masson's trichrome and congo red. Urinary 17-ketosteroids and gonadotropins were determined in two patients subjected to testis biopsy.[9]

PATHOLOGICAL CHANGES

In every patient examined there was definite microscopic evidence of severe involvement of the testis. The histological changes varied markedly; these changes have been arbitrarily divided into three phases: (1) vascular, (2) interstitial, and (3) obliterative.

Read before the U. S. Public Health Service Clinical Society, Detroit, June 7, 1952.

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Vascular Phase (Active Leprous Orchitis).—The major changes during this phase are vascular. The vessels of all sizes are infiltrated by lymphocytes. The width of the vessel wall is increased at the expense of the lumen (fig. 1), and lepromas are occasionally seen as nodules projecting into the lumen. Perivascularly, in the interstitial tissues, there are numerous lymphocytes, large pale cells, and vacuoles measuring up to 40 μ. Acid-fast organisms are present in impressive numbers (fig. 2). Many of the vacuoles seen with the hematoxylin and eosin stain are globi containing packets of Myco. leprae, and the large pale cells have many bacteria contained within their cytoplasm. These are the lepra, or Virchow, cells. Acid-fast bacilli are also present in the vessel walls and globi, and lepra cells are observed in the interluminal lepromas. There are all phases of spermatogenesis during this stage. The Sertoli cells and the interstitial cells of Leydig are not affected. The basement membrane and lamina propria of the tubules are not changed.

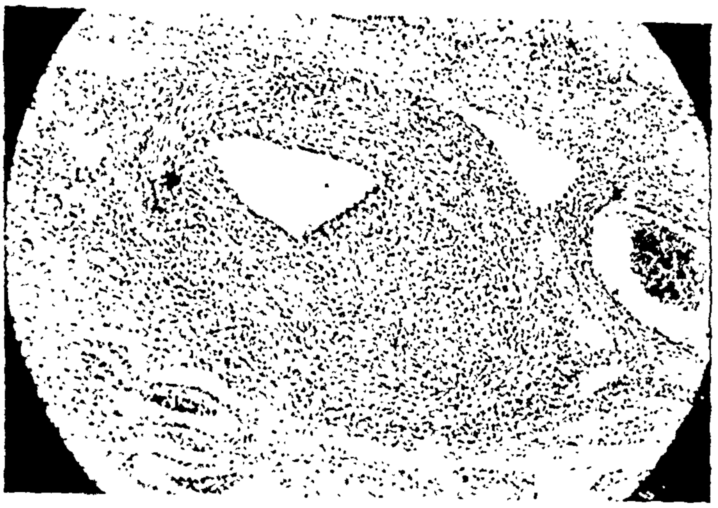

Fig. 1.—Lymphocytic infiltration of artery immediately adjacent to the tunica albuginia. There is widening of the arteriole wall and perivascular lymphocytic infiltration (hematoxylin and eosin × 83).

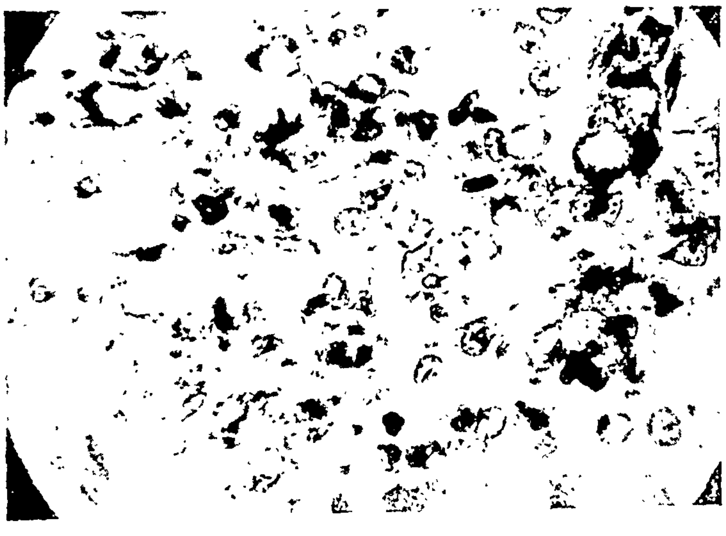

Fig. 2.—Acute leprous orchitis; acid-fast preparation showing typical organisms (× 430).

Interstitial Cell Phase.—Gradations into this phase are gradual, showing progressive reactive obliterative endarteritis, but the major findings consist of masses of Leydig cell clumps and interstitial fibrosis (fig. 3). The vessel walls are moderately to greatly thickened but lack any evidence of active inflammation as described in the preceding phase. The interstitial tissues show diffuse collagen deposition, with aggregates of Leydig cells. These cells appear to be larger, more eosinophilic, and granular than normal. There are numerous coarse brown granules in the cytoplasm in the so-called ectoplasmic zones of many cells. Crystalloid forms have not been observed within the cytoplasm. The seminiferous tubules are seen to be relatively smaller. The lumens are almost entirely filled with Sertolian syncytium. Occasionally spermatogonia are seen, but no mature spermatozoa are found. The basement membrane is wider than normal, and the lamina propria is considerably widened by concentric lamellae of delicate fibrous tissue. Acid-fast stains show considerable variation in the number of organisms at this stage. When they are present, globi are less frequently seen than in the preceding stage. These are present in the interstitial tissues in close relationship to the interstitial capillaries.

Obliterative Phase.—Again all degrees of gradation can be seen from the preceding phase to this final picture of complete fibrosis. Every microscopic field examined reveals the normal pattern to be replaced by a homogeneous fibrosis (fig. 4). No outline of seminiferous tubules can be detected, and vascularity is greatly diminished and almost limited to very small capillary-sized vessels. Acid-fast organisms, globi, and lymphocytes are almost uniformly absent.

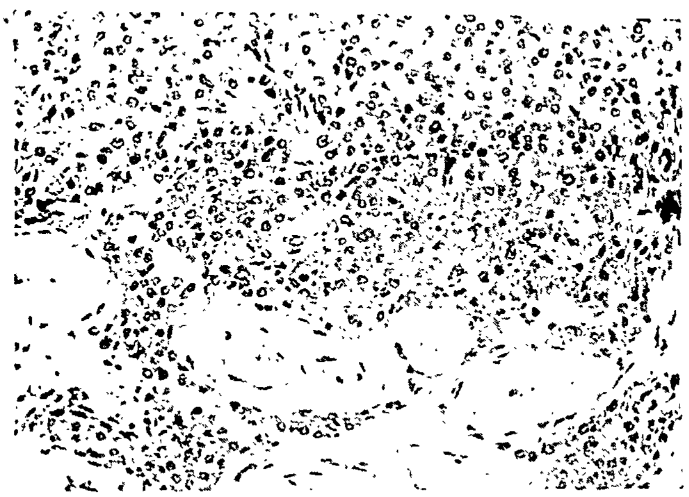

Fig. 3.—Aggregations of Leydig cells and atrophic seminiferous tubules in which a few Sertoli cells may be seen; acid-fast stain is negative (hematoxylin and eosin × 83).

Special stains of testis, epididymis, and ductus deferens for amyloid have been uniformly negative despite the fact that renal, adrenal, hepatic, and splenic amyloidosis is found in 65% of male patients studied postmortem. It appears that there is minimal vascular involvement of the epididymis during the bacteremia. Organisms, lymphocytic infiltration, and fibrosis of a lesser degree than in the testis are present in the epididymis. Giant cells, described by Young as being characteristic of leprous orchitis, have not been observed in our series.

PATHOGENESIS

Several features of the pathological picture are enigmatic. However, it can be stated with certainty that there is some degree of orchitis, or its residuals, in every male patient examined postmortem in this laboratory. At present it is difficult to correlate degree of atrophy with the age of the patient, or with duration or severity of disease.

It is remarkable that in leprosy the testis is so frequently involved and that in no other infectious disease process is there a quite comparable testis specificity. Obviously inherent physiological characteristics of the gonads or common embryological ancestry of the organs is of no significance, for the ovaries are not involved in any female patients studied postmortem in our series. It may be postulated that the environmental temperature difference between testis and ovary may account for this finding.

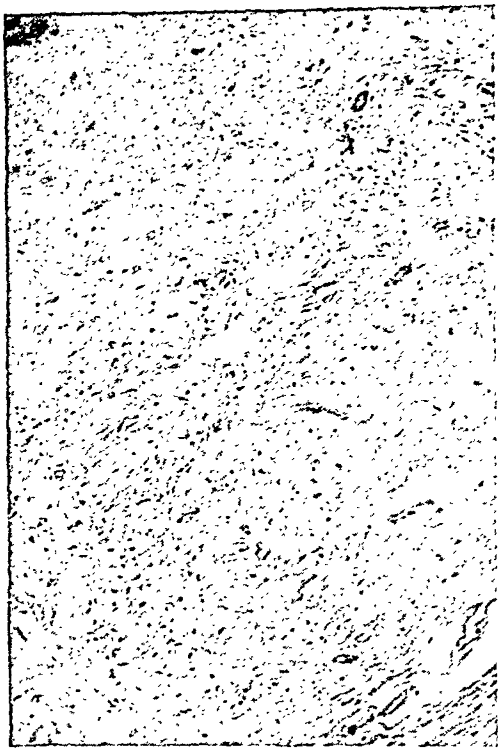

Fig. 4.—Complete fibrosis of testis; acid-fast stains negative (hematoxylin and eosin × 150).

The pathogenesis of leprous orchitis and final atrophy appears to be a transition from an active, acute lepromatous orchitis to a final picture of complete atrophy of the testis with obliteration of all landmarks by fibrous tissue. It is presumed that the initial lesion is infiltration of the vessel walls and adjacent tissue by Myco. leprae. There is a concomitant lymphocytic reaction, and large numbers of acid-fast organisms are seen. The bacteria are present in typical aggregates as elsewhere in the body. Next is seen a focal and, finally, a complete disappearance of germinal cells, with replacement by a syncytium of Sertoli cells, which completely fill the lumens.

The interstitial tissues appear to be next involved. There is often rather striking clumping of interstitial cells. It is doubtful that there is a true interstitial cell hyperplasia. Myco. leprae have not been demonstrated during this stage. Apparently, by a process of fibrosis, the final phase is a complete disappearance of Leydig cells and seminiferous tubules, with terminal obliterating endarteritis. It has been observed in our group that adrenal amyloidosis is found in all subjects whose testes exhibited interstitial cells in larger than normal numbers. The exact significance of this is not known.

CLINICAL FINDINGS

The duration of known leprosy in these patients, whose ages were between 17 and 79 years, was six months to 50 years. Of 50 testes measured in 25 patients, 14 (28%) were atrophic on the basis of measurements below 3.0 by 2.0 cm. (normal, 4.5 by 2.5 to 3.5 by 2.0 cm.). It has been observed that atrophy is almost always bilateral. In many instances the presence of rather large hydroceles, which is not unusual in this group of patients, has made it difficult to obtain accurate measurements. It has been our impression that testis atrophy is far commoner in patients with the lepromatous type than in those with the tuberculoid type of leprosy. Gynecomastia was present in 32 of 179 patients (19%) (fig. 5). It is obvious that many patients observed to have testis atrophy had no gynecomastia. In only one patient was the height-span measurement suggestive of a eunuchoid stature. There were no voice changes except in a 65-year-old patient suspected of having leprous laryngitis. Thyroid cartilage was normal in all subjects examined.

It is difficult to evaluate the shaving habits accurately in this group of patients. Many were Negroes from the Virgin Islands, who characteristically had a scanty beard. Many nonleprous patients reportedly shaved only once or twice weekly. There was no history of impotence in the group, and ejaculations in all were apparently normal. Sperm cell counts were not made. There was scanty trunk distribution of hair in 8 of 25 patients and sparse axillary hair in 5 of 25. It must be remembered, however, that leprosy in itself produces changes associated with decrease in body hair. In general, then, these patients have not had classical eunuchoid character

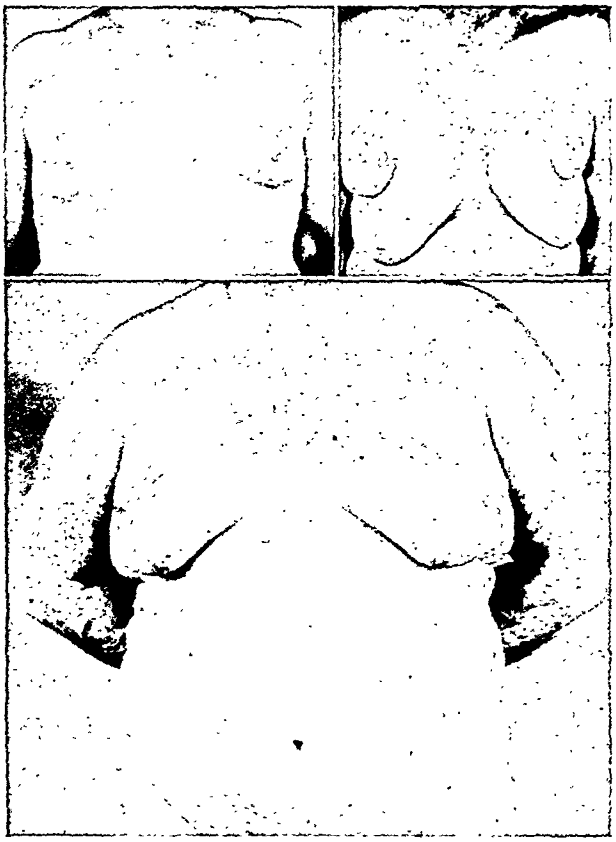

Fig. 5.—Types and degrees of gynecomastia.

A history of orchitis was obtained in 9 of 25 patients with atrophy, an incidence of 36%. This figure is rather high, and it is suggested that in many epididymitis, and not true orchitis, is the significant lesion. The sizes of testes varied from 4.7 by 3.0 to 1.7 by 1.4. These testes are not so small as to be immeasurable and are, therefore, slightly larger than those described in the group with the Klinefelter syndrome.[10]

The 17-ketosteroids were normal (7.8 and 9.7 mg. per 24 hours), and gonadotropins were markedly elevated (less than 288 and more than 384) in two subjects, only one of whom had gynecomastia. In the method utilized in these determinations no attempt is made to separate follicle-stimulating hormone and luteinizing hormone, and they are reported at total gonadotropins.

COMMENT

It is important to differentiate the terms "testis atrophy" and "hypogonadism" or "hypogenitalism." Whereas not all subjects with atrophic testes show overt manifestations of clinical hypogonadism, the vast majority with hypogonadism have either atrophic or completely absent testes (true eunuchism).

Rolnick [11] uses the term "primary hypogenitalism" to denote deficiency of pituitary gonadotropic factor with secondary testis failure. The more accepted terminology reserves the term "primary hypogonadism" for conditions involving intrinsic testis defects of either seminiferous or of interstitial cell elements. Secondary hypogonadism then implies the failure of either the production or the release of gonadotropic hormones. The classical causes of testis atrophy are legion and include cryptorchism; direct trauma; surgical trauma, as following orchiopexy or other scrotal surgery; vascular lesions, including infarction or torsion; senility; mumps; prolonged hyperpyrexia; debility; irradiation; avitaminosis; cirrhosis; schizophrenia; hypothyroidism; diethylstilbestrol therapy; diseases of the pituitary and hypothalmus; pseudohermaphroditism and true hermaphroditism; and inflammatory lesions, including leprosy. The atrophic testis is as vulnerable to secondary disease processes as is the normal testis. Seminoma and interstitial cell tumor formation have both been described in atrophic testes.[12] Secondary disease processes in the testes of those with clinical hypogonadism, however, have interestingly enough not been described.

In 1942 Klinefelter, Reifenstein, and Albright [10] reported in some detail a syndrome characterized by hyalinization of seminiferous tubules in small testes, aspermatogenesis, gynecomastia, normal Leydig cells, normal or decreased 17-ketosteroids, and increased follicle-stimulating hormone. These findings were described in nine subjects. Heller and Nelson [13] in 1945 described a similar entity. These authors studied 20 patients within a period of one year and had 30 additional cases over this same period that were not reported. They stressed the frequency of this syndrome. Three constant features of the syndrome were described by Heller and Nelson as (1) small testes, (2) azoospermia, and (3) high level of gonadotropins. Nine variable features were referable to skeletal changes, gynecomastia, external genitalia, hair distribution, pitch of voice, muscular development, Leydig cell appearance, 17-ketosteroid excretion, and excretion of estrogens. After a comparison of these features with those observed in the subjects described herein, it is apparent that we are dealing with what seems to be a very similar disease process. We have observed atrophic hyalinized testes, normal levels of 17-ketosteroids, elevated levels of gonadotropins, and gynecomastia, the more constant features. Perhaps leprosy is one of the known causes of this syndrome characterized by hypergonadotropic eunuchoidism.

An often neglected, yet important, phase of studies of testicular atrophy is associated liver disease. In 1926 Corda [14] reported a case of hepatic cirrhosis with associated testicular atrophy and gynecomastia. Since that time many similar reports have been described. Bennett, Baggenstoss, and Butt [15] have recently reported the study of another group of cirrhotic patients with testicular atrophy and gynecomastia. It has been suggested that in patients with severe liver disease, including cirrhosis, the normal inactivation of estrogen is interfered with and increased amounts are responsible then for such changes as gynecomastia. There appears to be no consistent correlation between liver damage and testicular atrophy in our series, in that cirrhosis has not been observed at any of the autopsies.

That any of these syndromes may exist without all the factors that go into its composition does not seem unreasonable. As Heller and Nelson remark in their study, "Because they lack gynecomastia these cases cannot be regarded as examples of the syndrome as originally described (by Klinefelter et al.); yet we hesitate to call these cases examples of a new and separate syndrome." The same might be applicable to these patients with leprosy. We agree with Heller and Nelson and believe that these may be a part of a rather large group of hypergonadotropic eunuchoids, and we hesitate to create a new name for the condition in these cases.

It is known that the testis produces two definite hormones: testosterone and estrogen. It is not within the scope of this paper to discuss the matter of a third hormone of the testis. Briefly it has been postulated that the Sertoli cells of the seminiferous tubules or the germinal epithelium itself produces an "x-hormone," called variously "inhibin" or "contruin," which is a pituitary antagonist. The functions of this second hormone include the inhibition of production of follicle-stimulating hormone, the support of spermatogenesis, and inhibition of the mammotropic hormone. With an absence of this hormone as would of necessity occur with hyalinization of the seminiferous tubules, there is then an increase in production of follicle-stimulating hormone, azoospermia, and gynecomastia. It should be noted that there are several good theories that militate against the presence of a third hormone. One such theory (Heller and Nelson) is that the elevated gonadotropins result from failure of the seminiferous tubules to utilize follicle-stimulating hormone.

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[10] Klinefelter, H. F., Jr.; Reifenstein, E. C., Jr., and Albright, F.: Syndrome Characterized by Gynecomastia, Aspermatogenesis Without A-Leydigism and Increased Excretion of Follicle-Stimulating Hormone, J. Clin. Endocrinol. **2**: 615, 1942.

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[12] Grabstald, H.; Haines, J.S., and Morrow, A. G.: Interstitial Cell Tumor of Testis, J. Urol. **66**: 708, 1951. Haines, J. S., and Grabstald, H.: Tumor Formation in Atrophic Tests, Arch. Surg. **60**: 857, 1950.

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[14] Corda, quoted by Silvestrini, R.: Gynecomastia in Men with Cirrhosis of the Liver, Riforma med. **42**: 701, 1926.

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No specific therapy has been undertaken for the testicular atrophy or gynecomastia per se. Since this group of patients possibly fits into the group of hypergonadotropic eunuchoids, it is not likely that pituitary hormone in any form would be of avail. Heller and Nelson have reported favorable response in their group to substitution androgen therapy. This may be of some value in patients with leprosy, but it has not been tried to date.

SUMMARY AND CONCLUSIONS

Genitourinary tract involvement in patients with leprosy is briefly described. One hundred seventy-nine male patients with leprosy (Hansen's disease) were examined with respect to atrophy of the testes and gynecomastia. Twenty-eight per cent were found to have atrophy, and 19% were found to have gynecomastia. In addition, a histological study of the testes of 20 subjects with leprosy is reported. This includes material from autopsy and surgical biopsy. A rather characteristic picture of leprous orchitis with infiltration of the vessel walls and adjacent tissues by Mycobacterium leprae, interstitial cell clumping, and atrophy of seminiferous tubules progressing to complete fibrosis and obliterating endarteritis is described. The 17-ketosteroids were normal and gonadotropins were increased in two of the subjects whose testes were subjected to biopsy.

It is suggested that this group of patients, despite their not presenting typical eunuchoid features, fits into the general category of hypergonadotropic eunuchoids described by Klinefelter, Reifenstein, and Albright and by Heller and Nelson, with characteristic atrophic testes, normal 17-ketosteroids, elevated level of gonadotropins, and gynecomastia. It is postulated that leprosy is perhaps one of the few disease entities of this group in which the etiological factors are known.

RECURRENT LEFT LARYNGEAL NERVE PARALYSIS IN ARTERIOSCLEROTIC HEART DISEASE

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and
Gerald William Nice, M.D., Topeka, Kan.

Hoarseness due to recurrent left laryngeal nerve paralysis is a rare occurrence in patients with congestive heart failure of any cause but is most commonly seen with severe mitral stenosis, and less commonly with congenital heart disease,[1] when marked dynamic dilatation of the left pulmonary artery compresses the nerve against the aortic arch, its adjacent lymph nodes, and the ligamentum arteriosum.[2] It is extremely uncommon in patients with arteriosclerotic heart disease, having been reported previously only by King, Hitzig, and Fishberg.[3]

REPORT OF A CASE

A 55-year-old white male married engineer was first admitted to Winter Veterans Administration Hospital Aug. 12, 1948, complaining of short attacks of substernal pressure-like pain, occasionally radiating down both arms, during the previous two winters. He had also noticed easy fatigability and shortness of breath on exertion since February, 1948. This became severe and accompanied by nocturnal dyspnea in June, 1948. Since that he had been treated with digitalis, with improvement. For the two years prior to examination his voice would break occasionally for a few seconds at a time. On June 1, 1948, he awoke in the morning with hoarseness, which remained until the time of admission.

At the time of physical examination the patient was in no distress. His blood pressure was 110/90. His pulse was regular, with a rate of 72. The heart tones were somewhat faint, and there were no murmurs. The lung fields were clear to auscultation and percussion. The peripheral vessels were not remarkable. There was no cyanosis, edema, or clubbing. Laryngeal examination revealed complete paralysis of the left vocal cord. The arm to tongue circulation time (tested with dehydrocholic acid) was 27 seconds. The chest roentgenogram revealed enlargement of the left ventricle. Fluoroscopic examination of the mediastinum revealed no apparent cause for pressure on the left recurrent laryngeal nerve. The patient was fully digitalized, with prompt relief of dyspnea.

After discharge from the hospital on Aug. 30, he continued to take digitoxin for seven months, without shortness of breath. His hoarseness began to diminish at the time of discharge and completely disappeared five weeks later. Seven months after he left the hospital he stopped taking digitoxin on the advice of his family physician. Six weeks prior to his second admission, on July 29, 1951, he began to notice gradually progressive weakness, orthopnea, and nocturnal dyspnea and his hoarseness returned. He consulted a laryngologist, who found "inflammatory laryngitis" and referred him to an internist, who advised hospitalization.

Physical examination on the second admission revealed the patient to be dyspneic and in obvious respiratory distress. There was no distention of the neck veins. There were moist rales at both lung bases, with some dulness and diminished breath sounds at the base of the right lung. The blood pressure was 130/90. The pulse was regular, with a rate of 96. The heart sounds were distant, and no murmurs were heard. The liver edge was palpable 2 cm. below the costal margin and was not tender. There was no ankle edema. Laryngeal examination revealed complete paralysis of the left vocal cord.

Results of routine hematologic studies, including the sedimentation rate, were normal. Urinalysis and serologic studies disclosed no abnormalities. The serum cholesterol was 341 mg. per 100 cc. The plasma chlorides were 95 mEq. per liter, and carbon dioxide was 45 vol. %. The basal metabolic rate was +1%. A chest roentgenogram revealed obliteration of the right costophrenic angle, with a uniform density over the lower third

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From the Medical Service, Winter Veterans Administration Hospital.

[1] Erlanger, H., and Levine, S. A.: Atrial Septal Defect: A Report of 2 Cases in Which There Was Recurrent Laryngeal Nerve Paralysis, Am. Heart J. **26**: 520-527, 1943.

[2] Dolowitz, D. A., and Lewis, C. S.: Left Vocal Cord Paralysis Associated with Cardiac Disease, Am. J. Med. **4**: 856-862, 1948. Fetterolf, G., and Norris, G. W.: The Anatomical Explanation of the Paralysis of the Left Recurrent Laryngeal Nerve Found in Certain Cases of Mitral Stenosis, Am. J. M. Sc. **141**: 625-638, 1911.

[3] King, F. H.; Hitzig, W. M., and Fishberg, A. M.: Recurrent Laryngeal Paralysis in Left Ventricular Failure, Am. J. M. Sc. **188**: 691-697, 1934.

of the right lung. The heart appeared enlarged to the left, and there was some widening of the aortic arch. A chest roentgenogram on Aug. 10 revealed clearing of the right lung base. Electrocardiograms revealed left bundle branch block. The arm to tongue circulation time (tested with dehydrocholic acid) was 30 seconds; ether circulation time was 9 seconds. The vital capacity was 4.6 liters (99% of normal).

Fluoroscopic examination revealed moderate elongation of the left ventricular inflow and outflow tracts, with some hilar congestion and moderate posterior bowing of the esophagus by enlargement of the left auricle. An unusually low aortic arch was seen in the left anterior oblique view to encroach on the aortic window close to the left pulmonary artery.

Digitoxin was again given to toxicity levels, because of the slow decrease in circulation time and the persistent pulmonary basal rales. Weakness, orthopnea, and nocturnal dyspnea improved within a few days after digitoxin therapy was started. However, dyspnea on exertion persisted. At the time of discharge on Oct. 27, his hoarseness had begun to improve.

Sudden death, presumably a result of coronary occlusion, occurred Jan. 2, 1952. No autopsy was performed.

COMMENT

The primary cardiac strain in patients with arteriosclerotic heart disease is nearly always on the left ventricle, in contrast to the primarily left auricular strain imposed by mitral stenosis. Only when the arteriosclerotic left ventricle fails does increased pressure occur in the left auricle and pulmonary vessels. The right ventricle does not, as in patients with mitral stenosis, enjoy a prolonged period of adjustment to the increased strain. When failure of the left ventricle occurs, it is apt to be of relatively rapid onset, or intermittent in character, as in paroxysmal nocturnal dyspnea or exertional dyspnea, with sudden imposition of strain on the right ventricle.

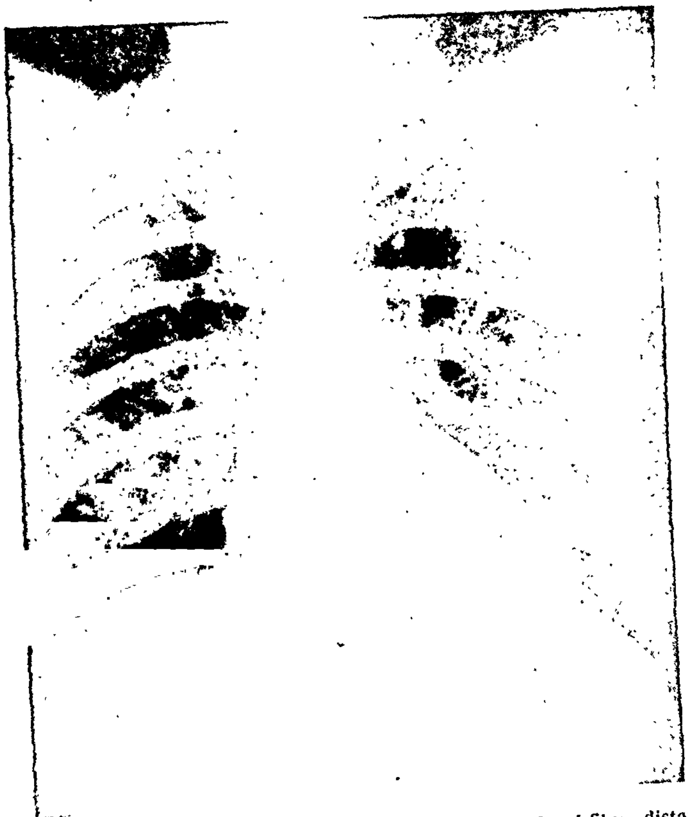

Fig. 1.—Posteroanterior view of the chest, 6-ft. focal-film distance. Note the low position of the aortic knob, elongation of the left ventricular outflow tract, and pulmonary congestion.

The extreme degree of chronic pulmonary congestion often seen with mitral stenosis is rare in patients with arteriosclerotic heart disease. The right ventricle fails more rapidly in the latter, with consequent relief of pulmonary hypertension. If dynamic dilatation of the left pulmonary artery is the common basis of recurrent laryngeal nerve compression, one would not, therefore, expect its occurrence other than rarely in patients with arteriosclerotic heart disease.

In our patient, left ventricular failure had been continuous, though varying in degree, for nearly four years. At no time had right heart failure appeared. In this respect he resembles patients with mitral stenosis, in having had prolonged pulmonary congestion and right heart strain. This is evident not alone in his symptomatic history and physical examination findings, but also in his circulation times, which had persistently shown relatively normal right heart (arm to lung) times with markedly prolonged left heart (lung to tongue) times. Digitalization even to toxicity, low-salt intake, and treatment with mercurial diuretics had not relieved entirely his left heart failure; insofar as relief was achieved, the hoarseness and vocal cord paralysis cleared repeatedly.

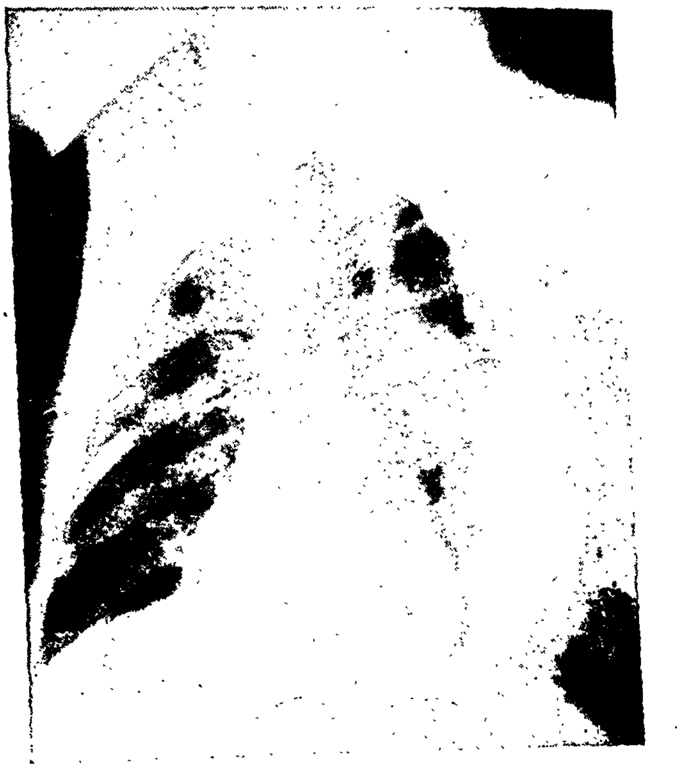

Fig. 2.—Left anterior oblique view of the chest, 6-ft. focal-film distance. Note the low position of the aortic arch, almost completely obliterating the usual "aortic window" between it and the pulmonary artery. The inflow tract of the left ventricle is elongated and fails to clear the spine.

An additional factor in the recurrent laryngeal nerve compression in this case may well have been the unusually low position of the aortic arch in relation to the left pulmonary artery. This was evident in the low position of the aortic knob seen on the roentgenogram (fig. 1) and was confirmed on fluoroscopic examination. The low position of the transverse portion of the aortic arch was seen to reduce sharply the vertical diameter of the "aortic window" in the left anterior oblique view, very nearly obliterating it (fig. 2). Very little pulmonary artery dilatation would, in this circumstance, suffice to compress the recurrent nerve against the aorta.

SUMMARY

The rare occurrence of left recurrent laryngeal nerve paralysis with arteriosclerotic heart disease is reported and its pathogenesis discussed. It is suggested that an unusually low transverse aorta encroaching on the "aortic window" was partially responsible for the nerve compression in the case reported.

for two more days. A total of 14 capsules had been given. About a week later, (April 19 or 20) the man again had taken 1 capsule every four hours for 4 doses.

On April 22, jaundice had been noted and four days later the patient had been admitted to his local hospital. In the history taken at that time it was recorded that he had "become ill a week ago after eating a ham salad sandwich, and had nausea, vomiting and vertigo." (This was the time of his second course of chloramphenicol.) Following the episode related in the quotation, the nausea and anorexia had persisted, along with the developing jaundice.

Results of laboratory work done at his local hospital were reported as follows:

The hemoglobin value was 99.5%. There were 5,190,000 erythrocytes and 5,850 leukocytes. A differential leukocyte count revealed 45% lymphocytes, and 54% neutrophils. The icterus index was 29.2. A van den Bergh test revealed 3.8 mg. of bilirubin per 100 cc. by direct reaction. The prothrombin level was 79%. The sedimentation rate was 12 mm. per hour (Westergren). Tests of stools for occult blood were negative. Serologic tests for syphilis were negative. Gastrointestinal roentgenograms revealed no gross abnormalities, and the comment was "liver and spleen shadows appear within normal limits."

In his referring letter the attending physician commented: "Our impression was that he had a toxic hepatitis." The patient stated "the jaundice cleared 100% in three days" and said that he had felt well thereafter.

The man came to the Mayo Clinic for examination and was admitted on the date given at the start of the report of this case, about two weeks after his dismissal from his local hospital. The only abnormalities found on physical examination were a few petechiae where his shoulders had rubbed on the back of the seat of the automobile while traveling, petechiae about the waist where his belt had rubbed, and other petechiae on the legs and ankles. The patient had not noticed the petechial spots. Laboratory work at this time gave the following results:

Urinalysis revealed red blood cells, grade 2. The hemoglobin level was 13.4 gm. per 100 cc., and there were 4,400,000 erythrocytes and 5,800 leukocytes. The differential leukocyte count revealed 49.5% lymphocytes, 2% monocytes, 47.5% neutrophils, and 1% basophils. There were 18,000 thrombocytes. The report on the blood smear was "Marked thrombocytopenia; some increase in lymphocytes; no immature forms were seen." There was no serum bilirubin on the direct test, with a level of 1.2 mg. per 100 cc. revealed by the indirect test. The flocculation test for syphilis was negative. The prothrombin time was 19 seconds. In a liver function test with sulfobromophthalein (bromsulphalein®) there was no dye retention in one hour.

A chest roentgenogram revealed no abnormalities. A gallbladder roentgenogram showed a normally functioning gallbladder. Volumetric study of aspirated bone marrow material revealed only a 1% nucleated cell layer. The report of smears of bone marrow read, "All elements are reduced. Megakaryocytes are virtually absent. The majority of the cellular elements are lymphocytes and plasmacytes." The description of fixed sections of the bone marrow stated that there were "small units which show the structure of a hypocellular marrow. Lymphocytes are the predominant cell."

In our opinion aplastic marrow was developing in this case, and the patient was dismissed with the advice to be under constant observation at home and to receive transfusions as he might need them. During the period June 25 to 30 inclusive, laboratory findings at the patient's home were essentially the same as those just noted. In this same period he received transfusions of 3 pints (approximately 1,500 cc.) of blood.

On July 1, the man was seen at another medical center and the physicians there supplied the following data: There were 2,400,000 erythrocytes, and 4,000 leukocytes. On a differential leukocyte count there were 55% lymphocytes, 5% monocytes, and 40% neutrophils. On blood smears the cells were qualitatively normal in appearance. Sternal aspiration revealed "markedly acellular bone marrow with only a rare normoblast and no megakaryocytes seen." There was, however, a "fair representation of myeloid cells." In view of these findings, it was suggested that cortisone might be used in addition to treatment by transfusion.

Following the patient's return home, bleeding continued, and he was admitted in emergency condition to another hospital on July 28. His attending physician and the staff pathologist have reported the findings of his final hospitalization. Physical examination disclosed the presence of multiple petechiae and ecchymotic lesions of the skin, as well as multiple areas of bleeding and ulceration in the mouth, including the gums. There was tenderness of the left lower extremity. (This raises the question whether there was hemorrhage into the muscles.)

Results of laboratory work done on July 28 were reported as follows:

The hemoglobin level was 9.2 gm. per 100 cc. There were 2,850,000 erythrocytes, and 2,400 leukocytes, with 96% lymphocytes, 2% monocytes, and 2% neutrophils. There were 19,000 thrombocytes. The coagulation time was seven minutes and four seconds, and the bleeding time was two and one-half minutes (?). The prothrombin time was 68% of normal.

The patient had moderate elevation of temperature while in the hospital. Terminally Cheyne-Stokes respiration developed, and the man died four days after his admission. Significant findings at necropsy included considerable blood in the subarachnoid space, as well as evidence of hemorrhage in other organs. The liver, spleen and lymph nodes appeared grossly normal. Microscopically, the structure of the liver and the spleen seemed undisturbed. Concerning the bone marrow, the report was, in part, as follows: "Its cellularity varies considerably in different areas. In some, the marrow is found to be quite cellular while, in others, it is largely fatty. We fail to see megakaryocytes. Erythroid and myeloid elements also seem to be diminishing."

Case 3.—This white girl, 3 years of age, was admitted on March 19, 1951, because of severe bleeding. She had had repeated infections of the upper respiratory tract but otherwise her past history and family history were not remarkable. Three siblings were living and well.

About five weeks before admission she had had acute pharyngitis and had been treated with "about 20 capsules of chloromycetin® during the three weeks" before the onset of her hemorrhagic tendency. Two weeks before admission, petechiae and ecchymotic areas had developed on the body. "Some penicillin and vitamin K" were given at this time. Severe and persistent epistaxis had appeared and repeated transfusions of blood, totaling 1,250 cc. had been administered. Studies of blood and bone marrow had indicated that the girl had hypoplastic anemia.

At the time of admission to the Mayo Clinic, the patient was acutely ill; her temperature was 103 F. Physical examination revealed multiple petechiae and ecchymotic areas and the girl was actively bleeding around the nasopharyngeal pack, which was in place at the time of her admission. Adenopathy or other abnormalities were not noted.

Laboratory studies made while she was in hospital in Rochester revealed the following:

The results of urinalysis were normal. The hemoglobin level decreased from 15.5 to 9.2 gm. per 100 cc. The erythrocytes decreased from 5,030,000 to 2,710,000, and the leukocytes from 1,700 to 600, with lymphocytes increasing from 95 to 97%, monocytes from 1 to 3%, and the neutrophils from 1 to 2%. The thrombocytes varied from 5,000 to 22,000. There were 0.1% reticulocytes. Blood smear revealed marked neutropenia and thrombocytopenia. The blood urea level was 32 mg. per 100 cc. The prothrombin time was 21 seconds. A chest roentgenogram revealed no abnormality. Volumetric study of aspirated bone marrow material revealed a myeloid-erythroid layer of only 1%.

The patient was in the hospital for nine days. The course of the illness was stormy, and her temperature ranged from 99 to 104 F. Treatment consisted of four blood transfusions of 250 cc. each. By mouth, each day, the patient received 750 mg. of aureomycin, as well as 100 mg. of cortisone. She died on March 27, from massive gastrointestinal hemorrhage.

Necropsy revealed markedly hypoplastic bone marrow, with little evidence of hematopoiesis. There was a relative increase in reticuloendothelial cells and lymphocytes. Hemorrhages were numerous and diffuse in many viscera, and moniliasis of the esophagus, stomach, spleen and kidneys was observed.

CASE 4.—This white girl, 5 years of age, was admitted on Dec. 6, 1951, with a chief complaint that purpura had been present for four weeks. A subordinate complaint was of chicken pox of four days' duration. The girl had had the usual infections of childhood. Her past history and her family history were not remarkable.

Four weeks before admission, bruises, which were unusual for her, had appeared. About a week later noticeable irritability had developed, and she had begun to have spontaneous epistaxis. By the time another week had passed, definite petechiae had appeared. Rutin and vitamin K had been given at this time. During this period, the patient's 8-year-old brother had had chicken pox and, on Dec. 2, the patient and her 6-month-old brother both had displayed the first signs of chicken pox. By the evening of Dec. 3, the vesicles of chicken pox had appeared on the girl's body and had become hemorrhagic. Meanwhile her temperature became elevated. The temperature ranged from 103 to 105 F until the time of admission to the hospital in Rochester. On Dec. 3, she had received aureomycin, 150 mg. every four hours, and 300,000 units of penicillin twice a day. On Dec. 4, she had vomited blood-streaked material and administration of aureomycin had been discontinued. The daily dose of penicillin had been increased to 1,000,000 units on Dec. 5.

On Dec. 4, the concentration of hemoglobin had been 11.6 gm. per 100 cc. of blood. Erythrocytes had numbered 3,360,000 and leukocytes 1,240 per cubic millimeter of blood.

The patient had received 18 capsules of chloramphenicol of 50 mg. each in late August, 1951. In October of the same year, also, she had been given *p*-benzylphenyl carbamate ("diphenan"), 0.25 gm. three times a day for seven days, for pinworms.

On physical examination in Rochester, the child appeared to be alert but acutely ill. A severe, hemorrhagic, vesicular rash extended "from head to toe." The lesions varied from ordinary maculae to hemorrhagic vesicles, 3 mm. in diameter, with numerous associated petechiae and ecchymotic areas. The gums were bleeding, and there were numerous small areas of hemorrhage and ulceration in the mouth and pharynx. Adenopathy worthy of remark and hepatosplenomegaly were not found. The temperature was 103 F and a pulse rate of 110 per minute was charted.

Laboratory findings were as follows: The urinalysis revealed no abnormalities. The hemoglobin level was 8 gm. per 100 cc. There were 3,080,000 erythrocytes, and 3,200 leukocytes, with 91% lymphocytes, 2% monocytes, and 7% neutrophils. There were 41,000 thrombocytes. The bleeding time was 14½ minutes. The sedimentation rate was 72 mm. per hour (Westergren). There were from 0.3 to 1.1% reticulocytes.

The report on the blood smear read, "Left shift and toxic changes in neutrophils seen. Moderate number of plasma cells with a few atypical lymphocytes. Very rare platelet seen. There is no myeloid immaturity found." Bone marrow obtained by sternal aspiration revealed, on volumetric study, no demonstrable myeloid-erythroid layer. In smears of the bone marrow there were an occasional normoblast and myeloid element. No megakaryocytes were seen, but a few platelets were encountered. The predominant cells were adult lymphocytes. Material for study of fixed sections was not obtained. A chest roentgenogram, made with portable apparatus, revealed conditions that were within normal limits.

The course of the illness for the 14 days while the patient was in hospital was satisfactory. Four blood transfusions and daily penicillin therapy were given for support. Meanwhile, the lesions of chicken pox cleared and the temperature fell to normal. The concentration of hemoglobin could not be maintained, however; after it had been raised from 7.2 gm. to 11 gm. per 100 cc. of blood, by means of transfusions, it was back to 8.4 gm. at the time of dismissal. The other blood values, on repeated reexamination, stayed essentially within the same range as those in the initial studies.

In the interval of four weeks while the patient was away from the clinic, she received two blood transfusions, one of 200 cc. and the other of 250 cc. When she returned on Jan. 22, 1952, the only remarkable features discovered on physical examination were many petechiae and ecchymotic areas, especially on the legs.

Laboratory work on this occasion gave the following results: The hemoglobin level was 9.4 gm. per 100 cc. There were 3,010,000 erythrocytes, and 4,800 leukocytes, with 65.5% lymphocytes, 12.5% monocytes, 21% neutrophils, and 1% eosinophils. There were 1% reticulocytes and 35,000 thrombocytes. The blood smear was said to be "essentially same as before."

On Feb. 19, the patient was again brought back to the clinic. In the interval during which she had been away, three more transfusions had been administered. Results of physical examination were negative except for the evidence of purpura.

Laboratory findings were as follows:

The hemoglobin value was 10.5 gm. per 100 cc. The erythrocyte count was 3,510,000 and the leukocyte count 3,700. A differential leukocyte count showed 87% lymphocytes, 6% monocytes, and 7% neutrophils. There were 0.5% reticulocytes and 27,000 thrombocytes. The report on blood smear stated: "Nothing changed over previous reports." Studies of bone marrow obtained on sternal aspiration gave the same results as on the previous occasion.

The final episode of the illness of this patient began on April 1 and terminated 24 hours later with death from cerebral hemorrhage. Her condition had remained essentially unchanged since we had last seen her; she had been active and had felt well most of the time. At 3 a. m. she awoke with a headache and about one and a half hours later started to vomit blood. Throughout the day hematemesis recurred, and, by evening, further signs of cerebral bleeding were developing. She had repeated convulsions until she died.

CASE 5.—This child was not our patient but was a playmate of the girl in case 4. We feel that a brief report of her case should be included in this series for reasons that will become apparent. The physician in the two cases was the same, and he provided the data in this case as well as those parts of the data in case 4 that were gathered elsewhere than in Rochester. The illnesses of the two little girls ran nearly identical courses.

This patient (case 5) received a total of 42 capsules (50 mg. each) of chloramphenicol. These were administered in doses of 100 mg. every six hours from Oct. 13 through Oct. 19, 1951. A careful review of the case with the parents of the child verified the physician's belief that the parents gave no other medication and that the only drug both children had received was chloramphenicol; in other words, *p*-benzylphenyl carbamate was not a factor in this case (case 5).

Aplastic anemia developed, and the course of the illness was the same as that in case 4. The cause of death was cerebral hemorrhage. To quote from the letter of the physician: "Her drug (chloromycetin®) was started about six weeks after Mary's [11]—her purpura appeared on Dec. 18, about six weeks after Mary's and her death came six weeks after Mary's."

CASE 6.—A white girl, 13 years of age, was admitted to the Mayo Clinic on April 8, 1952, complaining of bleeding. The family history was essentially negative; there were three siblings, living and well. There was a personal history of measles at the age of 9 years; of mumps in June, 1951; of appendectomy in November, 1951, and of chicken pox in March, 1952.

The significant part of the history of her present illness goes back to Jan. 26, 1951, when, in her home community, chloramphenicol had been given to her father and the patient had taken 4 capsules (not at the same time) for the "flu." She had not displayed any signs of reaction.

At the end of February, 1952, 13 months later, the patient again had had the "flu." The capsules of chloramphenicol still had been in the house, unused, and the girl had been given 1 capsule at a time for 4 doses.

On March 1, the patient had undergone dental extraction and a dental penicillin-sulfonamide pack had been used. On March 26, petechiae had developed over the feet and limbs. On April 2, a "sore" had appeared on the lower lip and ecchymosis and petechiae had been generalized. On April 4, protracted epistaxis had occurred, and the girl had been admitted to a hospital where laboratory work had revealed a "leukocyte count of 2,200 and no granulocytes present." A blood transfusion of about 500 cc.

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[11] Mary is the name representing the patient in Case 4, but is not the real name of the patient.

had been given on each of the three following days (April 5, 6 and 7), and the girl had been brought to Rochester where she was admitted directly to hospital.

The patient was normally developed and nourished but petechiae and ecchymotic areas were extensive. The gums were swollen and slightly hemorrhagic, and there was an ulcerated area, 1 cm. in diameter, on the lower lip. Results of the rest of the physical examination were not remarkable. The spleen was not palpable, and adenopathy was not noted. The girl's temperature was 101 F.

Laboratory findings were as follows:

Urinalysis revealed no abnormalities. The hemoglobin level was 14.6 gm. per 100 cc. There were 4,030,000 erythrocytes and 1,400 leukocytes. Differential leukocyte count revealed 94% lymphocytes, 2% monocytes, 2% neutrophils, and 2% eosinophils. There were 51,000 thrombocytes and 0.1% reticulocytes. The bleeding time was 15½ minutes, and the prothrombin time 17 seconds.

The report of the blood smear read, "Only one granulocyte is seen and no immature forms of any type of cell are found. The platelets are considerably reduced on the preparation." Aspirated bone marrow, on volumetric study, yielded a myeloid-erythroid layer of only 1%, while in all smears of bone marrow small lymphocytes predominated. There were some plasmacytes, reticuloendothelial cells, and a rare myeloid-erythroid element among the lymphocytes. In fixed sections there was only fatty marrow containing an occasional nest of cells.

During the period of hospitalization, the course of the illness was septic and the temperature ranged from 100 to 105 F. Acute otitis media yielded material from which Escherichia coli were cultured. After studies to detect sensitivity had been made, the patient was given 0.5 gm. of dihydrostreptomycin intramuscularly twice a day, in addition to 1,000,000 units of penicillin (duracillin®) daily, to protect her in her agranulocytic state. She also received 18 blood transfusions of 500 cc. each, given as they were indicated for bleeding. Cortisone also was given in increasing doses, to 300 mg. daily (intramuscularly), then the quantity was reduced to a maintenance dose of 100 mg. (50 mg. twice a day). When it became evident that the girl's condition was not improving with the help of cortisone, corticotropin was employed and its use was continued for the last four days of her life.

Throughout the 22 days that the patient was in hospital, leukocytes varied in number from 600 to 1,500 per cubic millimeter of blood, 99 to 100% of which were lymphocytes. Thrombocyte counts continued to be below bleeding levels at all times, ranging from 16,000 to 35,000 per cubic millimeter of blood. The cause of death was hemorrhagic pulmonary edema and gastric hemorrhage.

At necropsy, significant findings were: widespread hemorrhage of the viscera, focal necrosis and organizing bronchopneumonia of the right upper pulmonary lobe and terminal hemorrhagic edema of the lungs. Microscopic examination disclosed severe hypoplasia of the bone marrow, with little or no evidence of hematopoiesis.

Case 7.—A 36-year-old man was admitted to the Mayo Clinic on Feb. 25, 1952, because of a diagnosis, made elsewhere, of hypoplastic anemia with thrombocytopenic purpura. His own past history was not remarkable. His father had Raynaud's disease and diabetes. A sister had died with leukemia, and he had six siblings living and well.

The history of his present illness dated back to June, 1951, when he had had the "flu." He had received penicillin, which had given him hives. In July of the same year, Raynaud's phenomenon had developed in his hands. He had been hospitalized in October for 14 days with pneumonia and had received aureomycin for 10 days and chloramphenicol for 3 days. "His leukocyte count was 4,500 (per cubic millimeter of blood) but the remaining blood picture was normal"; this is the only hematological information we have concerning that illness.

Left cervicothoracic sympathectomy had been performed on Nov. 6 for the persistent Raynaud's phenomenon. Postoperative pleural effusion had developed, and again he had been given chloramphenicol and aureomycin. On Jan. 10, 1952, right cervicothoracic sympathectomy had been performed, at which time the concentration of hemoglobin had been found to be 9.2 gm. per 100 cc. of blood. He had had considerable trouble with bleeding from the nose, the gums, and the wound. Again he had received postoperative treatment with chloramphenicol and aureomycin.

On Jan. 22, he had been readmitted to the hospital with purpura, that is, he had had petechiae and ecchymosis, epistaxis, and hemorrhages into the muscles of the thigh. A diagnosis of thrombocytopenic purpura had been made. The thrombocyte counts then had been 15,000 and 25,000, while studies of the bone marrow had revealed the presence of hypoplastic anemia. Corticotropin had been administered, without benefit, for about 20 days. He had received numerous blood transfusions.

On Feb. 25, the date that was mentioned at the beginning of the report of this case, the patient entered a hospital in Rochester. Laboratory data at this time were as follows:

Urinalysis revealed no abnormalities. The hemoglobin level was 10.2 gm. per 100 cc. There were 3,350,000 erythrocytes and 2,600 leukocytes. The differential leukocyte count revealed 64% lymphocytes, 5% monocytes, 30% neutrophils, and 1% basophils. There were 0.1% reticulocytes and 50,000 thrombocytes.

A blood smear showed leukopenia with toxic neutrophils and marked thrombocytopenia. The bleeding time was more than 30 minutes. The sedimentation rate was 84 mm. per hour (Westergren). Aspirated bone marrow, on volumetric study, yielded a myeloid-erythroid layer of 2%, while study of smears of bone marrow revealed an occasional toxic granulocytic cell, a few normoblasts, but no megakaryocytes. The patient was dismissed five days after his admission with advice to continue with supportive blood transfusions. By correspondence we learned that he died six weeks after his dismissal.

Case 8.—This white woman, 70 years of age, was admitted to the Mayo Clinic for the fifth time on April 22, 1952. We first had seen her in 1939, when she was 58 years of age. At that time her chief complaint was symptoms of nervous tension. There also was a history of chronic discoid lupus erythematosus with onset in 1932, when the woman had been 51. The lesions had been confined to the scalp and face and had been well controlled by administration of quinine. In 1939, the woman weighed 175 lb. (79.4 kg.), and she had scars from past activity of her lupus erythematosus. Results of physical examination otherwise were not remarkable. Laboratory examinations at this time revealed values within normal ranges.

The woman came to the clinic for a second examination on April 21, 1944, at the age of 62 years. At this time her most significant complaint was urinary frequency of six months' duration, and she had been told that the urine contained pus, casts, and albumin. There were still evidence of her state of nervous tension. She weighed 166 lb. (75.3 kg.) and had alopecia and a blood pressure of 140 mm. Hg systolic and 82 diastolic. Examination of the eye grounds revealed incipient cataracts, grade 2 sclerosis, and grade 1 narrowing of the retinal arterioles, together with multiple, small retinal hemorrhages.

Results of laboratory studies made at this time were reported as follows:

The urine contained albumin, grade 1 to 2, red blood cells, grade 3 to 4, and pus, grade 1. The hemoglobin level was 11.2 gm. per 100 cc. There were 3,490,000 erythrocytes and 6,800 leukocytes. The flocculation test for syphilis was negative. Roentgenograms of the thorax and of the kidneys, ureter, and bladder revealed no abnormalities. The blood urea level was 72 mg. per 100 cc., and the creatinine level 2.3 gm. The urine culture was negative. Excretory urogram and cystoscopic examination revealed no abnormalities. A final diagnosis was made of chronic glomerulonephritis with renal insufficiency.

Our third experience with the patient was in November, 1944, when she was seen at home by one of our staff members in consultation with her home physician. At this time she was convalescing from bronchopneumonia and gave evidence of renal insufficiency and urinary infection. A prescription was given for sulfathiazole, 0.5 gm., three times a day, and a supportive regimen was advised.

She returned to the clinic on Jan. 3, 1949. In the five year interval her condition had not greatly changed. She did complain anew,* however, of very troublesome epigastric distress and increase in exertional dyspnea associated with some substernal distress. Three weeks before this visit she had been awakened with pain in the jaws and in both arms, for which she had been hospitalized. This distress had been recurring about five minutes after meals. Her blood pressure was 155 mm. Hg systolic and 86 diastolic. She was still obese but, otherwise, nothing remarkable was found on physical examination.

Laboratory work on this occasion disclosed the following:

The urine contained albumin, grade 1 to 3, and pus, grade 1 to 2. The specific gravity was 1.003 to 1.010. The hemoglbin level was 15.9 gm. per 100 cc. The erythrocyte count was 4,700,000 and the leukocyte count 7,700. The blood urea level was 58 to 84 mg. per 100 cc., and there was 7.3 mg. of sulfate per 100 cc. The *sedimentation* rate was 36 to 43 mm. per hour (Westergren). A roentgenogram of the stomach revealed "esophageal hiatal hernia with the cardiac third of the stomach above the diaphragm."

The patient was in the hospital for 37 days on a reduction regimen and conservative treatment for the diaphragmatic hernia was advised. In March, 1952, she had had increasing distress in the thorax and arms while at home. Her erythrocyte count had been 2,400,000 per cubic millimeter of blood and the value for hemoglobin, 9.2 gm. per 100 cc. of blood. Occult blood had been found in the stools. Treatment for peptic ulcer and repeated transfusions of 250 cc. of blood had been given. Results of laboratory work the next week had been reported as follows: There were 2,080,000 erythrocytes and 2,300 leukocytes. The hemoglobin level was 8.2 gm. per 100 cc. There were 65% lymphocytes. There were 43,000 thrombocytes. The stool contained occult blood. The sedimentation rate was 131 mm. per hour (Westergren). Studies of bone marrow had revealed "a hypoplastic marrow with an increase in plasmacytes."

The patient had received five transfusions, after which her erythrocyte count had been 3,200,000 per cubic millimeter of blood and the value for hemoglobin was 9.1 gm. per 100 cc. of blood. On April 22, 1952, as has been said, the patient returned to the clinic for further study, especially regarding the previously reported findings referable to the bone marrow. The pains in the thorax, jaw and arms were about the same as on previous visits. The patient repeated the nontechnical language in which her condition had been explained to her, saying that she had been told that she had a "weak bladder that did not empty well" (residual urine: 350 cc.) and that for the resultant urinary infection she had been receiving chloramphenicol. This drug had been given off and on for two years and, in the last six weeks, the dosage, while variable, "averaged 2 or 3 capsules a day, four or five days a week."

She was still moderately obese (150 lb. [68 kg.]) was pallid, and possibly was slightly icteric. The remainder of the physical examination was not productive of any evidence worthy of remark. Laboratory examinations revealed the following:

The urine contained albumin, grade 2, and pus, grade 1. The hemoglobin value was 11.3 gm. per 100 cc. There were 3,310,000 erythrocytes and 3,800 leukocytes, with 31% lymphocytes, 2% monocytes, 65% neutrophils, and 3% eosinophils. There were 1.1% reticulocytes and 65,000 thrombocytes. The report of the blood smear read, "Platelets markedly reduced, moderate leukopenia, anisocytosis without much polychromasia." The total protein level of the blood was 7.47 mg. per 100 cc. The albumin-globulin ratio was 3.91:3.56. The blood urea level was 106 mg. per 100 cc., creatinine 2 mg., and sulfate 8.2 mg. The sedimentation rate was 131 mm. per hour (Westergren). There was no bilirubin on the direct test, but the indirect test revealed 1.4 mg. per 100 cc. of serum. Serologic tests for syphilis were negative. The lupus erythematosus test was negative.

Cystoscopy revealed "moderate urethritis and cystitis cystica over the base." A chest roentgenogram revealed no abnormali-ties. A gastric roentgenogram showed a "hiatal hernia with about one-third of the stomach above the diaphragm." Bone marrow (sternal aspiration) on volumetric study yielded 3% fat, 67% plasma a myeloid-erythroid layer of 1% and 29% erythro-cytes. Examination of bone marrow smears revealed "a hypo-cellular marrow with lymphocytes being the predominating cell. There are a few granulocytes present, only an occasional normoblast seen and no megakaryocytes found." Fixed tissue sections of the marrow disclosed aplasia of the bone marrow, with replacement by fatty tissue.

The patient's course in the hospital for 22 days was relatively quiet, since she had neither active hemorrhage nor agranulocytic complications. The anemia, however, was troublesome. The concentration of hemoglobin fell from 11.3 gm. to 9.7 gm. to 8.8 gm. per 100 cc., on a weekly basis, while the reticulocytes ranged between 1% and 1.5%. Three blood transfusions, of 500 cc. each, were administered and the concentration of hemoglobin was 13.4 gm. per 100 cc. of blood at the time of dismissal. Cortisone was administered for two weeks without any evidence of benefit, and the woman was returned to the care of her family physician, who was to give transfusions as indicated. Four weeks after her return home she had continued on the same course, requiring transfusions to combat her anemia but otherwise doing well.

CASE 9.—In 1926, this white woman had received a diagnosis of chronic, discoid lupus erythematosus. She had been given multiple treatment, including quinine, ultraviolet light and colloidal gold. To the last of these she had reacted unfavorably. In the autumn of the year in which the diagnosis had been made, she came to the Mayo Clinic, where she spent 58 days in a Rochester hospital. On Oct. 19, 1926, results of laboratory work were reported as normal except that evidence of mild hypochromic anemia was found.

On March 12, 1928, because of a flare-up of the lupus erythematosus, the patient was readmitted to the Rochester hospital, where she remained for 49 days. At that time the laboratory findings again were normal except for anemia (the hemoglobin level was 70%). The patient had no other illness until 1934, when the cutaneous lesions of lupus erythematosus flared again. This condition waxed and waned in a mild way until her next admission.

At the time of the admission just mentioned, which was in January, 1946, the woman complained of shifting joint pains of about two months' duration, with associated afternoon fevers and night sweats. The joint pains were severe enough to keep the patient in bed. There was also a history of recurring urinary infection. It was believed at this time that she had "chronic discoid lupus erythematosus with arthritis." There was evidence of pyuria. Esch. coli were cultured from the urine and sulfathiazole, 7½ grains (0.482 gm.), was given three times a day. All symptoms improved, with clearing of the urinary infection. The patient spent 110 days in the hospital on this occasion, during which time she was given, in addition to the treatment mentioned, bismuth arsphenamine sulfonate (bismarsen®) and quinine. She received, also, two transfusions of blood of 250 cc. each.

Results of laboratory work at the time of her admission in 1946 were reported as follows:

The urine contained albumin, grade 2, pus, grade 2, and Esch. coli on culture. The hemoglobin level was 10.2 gm. per 100 cc. There were 3,250,000 erythrocytes per cubic millimeter and 5,900 leukocytes, with 33% lymphocytes, 11% monocytes, 54% neutrophils, 1% eosinophils and 1% basophils. The blood smear showed a "toxic picture." Serologic tests for syphilis were negative. A chest roentgenogram revealed no abnormality. Roentgenograms of the joints showed "rheumatoid arthritis involving most of the joints examined." The sedimentation rate was 126 mm. per hour (Westergren). The serum protein level was 6.1 mg. per 100 cc. The albumin-globulin ratio was 1.4:1 mg. per 100 cc. At the time of the woman's dismissal, there were no abnormal laboratory findings other than mild elevation of her sedimentation rate.

In 1948, at the age of 60 years, she went to her local physician because of urinary frequency and, on pelvic examination, an early squamous cell epithelioma, grade 2, of the uterine cervix, was found and was cauterized. She was then referred to the Mayo Clinic for further advice and treatment. Pelvic examination at the clinic gave no evidence of extension; consequently, treatment for the carcinoma was limited to a course of roentgen rays. Examination in the section of dermatology revealed "mild lesions of chronic discoid lupus erythematosus, with no systemic reflections." Results of general examination otherwise were not remarkable. The patient had had no trouble with the arthritis in the two years since she had been at the clinic, but she had had recurring urinary infection, which had become more frequent and troublesome. Streptococcus faecalis was found in the urine. Therefore, calcium mandelate was administered and acidification of the urine was effected. Laboratory studies at this time revealed that leukocytes numbered 4,500, of which 50% were neutrophils. All other values were within normal range.

In January, 1949, the patient returned to have her pelvis reexamined. She related that she had had another attack of cystitis, which had persisted for 34 days before responding to treatment. On examination there was no evidence of local recurrence of the cervical carcinoma. No other examinations or laboratory procedures were carried out at that time.

In June, 1949, reexamination of the pelvis gave no evidence of recurrence of the carcinoma and the remainder of the physical examination also indicated that the patient's condition was satisfactory. An attack of cystitis again had occurred in May, 1949, and had been treated with a total of 9 gm. of aureomycin. There was still some evidence of urinary frequency and the "lupus erythematosus was the same." Laboratory examinations at this time disclosed nothing abnormal except pyuria. Results of cystoscopic examination were reported as follows: "Bladder capacity reduced. Moderate, subacute, diffuse cystitis and dilated vessels. Factitial cystitis." Local treatment for 10 days consisted of the use of suppositories of strong silver protein and instillations of 5% mild silver protein. Aureomycin was administered for five days. Much symptomatic improvement was brought about and the urine returned to normal.

Reexamination in November, 1949, gave no evidence of recurrence of the cervical carcinoma. The lupus erythematosus was the same, and the patient was having very little trouble with the cystitis. Laboratory studies again yielded values within normal limits. The patient returned again, in May, 1950, for her usual six months' reexamination, and no evidence was found of recurrence of the carcinoma. The discoid lupus-erythematosus was unchanged. "A severe anemia" was found and the woman was referred to the medical section for further study. The following history was obtained:

During the last month the patient had noticed excessive fatigue as well as exertional dyspnea. She had had mild epistaxis without any previous experience of such trouble. The patient also noted that she was bruising easily and ecchymotic areas appeared spontaneously on the limbs and other parts of the body. It developed that her urinary distress had recurred and that she had been given chloramphenicol; in fact, she had been taking 1 capsule of the drug (250 mg.) every night at bedtime for the last six months. The history and results of physical examination otherwise were not remarkable. Laboratory survey revealed the following:

The hemoglobin level was 11.0 gm. per 100 cc. There were 2,960,000 erythrocytes and 4,200 leukocytes, with 62.5% lymphocytes, 3.5% monocytes, 32.5% neutrophils, 0.5% eosinophils, and 1% basophils. There were 1.1% reticulocytes and 37,000 thrombocytes. The sedimentation rate was 56 mm. per hour (Westergren). The blood urea level was 28 mg. per 100 cc. There was no serum bilirubin on the direct test, and 1.2 mg. on the indirect test. Serologic tests for syphilis were negative. A blood smear revealed anisocytosis, with scattered macrocytes, some polychromasia, neutropenia, and diminished number of thrombocytes.

There was no dye retention on a sulfobromophthalein test of liver function. Analysis of gastric content showed 56 degrees of total acidity and 36 degrees of free hydrochloric acid in a quantity of 125 cc. A roentgenogram of gallbladder revealed cholelithiasis. A roentgenogram of stomach revealed no abnormalities. Volumetric study of bone marrow material aspirated from the sternum revealed 13% fat, 57% plasma, 1% myeloid-erythroid layer, and 29% blood cells. Examination of smears of marrow revealed a "hypoplastic marrow." Results of examination of fixed tissue preparations of the marrow were reported thus: "structure of a hypoplastic marrow."

Following withdrawal of chloramphenicol, the cystitis immediately flared up but local treatment with 5% mild silver protein, with tampons of strong silver protein and procaine penicillin gave relief. Treatment recommended for the hematological condition consisted of a high protein, high vitamin diet, with intramuscular injection of 15 mg. of vitamin B_{12} twice a week.

The patient's home physician supplied the follow-up data. It is now two years since we last observed her. Following her return home, hematinics and vitamin supplements were used and all other drugs were withdrawn. Treatment with cortisone was carried on for about two months, and then was discontinued for lack of evidence of benefit. No episodes of acute bleeding occurred, however, and as the months passed, the blood values slowly returned to normal.

In July, 1951, the following values were reported:

There were 4,100,000 erythrocytes and 6,000 leukocytes, with 55% lymphocytes. There were 90,000 to 160,000 thrombocytes.

In May, 1952, the following data were reported: The hemoglobin level was 12 gm. per 100 cc. The erythrocyte count was 4,700,000 and the leukocyte count 4,000 to 6,000. There were 238,000 thrombocytes. The differential leukocyte count revealed 50 to 60% lymphocytes, and 40 to 45% neutrophils. The patient has not received chloramphenicol since 1950.

Case 10.—A white woman, 18 years of age, was admitted to the Mayo Clinic on May 8, 1952, because of severe and persistent anemia, which had necessitated repeated blood transfusions. She had had what she called "chronic sinusitis," the evidence for which seemed to consist primarily of a postnasal drip. Because of this, she had been given chloramphenicol on Dec. 11, 1950. The prescription had called for 16 capsules, and she had had the prescription refilled until, by July, 1951, she had taken about 60 capsules. By the time she had taken that many capsules, she had begun to notice that she bruised easily but, she stated, she had been rather listless and tired since April, 1951. In August, 1951, she had noticed ecchymosis in areas of slight trauma and had visited her physician. Repeated studies of the blood at that time were reported to have given evidence of a satisfactory condition. For example, the hemoglobin level was 11.35 gm. per 100 cc. There were 4,180,000 erythrocytes and 5,700 leukocytes, with 38% lymphocytes, 60% neutrophils, and 2% eosinophils. There were 317,000 thrombocytes.

Two months later (November, 1951), the ecchymotic areas still had been recurring and laboratory work had revealed slight decrease in all blood values: The hemoglobin level was then 65%. There were 3,370,000 erythrocytes, 3,800 leukocytes, and 200,000 thrombocytes.

In December, 1951, petechiae had appeared for the first time and again she had consulted her physician. At this time the blood thrombocytes had decreased to 50,000 per cubic millimeter of blood while the other blood values were about the same. The hemoglobin level was 9.3 gm. per 100 cc. The erythrocyte count was 3,450,000 and the leukocyte count 3,600, with 59% lymphocytes, 3% monocytes, and 38% neutrophils. Studies of bone marrow were made at this time, and the report indicated the presence of "hypoplastic bone marrow."

In January, 1952, reexamination of the blood yielded essentially the same results as before, except that the blood thrombocytes had decreased to 21,000. Examination of the blood in February, 1952, revealed a hemoglobin level of 8.4 gm. per 100 cc. There were 3,070,000 erythrocytes per cubic millimeter and 1,900 leukocytes with 68% lymphocytes, and 32% neutrophils. At the time of this examination the hematologist reported that some of the lymphocytes were atypical. Corticotropin was given for three weeks without noticeable improvement in the blood picture, and then its administration was discontinued.

The laboratory survey in April, 1952, resulted in the following data: The hemoglobin value was 9.4 gm. per 100 cc. There were 3,450,000 erythrocytes, and 2,800 leukocytes with 70% lymphocytes, 27% neutrophils, 2% eosinophils, and 1% basophils. There were 28,000 thrombocytes.

At this time the patient had had a massive gastrointestinal hemorrhage, with syncope, requiring hospitalization and two transfusions of blood. After that time (March, 1952), she had received a transfusion of blood every two weeks to combat her anemia. Cortisone also had been given intramuscularly three times a day for seven days, but with questionable benefit. At the time of the patient's admission to the Mayo Clinic, she was pale and her blood pressure was 80 mm. Hg systolic and 50 diastolic. Her temperature was 99 F. Scattered ecchymotic areas and multiple petechiae were noted over the lower extremities.

Results of laboratory work on this occasion were reported as follows: The urine was normal. The hemoglobin level was 7.9 gm. per 100 cc. There were 2,470,000 erythrocytes per cubic millimeter and 2,600 leukocytes, with 65% lymphocytes, 9% monocytes, 25% neutrophils, and 1% eosinophils. There were 1.3% reticulocytes and 80,000 thrombocytes. The bleeding time was 17 minutes.

The report of the blood smear read "Anisocytosis with some macrocytosis, polychromasia and basophilic stippling. Platelets are reduced. Leukopenia with relative lymphocytosis. No remarkable immaturity was evident." (One normoblast was seen.) Serologic test for syphilis was negative. A Coombs' test was negative. The serum bilirubin level by the indirect test was 0.7 mg. per 100 cc., and negative by the direct test. The prothrombin time was 19 seconds. Volumetric study of aspirated bone marrow yielded a myeloid-erythroid layer of 2%, while examination of the bone marrow smear revealed "a hypoplastic marrow with decrease in the myeloid line and only a rare immature megakaryocyte seen. The normoblastic line is abnormal in that the cells are suggestive of megaloblastic changes, many with fragmentation and karyorrhexis of nuclei. These abnormal appearing erythroid elements show much mitotic activity. Fixed preparations show little nests of these cells with many mitotic figures. Plasma cells are numerous and the fixed tissue sections show a fatty, hypoplastic structure."

A study of bone marrow on the day of dismissal, May 19, 1952, revealed a myeloid-erythroid layer of less than 1%, while examination of the bone marrow smear preparations showed, "The erythroid line is about as before while the myeloid line is depressed. There is an increase in mature lymphocytes and plasmacytes. Only one megakaryocyte could be found." The patient was returned to the care of her home physician, who has continued to administer cortisone in therapeutic doses and to give transfusions of blood as her anemia dictates. Three weeks after her dismissal from our care, her condition continued to follow the same course.

POSSIBLE MECHANISM, RAPIDITY OF EFFECTS, AND INEFFECTIVENESS OF TREATMENT

In analyzing the foregoing reports, there seem to be at least three patterns. One of these suggests a sensitivity mechanism. In four cases (cases 1, 2, 6, and 7) the initial course of treatment with chloramphenicol seemed to be unattended by ill effects, whereas the second course seemed to precipitate the failure of bone marrow. In two of these four cases (cases 1 and 2) also, there were immediate drug reactions of nausea and vomiting, followed by jaundice in case 2. Three cases in which treatment was intermittent (cases 3, 8, and 10) may well fit into the same pattern, but it was impossible to obtain a history whereby ingestion of the drug and appearance of signs and symptoms of aplastic anemia could be exactly related in time. It is worthy of note that only four capsules of the drug constituted a second course in two cases (cases 2 and 6).

Acute aplasia of bone marrow was precipitated in two cases (cases 4 and 5) by a single course of chloramphenicol. However, gradual suppression of the bone marrow may be a third mechanism. In case 9 such suppression followed daily ingestion of 1 capsule of chloramphenicol for six months and recovery eventually resulted after withdrawal of the drug. Some of the cases (especially case 8) in which treatment with chloramphenicol was intermittent, might actually belong to this group.

There was a history of previous sensitivity to penicillin in two cases (cases 2 and 7), a history of reaction to gold in case 9, a history of hay fever in case 1, and a history of vasomotor rhinitis in case 10. Perhaps, in these cases, allergic diathesis existed. Such a history should be considered important when therapeutic agents must be chosen.

It is not possible to tell from these cases whether careful hematological examinations of the patients who were receiving the drug would have forestalled the tragic complication but we think not. The dosage of the drug for most of the patients who died was figured in hours rather than in days or weeks. In most cases purpura appeared about four weeks after the beginning of the precipitating course of treatment. All fatalities were attributable to uncontrolled hemorrhage.

No treatment has been of avail in these cases. It seems that cortisone, corticotropin (ACTH), and vitamin B_{12} have had fair trials without obvious effect. Had the cases not terminated fatally in such a short time, more satisfactory evaluation of such treatment might have been possible. Repeated transfusions are indicated for supportive treatment.

ADDENDUM

Two more patients who have aplastic anemia associated with ingestion of chloramphenicol have been admitted to our hospital service since this paper was submitted for publication. Both patients are men of middle age, and the purpuric manifestations, anemia and findings relative to the peripheral blood and bone marrow are similar to those of the patients whose cases have been reported here. One man initially took only 4 capsules of the drug in treatment for a sore throat. He repeated the dose both one and two months later; thus, he took a total of 12 capsules. The other man intermittently took capsules for nine months, in treatment of a chronic infection of the urinary tract, until he had taken 160 to 180 such capsules. The bone marrow of the latter patient contained distinctly more normal cellular elements than that of the former one.

Treatment of Prostatic Cancer.—Hormonal therapy is at present the treatment of choice for the great majority of patients suffering from prostatic cancer. Remarkable relief of symptoms with regression of the local lesion and of metastases has been observed in about three-fourths of the treated cases. Many, however, soon relapse.

Statistics now indicate some increase in the ultimate survival time of patients treated by castration and estrogens. The latter are usually first used in the still localized cases without metastases, reserving orchiectomy for the patients who do not respond or in the event of a later recrudescence.

Advanced carcinoma with metastases is treated by orchiectomy combined with estrogen therapy. Experiments with bilateral total adrenalectomy in the treatment of relapsed prostatic carcinoma await the judgment of time.

The only reported cures, however, have been effected by radical surgery. Nevertheless, the over-all prognosis is discouraging, since radical prostatectomy is applicable only to a small fraction of the total number of cases. This low percentage of operability is due mostly to the advanced nature of the lesion when first detected, as well as to the age and the debilitated condition of some of the patients. Effort should be made toward early diagnosis by means of periodic rectal examination. There is doubt, however, as to whether all the small so-called "occult carcinomas" accidentally discovered and cured by radical prostatectomy are indeed biologically malignant.

Preoperative hormonal therapy may render more patients amenable to radical surgery and may thus in the future lead to a higher rate of curability.—Seymour F. Wilhelm, M.D., Carcinoma of the Prostate, *The Medical Clinics of North America*, May, 1952.

CHLORAMPHENICOL AND APLASTIC ANEMIA

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Since chloramphenicol is an efficient broad spectrum antibiotic that is widely used, the possibility that it may be the cause of hematological disorders is, naturally, a serious one. Recently, five cases of fatal aplastic anemia have come to our attention. In each instance, we believe chloramphenicol to be etiologically implicated. It is our purpose to report these cases and to review the literature on hematological disorders believed to be caused by this drug.

REPORT OF CASES

CASE 1.—A 31-year-old housewife had received phenobarbital and atropine intermittently for over a year because of nervousness. In December, 1951, an upper respiratory tract infection developed, and she was given a total of 15 capsules of chloramphenicol. Early in January, 1952, she received an additional six capsules. On this occasion, urticaria developed over the lower extremities and lasted for several days. No other recent history of exposure to drugs or toxic agents could be obtained, and the allergic history was otherwise negative. About Feb. 1, 1952, she first noticed malaise and lack of energy, and on Feb. 8, petechiae appeared on her arms and legs. She was hospitalized on Feb. 15, and pancytopenia with hypocellular bone marrow was found. The patient was transferred to the Walter Reed Army Hospital on Feb. 26.

On physical examination she appeared to be in no distress, and the only conditions of note were pallor of the skin, scattered petechiae, and several ecchymoses over the arms and legs. Laboratory tests on admission showed 3,250,000 red blood cells per cubic millimeter; hemoglobin, 9.7 gm. per 100 cc.; 3,000 white blood cells per cubic millimeter, with 88% lymphocytes, 9% segmented neutrophils, 2% monocytes, and 1% eosinophils; 0.3% reticulocytes; and 20,000 platelets per cubic millimeter. The bleeding time was 43 minutes and the coagulation time, 4 minutes. A tourniquet test was highly positive. Results of urinalysis were normal, except for a trace of albumin. A roentgenogram of the chest was normal. Two examinations of aspirated bone marrow showed an extremely hypocellular marrow consisting of about 85% adult lymphocytes. The same condition was observed in marrow slides transferred from the hospital in which the patient had been previously.

The patient was given corticotropin (ACTH), 40 mg. daily administered intravenously, and large doses of vitamin B_{12}, ascorbic acid, and vitamin K. Transfusions of freshly drawn blood (using plastic bags) were given as indicated. Because of difficulty with intravenous administration, the corticotropin therapy was stopped after four days, and cortisone, 200 mg. daily given orally in divided doses, was administered. Despite treatment, the patient's course over the next two weeks was gradually downhill. She continued to bleed vaginally after the onset of her regular menstrual period on Feb. 22. Petechiae and ecchymoses became more evident. Nine transfusions in a two week period were required to keep the hemoglobin level at about 10 gm. per 100 cc. Results of blood determinations done on March 11 were 1,900,000 red blood cells; hemoglobin, 7.4 gm. per 100 cc.; 1,450 white blood cells, with 95% lymphocytes; 0.2% reticulocytes; and 4,700 platelets. In the evening of the same day, the patient complained of abdominal distention, and within an hour the blood pressure had dropped to a shock level. Despite administration of 10 units of blood during the next 24 hours, the patient remained in shock and died on March 15, after having a short convulsive seizure.

At autopsy, numerous petechial and ecchymotic lesions scattered over the body were seen. The peritoneal cavity contained about 800 cc. of fresh blood. There was a large hematoma of the left ovary, obscuring most of the ovarian tissue. On microscopic section, the blood was seen to be coming from a hemorrhagic corpus luteum. Petechial hemorrhages were scattered throughout the white matter of the brain, the heart, gastrointestinal tract, and kidneys. The bone marrow grossly appeared pale brown and on section showed marked hypocellularity, with small foci of cells, which were mainly lymphocytes, seen only occasionally. Small foci of extramedullary hematopoiesis were seen in the abdominal lymph nodes, liver, and spleen.

Comment.—This was a 31-year-old housewife who took a total of about 5.5 gm. of chloramphenicol in two courses and in whom fatal aplastic anemia developed three to four weeks later. It is of interest that urticaria lasting several days developed when the second course was administered. The patient had had no other drugs known to cause aplastic anemia within a reasonable period prior to her fatal disorder.

CASE 2.—A 23-year-old man gave a history of having had recurrent sinusitis over a period of several years. For the first time, in November, 1951, he took 24 capsules of chloramphenicol because of an upper respiratory tract infection and sinusitis, and he took the same number again in early December. At the same time he took APC (aspirin, acetophenetidin, and caffeine) tablets, codeine, phenylephrine (neo-synephrine®) hydrochloride nose drops, and "black-colored nose drops." On Dec. 10, 1951, scattered petechiae and ecchymoses developed. Over the next two months, he noticed increasing weakness and dull frontal headaches. On Feb. 6, 1952, he had a mild epistaxis and was admitted to another hospital. Extensive studies done at that time showed pancytopenia and extremely hypocellular bone marrow on repeated aspiration. The patient stated that during this hospitalization he again received about 24 capsules of chloramphenicol because of a small ulcer on his lip. Other medication at that time consisted of analgesics for headache and ascorbic acid. Although the patient had worked in an army hospital pharmacy for 11 months previously, he denied taking any other drugs. The allergic history was negative.

On admission to Walter Reed Army Hospital, physical examination showed a fair degree of pallor and scattered petechiae over the trunk and lower extremities. Laboratory data were as follows: 1,800,000 red blood cells per cubic millimeter; hemoglobin, 6.2 gm. per 100 cc.; 1,800 white blood cells per cubic millimeter, with 10% segmented neutrophils and 89% lymphocytes; 7,500 platelets per cubic millimeter; 0.2% reticulocytes; and a hematocrit of 20%. The bleeding time was more than 16 minutes, and the clotting time was 5 minutes. A tourniquet test was highly positive. Roentgenograms of the chest, sinuses, mastoids, and abdomen were normal. Urinalysis showed many red blood cells and a few white blood cells per high power field. Aspiration of sternal marrow showed it to be extremely hypocellular, with 90% adult lymphocytes.

The patient was given cortisone, 300 mg. per day administered orally in divided dosage, vitamin B_{12}, ascorbic acid, and fresh blood transfusions as indicated. He was given penicillin, aureomycin, or terramycin during most of his hospital stay. Ten pints of blood were required to maintain the hemoglobin level at about 10 gm. per 100 cc. Gastrointestinal bleeding, high fever, jaundice, and pneumonitis developed terminally, and the patient died on May 4, 1952.

At autopsy, the skin of the entire body was moderately icteric and covered with petechiae and ecchymoses. Scattered throughout the myocardium, lungs, liver, spleen, and kidneys were large numbers of round soft white nodular areas 4 to 5 mm. in diameter. The entire gastrointestinal tract, from stomach to anus, showed myriads of similar mucosal nodules with ulcerated punched out centers. On microscopic section, these areas were observed to be large colonies of bacteria and fungi, which on culture were found to be hemolytic Staphylococcus aureus and Candida. Surrounding the colonies there was practically no in-

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flammatory reaction, although some necrosis was present, particularly in the liver. The lungs showed, in addition, diffuse hemorrhage and hemorrhagic infarction. There was a diffuse organizing pneumonitis. The bone marrow grossly appeared light tan in color; on section there was moderate hypoplasia, with virtual absence of granulocytic elements and megakaryocytes. The cells consisted mainly of lymphocytes and plasma cells, with a few nucleated red blood cells. Foci of extramedullary hematopoiesis were found in the liver, lymph nodes, and spleen. The spleen also showed considerable erythrophagocytosis.

Comment.—This was a 23-year-old soldier who had taken three courses of chloramphenicol for a total dose of about 18 gm. The aplastic anemia became apparent about one week after the second course, and a third course was taken during the aplastic process. Despite therapy, he died about 145 days after onset.

CASE 3.—Furuncles developed in an 18-year-old college student in the late summer of 1951. She said she had been given chloramphenicol in August, 1 capsule every 3 hours while awake for 2 weeks. The records of her physician showed that she had received a short course in June and another in July for furunculosis, with no record of any drug being taken in August. In early October, the patient first began to notice scattered ecchymoses, which continued to recur. Late in October, she had an upper respiratory tract infection and took two more capsules of chloramphenicol without the physician's advice. In late November, she first began to notice petechiae. Blood studies on Nov. 21, 1951, showed 3,800,000 red blood cells per cubic millimeter; hemoglobin, 11.6 gm. per 100 cc.; 5,100 white blood cells per cubic millimeter with 37% segmented forms, 6% band forms, 56% lymphocytes, and 1% monocytes. The platelet count was 34,200 per cubic millimeter. The patient had another furuncle about this time, which responded to hot soaks and ammonium ichthyolsulfonate ("icthyol") ointment. Pallor, fatigability, and dizziness had been noticed about this time. The patient was admitted to the George Washington University Hospital on Dec. 8, 1951. Physical examination at that time revealed mild pallor, crops of petechiae, ecchymoses, and several mucous membrane hemorrhages in the mouth. Laboratory data revealed 9.7 gm. of hemoglobin per 100 cc., 2,700,000 red blood cells, and a hematocrit of 25%. The platelet count was 27,000, and the white blood cell count was 6,000, with 50% segmented neutrophils, 49% lymphocytes, and 1% monocytes. The bone marrow aspirated from the sternum had a fair number of cells with no megakaryocytes. Macronormoblasts were seen, and there was some increase in adult lymphocytes and plasma cells. The result of the van den Bergh test was normal, and that of Coombs' test was negative. There was no response to vitamin B_{12}, liver extract, or folic acid therapy, which was tried because of the macronormoblasts. Bone marrow aspiration repeated a week later showed a definite decrease in the number of cells and a further increase in the percentage of lymphocytes and plasma cells. The patient was started on corticotropin therapy, which later was changed to cortisone therapy, 200 mg. per day administered orally in divided doses, for ease of administration. From this time until her death on Feb. 27, 1951, a progressive aplastic anemia gradually developed, which was characterized by anemia, thrombopenia, leukopenia, and an aplastic bone marrow. Therapy was continued with large doses of cortisone (100 to 300 mg. per day given orally) throughout this time. The only apparent effect of this drug seemed to be suppression of bleeding, at least until the terminal complication. The bone marrow showed complete aplasia, with only lymphocytes and plasma cells remaining. Other treatment included testosterone, thyroid, vitamin B_{12}, folic acid, liver extract, and repeated transfusions as indicated. Five cubic centimeters of normal marrow injected into the patient's sternal cavity resulted in only a severe febrile reaction. Her course was marked by occasional furunculosis in the axillae, which responded to large doses of penicillin. Bleeding occurred into the skin and mucous membranes and on one occasion from the kidneys for three days. In early February, the patient had severe epigastric pain and several days later passed liquid reddish and black stools. Her final episode was an overwhelming melena that persisted for three days, with marked abdominal distention, and she finally died, despite continuous transfusions.

Comment.—This 18-year-old girl took chloramphenicol for recurrent furunculosis, and about five weeks later purpura developed. Over the succeeding months an aplastic anemia developed, and she finally died of overwhelming gastrointestinal bleeding. She had taken no other drugs previously. It is a point of issue whether the patient took additional chloramphenicol on her own initiative in August or whether the drug administered in July was the last she took prior to the development of symptoms of thrombopenia in early October.

CASE 4.—A 36-year-old white waitress was first admitted to the George Washington University Hospital on July 21, 1951, for treatment of an indolent ulcer on the medial aspect of her right ankle. This had developed several years previously following an injury and had resisted treatment since that time. Blood determinations at this admission were hemoglobin, 13.9 gm. per 100 cc.; hematocrit 46%; and leukocytes 10,450 per cubic millimeter; with 68% segmented neutrophils and 32% lymphocytes. The ulcer base cleared following aluminum acetate (Burow's) solution soaks, and granulation and healing finally occurred. During the patient's stay, a serologic test for syphilis was found to be positive. She received the following therapy: chloramphenicol, 500 mg. every 6 hours for 2 days, then 250 mg. every 4 hours for 26 days, and finally 500 mg. every 6 hours for 8 days, for a total of 61 gm.; sulfisoxazole (gantrisin®), 1 gm. every 4 hours for 9 days, for a total of 54 gm. (discontinued July 9, 1951); in addition, she received aluminum acetate solution compresses; pentamethonium chloride; phenylephrine hydrochloride nose drops; aureomycin "pharyngets," 1 every 2 hours from July 25, 1951, to Aug. 7, 1951; "vi-syneral" (a multiple vitamin preparation), intramuscularly administered; dihydrocodeinone (hycodan®) bitartrate, and lyocyte® (dried red blood cell concentrate) powder applied to the ulcer crater.

The patient was readmitted on Nov. 23, 1951, saying that she had continued taking chloromycetin for another three weeks (dosage unknown) following her discharge from the hospital. She also had had a short course of priscoline® (2-benzyl-2-imidazoline hydrochloride), which was discontinued because of gastrointestinal intolerance. She had last taken chloramphenicol near the middle of September. About 10 days before admission, she first noted petechiae and ecchymoses. Ulorrhea began three days before her admission to the hospital. Examination revealed an acutely ill woman with a temperature of 39.4 C (102 F). Petechiae were scattered over all the extremities, the face, and the trunk, and ecchymoses were noted on the thighs and arms. The gums were spongy, with a few small areas of necrosis. Blood determinations at that time showed 12.4 gm. of hemoglobin per 100 cc.; hematocrit of 40%, 4,300,000 red blood cells, 1,900 white blood cells, with 20% segmented neutrophils, 4% band forms, and 76% lymphocytes. Sternal puncture (and later rib puncture) revealed an extremely hypoplastic marrow infiltrated or replaced by lymphocytes and plasma cells. The platelet count was 24,720, and the bleeding time was 15 minutes. There was only slight clot retraction in 24 hours. The clotting time (3 tube Lee and White) was 15 minutes. The patient was started on terramycin and cortisone therapy. Cortisone therapy was discontinued after 11 days, because a high spiking fever developed, and it was believed a bacteremia was being masked. This was borne out by a positive blood culture of a hemolytic Staph. aureus. The patient was given large doses of terramycin, streptomycin, and penicillin. Despite antibiotic therapy, the patient's condition became increasingly toxic. Her red blood cell count, hemoglobin level, and hematocrit gradually fell, despite repeated transfusions, reaching the following low points: 7.3 gm. of hemoglobin per 100 cc., 2,550,000 red blood cells, and a hematocrit of 24%. Her white blood cell count had increased from 1,900 on admission to 7,200 during her hospital course, but the lymphocyte count remained high, and at the time of her death her white blood cell count had dropped to 950. Cellulitis of one arm, severe generalized muscular aching, bilateral chest rales, and abdominal distention developed, and the patient died, having the clinical picture of extreme toxemia.

Comment.—This was a 36-year-old woman with a chronic ankle ulcer who had a positive reaction to a syphilis test and was treated with chloramphenicol and a number of other agents. About two months after she received chloramphenicol, an aplastic anemia became apparent. About four months prior to the onset of the aplastic process, she had received sulfisoxazole and a variety of other medicaments, including aureomycin "pharyngets" (last given three and one-half months before the onset). She had evidence of thrombopenia, leukopenia, and anemia, and died as the result of an overwhelming Staphylococcus bacteremia and toxemia. This case points up the difficulty of establishing an etiological agent, since so many medicaments had been used; however, chloramphenicol was the last agent used before her admission to the hospital, and it had been taken one and one-half to two months after any of the others.

CASE 5.—A 26-year-old white woman had had eczema and acne on her extremities and face for about two years. Her most recent treatment had consisted of chloramphenicol, four 250 mg. capsules taken daily for about two months. This was discontinued on May 18, 1952, because of nausea. On May 2, during therapy, blood tests showed a hemoglobin level of 90% and 8,950 white blood cells, with 72% segmented neutrophils. The patient felt well until about June 10, when profuse vaginal bleeding developed with the onset of her menses. She continued to have hypermenorrhea and was admitted to another hospital on June 16. She said she had had fever for several days, a sore throat, and swollen glands in her neck. She had not taken chloramphenicol previously and had a negative allergic history. She had taken no other drug in the three months preceding the onset of symptoms, except for two butabarbital (butisol®) sodium tablets the day her menses began. She had taken this drug prior to the use of chloramphenicol in an undetermined amount.

Physical examination revealed a young woman who did not appear ill. There was mild bilateral cervical adenopathy, a small amount of whitish exudate on the tonsils, and a small ecchymosis on the posterior pharynx. Her temperature was 100 F, and it varied from 100 to 103 F during her stay at this hospital. Initial blood studies revealed 1,690,000 red blood cells per cubic millimeter, 5 gm. of hemoglobin per 100 cc., 3,300 white blood cells per cubic millimeter, with 50% segmented neutrophils, 46% lymphocytes, 1% monocytes, and 3% eosinophils. The platelet count was 12,500 per cubic millimeter. Petechiae on the extremities and ecchymoses at the site of needle puncture developed during this hospitalization. The patient was given three pints of whole blood, and penicillin therapy in large doses was started. She was transferred to Walter Reed Army Hospital on June 21, 1952.

Physical examination revealed several furuncles in the left axilla, scattered petechiae on the mucous membranes and extremities, a few ecchymoses, and no enlargement of the lymph nodes, liver, or spleen. Laboratory examination revealed 3,900,000 red blood cells, 12.4 gm. of hemoglobin per 100 cc., and 400 white blood cells, with 6% segmented neutrophils and 94% lymphocytes. There were 12,000 platelets. Urinalysis showed nothing remarkable except for numerous epithelial cells. Marrow obtained by sternal puncture was extremely hypocellular, the great majority of cells being adult lymphocytes, with a few plasma cells and, rarely, normoblasts and granulocytes; no megakaryocytes were seen. The diagnosis of aplastic anemia was made.

The patient was continued on penicillin therapy with large doses. It is our intention to begin administering cortisone when her infection has been controlled. Transfusions will be used as indicated.

Comment.—This is a 26-year-old woman who took 1 gm. of chloramphenicol daily for two months for treatment of acne and eczema. Twenty three days after stopping use of the drug because of nausea, menorrhagia began. The diagnosis of aplastic anemia was made because of a pancytopenia and an extremely hypoplastic bone marrow. Her course is unknown since her condition was only recently diagnosed.

COMMENT

Four other cases of aplastic anemia believed caused by chloramphenicol have been reported. It is our understanding that reports of other cases are being readied for publication. Rich and his co-workers [1] reported the case of a 63-year-old white man who was treated for a urinary infection with chloramphenicol for about 80 days, receiving a total dose of 54 gm. Generalized purpura developed while the patient was under treatment, and shortly thereafter a complete aplastic anemia appeared. The patient died 12 days after the onset of symptoms. There were several times during the course of therapy when administration of the drug was discontinued for several days and then restarted. Loyd [2] reported the death of a 28-year-old woman who had been treated intermittently for a year with chloramphenicol for an Escherichia coli urinary infection. She then received two courses of chloramphenicol about a month apart, the first of 1.75 gm. and the second, 5.75 gm. About one and one-half months later, petechiae developed, and the patient showed evidence of a full-blown aplastic anemia. Autopsy showed widespread hemorrhage and replacement of active bone marrow by fat. Harris and co-workers [3] mention two cases of aplastic anemia in an article describing the use of chloramphenicol in the treatment of 68 patients with bronchiectasis. These cases were reported in detail by Wilson, Harris, and others.[4] In each case, aplastic anemia developed during the prolonged administration of chloramphenicol (26 and 28 weeks, respectively), with total doses of 52 and 56 gm., administered 1 gm. twice weekly. Disturbance in platelet function was first noticed. One of the patients died, while the other, at the time of the report, had a hypoplastic marrow and pancytopenia five months after the onset of the toxic symptoms. Gill [5] reported two patients treated for Salmonella infection with chloramphenicol, 250 mg. given immediately and 100 mg. every 3 hours thereafter. The first patient, who had received 6.85 gm., had a drop in the white blood cell count from 11,000 per cubic millimeter, with 42% segmented neutrophils, to 10,200, with only 9% segmented neutrophils. Administration of the drug was stopped, and five days later the patient had a white blood cell count of 9,400 per cubic millimeter, with 40% segmented neutrophils. Because of this case, the next patient was watched closely; with two days' treatment, the second patient's white blood cell count went from 7,000 per cubic millimeter, with 46% segmented neutrophils, to 5,000, with 23% segmented neutrophils. Chloramphenicol therapy was stopped, and the count rose to the previous level in several days.

Volini and his co-workers [6] reported three patients in whom toxic hematopoietic effects were seen following treatment with chloramphenicol. The first of these was a young Negro boy with typhoid, who had received a total dose of 53 gm. over a period of 18 days. Prior to treatment, his blood cell counts were normal, but after discontinuance of treatment, a red blood cell count of 850,000 per cubic millimeter and a white blood cell

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5. Gill, P. F.: Agranulocytopenia Following "Chloromycetin": Report on 2 Cases, M. J. Australia **1**: 768, 1950.
6. Volini, I. F., and others: Hemopoietic Changes During Administration of Chloramphenicol (Chloromycetin), J. A. M. A. **142**: 1333 (April 29) 1950.

count of 3,200 per cubic millimeter, with 8% segmented neutrophils was found. The bone marrow showed a maturation arrest in the red cell series and a diminution of granulocyte precursors. Twenty days after stopping treatment, the values had returned to normal. Two other cases of leukopenia and neutropenia with bone marrow involvement after chloramphenicol therapy were observed. The conditions reverted to normal after several weeks.

An abstract of a paper by Lindau [7] reveals other interesting data. He gave chloramphenicol for 30 days to eight patients, seven of whom had chronic degenerative diseases. In three of these, there was an arrest of the red blood cell maturation, with an average decrease in the hematocrit of 8.6% and a definite decrease of orthochromatic normoblasts in the marrow. After these conditions returned to normal, the drug was readministered to two of the three affected patients. Again the hematocrit fell and returned to normal after administration of the drug was discontinued. Janbon and Bertrand [8] recently reported anemia complicating the use of chloramphenicol in six patients (four with brucellosis, one with typhoid, and one with tuberculosis). The total doses in these cases were 140, 120, 52, 120, 54, and 21 gm., respectively. In each patient, the red blood cell count fell by about 2 million cells per cubic millimeter during the course of therapy. Four patients responded spontaneously, one responded to vitamin B_{12} and another to folic acid. The authors felt that this anemia was of the deficiency type, with inhibition of the antianemic factor due to the action of the antibiotic on the intestinal flora.

SUMMARY

Five cases of fatal aplastic anemia in patients who had taken chloramphenicol are reported. The chloramphenicol may have been the etiological factor in the development of this disorder. The current literature is reviewed.

ADDENDUM

The patient in case 5 died after this paper was submitted for publication.

1601 Argonne Place, N. W.

[7] Lindau, W.: Effect of Chloromycetin upon Erythropoiesis, read before the American Federation of Clinical Research, Atlantic City, N. J., May, 1952.

[8] Janbon, M., and Bertrand, L.: L'anemie du chloramphénicol, Soc. méd. hôp. Paris **67**: 990, 1951.

RELATION OF HOSPITAL ADMINISTRATORS, NURSING STAFF, AND MEDICAL STAFF

Thomas P. Murdock, M.D., Meriden, Conn.

In 1947 the President of the American Medical Association, Dr. Edward L. Bortz, on the recommendation of the House of Delegates, appointed a committee to study the nursing problem in the United States, with particular reference to the shortage of nurses. The members of the original Committee on Nursing Problems were Drs. Warren Draper, Howard Gray, Leland McKittrick, and Donald Smelzer, with myself as chairman.

I think it is fair to say that at that time the other members of the committee had only a general and not an intimate knowledge of the magnitude of the problem. I know it is more than safe to say that the chairman probably knew less about the nursing problem than any man in America. He was soon to learn far more than he had planned for or gambled on.

The study was an intensive and exhaustive one. During the year, the committee met many times with representatives of the American Nurses Association, the American Hospital Association, the American College of Surgeons, and the American Medical Association. Separate meetings were held with representatives of the various organizations, and finally representatives of all of the organizations met together. In the beginning it was quickly noticeable that there was an air of suspicion on the part of the representatives of each organization toward the representatives of the others. A humorous incident took place at one of the meetings when each group sat around a large table and did not talk to the others. Later, when it was recognized that most of the problems were common to all the groups represented and that each group had an interest, that suspicion was abolished.

Soon after the beginning of the study letters began to come in from physicians and hospital administrators condemning the actions of nurses. The tone of these letters was in general severely critical of the nurses. About this time publicity was given to the study, and then the letters from nurses began to arrive. They in turn were critical of the physicians and hospital administrators and, of course, of the committee. Some of the letters were signed and some were anonymous. There were many that were critical of the committee. In substance, they said that the committee could be better occupied by investigating the shortcomings of the members of the medical profession and leaving the nurses to solve their own problems. In both instances the attitude was due to lack of understanding. Unfortunately, I believe that some such feeling still exists among physicians, administrators, and nurses. I think, however, that it is gradually being dissipated.

In 1948 the committee made its final report to the Trustees of the American Medical Association. In the report the committee recommended that nurses be divided into two classes: (*a*) professional nurses and (*b*) trained practical nurses. The professional class was to be subdivided into college-trained nurses who would serve as educators and hospital-trained nurses who would serve as supervisory personnel. The percentages of the total of all nurses in the groups recommended were as follows: 15% were college-trained nurses, 25% were hospital-

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Read at the meeting of the Catholic Hospital Association of the United States and Canada in Cleveland, on May 27, 1952.

trained nurses, and 60% were trained practical nurses. The committee also recommended that the hospital training courses for nurses be reduced to two years, and the courses for trained practical nurses to one year.

I think the greatest achievement that came as the result of the study was the ability and willingness of representatives of the three groups involved to sit in the same room around a conference table and calmly and dispassionately discuss their common problems. Prior to the appointment of the American Medical Association Committee, Dr. Hugo Hullerman of the American Hospital Association had recommended the formation of a committee representing administrators, nurses, and physicians to study the problems common to all three groups. An invitation to join this group came to the American Medical Association while the Committee on Nursing Problems was making its study. The members of our committee were unwilling to affiliate with this group until the study was completed. In 1948 the Commission for the Improvement of the Care of the Patient was formed, with six representatives from each organization on the commission. The American Medical Association was one of the member organizations, and the Committee on Nursing Problems became part of the commission. Rules for operation were written and adopted, officers were elected, and the organization became a going concern.

Progress, as was to be expected, was slow in the formative years. Now the organization is one of the very hard-working organizations in medicine in this country. The commission meets twice yearly. The attendance has been excellent, and the discussions and debates have been stimulating.

The problems brought before the commission are many and varied. In the beginning, the problems were chiefly related to nursing. Now the commission studies such problems as those relating to administration, medical care, interns, pathologists, radiologists, and anesthetists. In several states committees of representatives of the three groups have been formed to discuss common problems on the state level.

Few physicians are familiar with the problems of the hospital administrator. Budgetary problems are a common headache of the administrator. The difficult personnel problem is another, personality conflicts are another, and allotment of hours of work and of work on different shifts or weekends or holidays is still another. Occasionally there is a controversy as to whether a problem is administrative or comes within the province of the medical staff.

Physicians in general are not familiar with the intricacies of nursing problems. The nursing school problem is one of the difficult ones. There are also the problems of providing nursing service in the hospital when the students are at class, and maintaining required courses to conform to state laws. The required hours for various courses and the required nursing hours per patient day must be scheduled. Budget problems must be solved, and late notices to the nursing administrator that illness will prevent working that day or evening dealt with. There is also the difficulty of getting the physicians to give lectures or bedside courses and of making physicians understand that these are absolutely necessary in the operation of an accepted nursing school.

In the same way, administrators are unable to know or to understand the problems of the medical staff. Suitable staff rooms for conferences are necessary. There must be adequate equipment for all reasonable procedures. A good library must be provided. Cooperation of the administrator must be secured in the teaching program for interns and residents. Cordial and cooperative personnel must serve in the admitting offices.

Again by the same token the nursing leaders and authorities should attempt to know the problems of the medical staff. Some of these problems are very burdensome and the responsibility is great. After all, the person who is chiefly responsible for the end result that his patient obtains is the physician. It should be determined whether there has been backbiting behind the scenes, whether the nursing leaders have been carelessly evaluating physicians to lay persons, and whether nurses have been making relatives unhappy by criticizing the ability of the attending physician.

Another delicate point is the position of the physician who has courtesy privileges but is not a member of the active staff. His position may be due to unwillingness on his part or nonacceptance of him by the regular staff. Every encouragement should be shown him by the administration and the nursing staff. The medical staff should see that he is invited to attend staff conferences so that his will be a continuing educational program. If the criticism comes from him, cordial relations can frequently be established by kindly actions of the administrator or nursing staff or members of the medical staff. If the criticism comes from the administrator or the nursing staff or the medical staff, he becomes a better physician by attending staff conferences and by being familiarized with the problems of the administrator or the nursing staff.

I feel that, whenever there is disagreement in hospitals between administrators, physicians, and nurses, they are due to lack of understanding of one group of the problems of the other. I am sure that a hospital Committee for the Improvement of the Care of the Patient would help a great deal in understanding and even in solving these problems. We are all too prone to be critical of the other fellow's shortcomings when we do not know or understand his problems. It seems to me that we will not be doing our full duty to the patient until we have set up in each hospital such a committee, bringing this advancement, and I feel it is a real advancement, directly to the patient. This hospital committee made up of representatives of the three groups would go far to aid in the solution of all of these problems. I would recommend also that the representatives be top flight persons from each group.

One of the great problems confronting the American people today is the cost of hospital care. At the turn of the century and as late as the beginning of the second decade of this century, the cost to the patient in a ward was $1 a day, in a semiprivate ward, $1.50 a day, and in a private room, $2 a day. What a sad awakening one has when he receives his hospital bill today. A two weeks' stay in a private room for a surgical patient will cost about $500.

We glibly state that the average patient now becomes ambulatory more quickly and is discharged in about eight days, whereas before he remained in the hospital about 14 days. Obviously this is not the whole answer. Heretofore, it was the common practice of the hospital to furnish all but the most unusual and most expensive medications without charge. There was only limited use of x-rays. Laboratory work was limited and usually insignificant. Anesthetics were administered by an untrained doctor or nurse. Ether and chloroform administered by drops were about the only anesthetics used. All of this has been changed, and necessarily so.

Are we as physicians depending too much on instruments of precision? Are we depending too much on the use of x-rays in chest examinations instead of the careful use of the stethoscope? Are we asking young laboratory technicians to make our diagnoses for us? Are we placing too much reliance on intricate laboratory tests when a careful history and physical examination would give us a more reliable answer? Are we placing too much stress on basal metabolic readings when a careful history of weight gain or loss, heart rate, blood pressure readings, hair distribution, and bodily stature would give us a more reliable answer? Are we leaning too heavily on electrocardiographic tracings when a careful history and physical examination would tell us more? These are some of the questions we must ask ourselves and answer if we are to contribute to the reduction of the cost of efficient hospital care.

How many of us as physicians are familiar with the cost of the antibiotics, sulfonamide drugs, cortisone, or corticotropin (ACTH)? Have we been using these preparations carelessly or without indication? Have we been using two antibiotics that might have antagonistic effect? These also are some of the questions we must ask ourselves and answer if we are to help solve this problem. How many of us are familiar with the cost of anesthesia, blood and blood derivatives, intravenous fluids, physiotherapy, basal metabolic readings, electrocardiograms, and roentgenograms? How many of our patients are being kept in private rooms in a general hospital when they would do as well or perhaps even better in a convalescent home if they are faced with long-term illnesses at great cost to the patient and the hospital?

In the past each department in the hospital has felt that its own department was a separate department and was not the concern of the others. Now we have come to know that all departments are definitely interrelated and that the work of each department must be coordinated with the others if we are to give our patients efficient and economical care.

For a long time, I have felt that a hospital operating too far out of the red was defeating the purposes for which it was intended. When great profits are shown, that profit comes from blood. God did not intend that. At the same time, I don't think it was intended that waste or carelessness should be permitted that would incur great losses. The dangers of socialism and communism are always before us. Both begin where there is want, and are thrown out as sops to a suffering people. Both are grasped for, as a drowning man grasps for a straw, little realizing the price he eventually pays. The socialist and communist uses the cost of medical and hospital care as one of the great reasons for the adoption of this abominable philosophy.

We must find ways and means to see that our patients are given efficient medical and hospital care and that the cost is kept at a reasonable amount. If the present procedures continue, the cost of medical and hospital care will continue to increase, and the law of diminishing returns will take command. I feel that all of these problems and differences can be solved and answered by the generous cooperation of the hospital administrators, nursing staff, and medical staff. This cooperation can be accomplished, I am sure, by the formation of a Committee for the Improvement of the Care of the Patient in each hospital. It is not a new idea, but it is a practical way of helping to solve these problems. With a joint national Commission for the Improvement of the Care of the Patient functioning on the national level and with committees on state level, it naturally follows that the proper place for implementation is on the hospital level.

I would further recommend that this committee meet monthly or bimonthly and that all of the problems of major or minor importance be reviewed and recommendations made for their solution. This committee should be made up of topflight persons. The seniors and leaders in each department should participate. In this way, persons responsible for policy making could speak for their divisions. We could in this way, and probably better than in any other way, familiarize all with the problems of each toward the end of providing the best medical care for all the people.

Finally we should keep before us, always, this fact: When a husband brings his wife to the hospital, a wife brings her husband to the hospital, a father and mother bring their child to the hospital, they are giving to us, for our care the body of a loved one, life's most priceless possession. We must not fail them.

147 W. Main St.

Histoplasmosis.—The highest levels of histoplasmin sensitivity in any region in the world occur in the east-central part of the USA near the junctions of the Missouri, Mississippi, and Ohio rivers, an area in which clinical cases of histoplasmosis have been repeatedly observed. In Canada, histoplasmin sensitivity has been found only in the south-eastern provinces. A somewhat lower prevalence of histoplasmin reactors than in the USA has been reported from certain localities in Mexico, Central America, and South America; the relatively small number of histoplasmosis cases diagnosed in these areas is suggestive but inconclusive evidence of the existence of endemic centres of histoplasmosis.

There appears to be very little histoplasmin sensitivity wher studies have been conducted outside the Americas. Practicall nothing is known concerning sensitivity in Asia, except for tw investigations in India which revealed a negligible number o reactors to histoplasmin. A great many reports covering mo of the European countries have failed to demonstrate any sens tivity areas, although single, isolated cases of histoplasmos have been diagnosed in Austria, Bulgaria, England, the Nethe lands, Portugal, Spain, and Turkey. Several cases have occurr in the Union of South Africa, which appears to be an area low prevalence of histoplasmin sensitivity, but little is kno of the rest of the African continent. Three cases have been o served in Java, but no studies of histoplasmin sensitivity ha yet been reported.—Alessandro Mochi, M.D., and Phyllis Edwards, M.D., Geographical Distribution of Histoplasmo and Histoplasmin Sensitivity, Bulletin of the World Health ganization, Volume 5, No. 3, 1952.

CLINICAL NOTES

EMERGENCY APPENDECTOMY IN A PATIENT RECEIVING ANTICOAGULANTS FOR MYOCARDIAL INFARCTION

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Alan P. Parker, M.D.
and
Frank R. Boyer, M.D., Philadelphia

The present widespread use of anticoagulant drugs for treatment and prophylaxis makes it probable that an occasional patient's treatment will be complicated by the necessity for emergency operation. This subject was brought to our attention by the occurrence of acute appendicitis in a patient receiving bishydroxycoumarin (dicumarol®) for acute myocardial infarction. There was no precedent, in our experience, for the handling of such a problem. The following report gives the important details of our management and the results obtained.

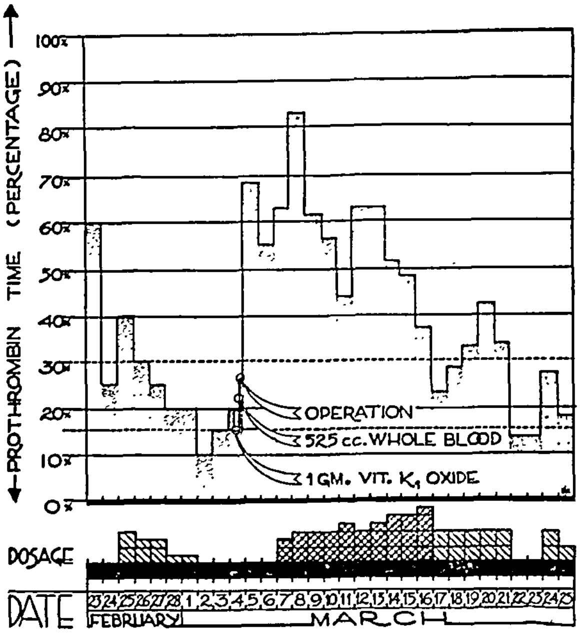
REPORT OF A CASE

T. G., a 47-year-old white man,[1] was admitted to the Coatesville (Pa.) Hospital on the morning of Feb. 19, 1951, with the diagnosis of acute coronary thrombosis. The clinical picture and course were typical, although the electrocardiographic changes were delayed. Anticoagulant therapy, with bishydroxycoumarin, was instituted on the third hospital day. Daily determinations of prothrombin time showed that an effective level of hypoprothrombinemia was maintained during the next 11 days with somewhat less than the average initial daily dosage. Fourteen days after admission, on March 4, the patient experienced diffuse abdominal pain and vomiting with gradual localization of the pain to the right lower quadrant of the abdomen. Definite localized tenderness and rigidity were present, and the diagnosis of acute appendicitis was made by the patient's family physician. After a telephone conversation with one of us (J. B. V.), who had seen the patient on Feb. 26, 1951, as a consulting cardiologist, it was decided to transfer the patient by ambulance to the Bryn Mawr Hospital for observation and possible operation (March 4). A surgical consultant confirmed the diagnosis of acute appendicitis, suspecting local peritoneal irritation, and advised immediate operation.

The prothrombin times on March 1, 2, and 3, were 20%, 10%, and 15% of normal, respectively. On the morning of the day of transfer (March 4) the prothrombin time was 20% of normal. A dose of 50 mg. of bishydroxycoumarin had been given on March 1. Because of the low prothrombin level, no bishydroxycoumarin was given on March 2 or 3. A dose of 50 mg. was scheduled for March 4, but fortunately was withheld because of the abdominal pain. Other medicaments that were given on the day of transfer were meperidine hydrochloride, 100 mg.; streptomycin, 1 gm.; penicillin, 400,000 units; and vitamin K (hykinone®), 20 mg. The last drug was given in two doses of 10 mg. each, intramuscularly, at 4 p. m. and 6 p. m.

It was decided to proceed with laparotomy after rapid preparation of the patient with vitamin K_1 oxide and citrated whole blood and, if unusual bleeding occurred in the abdominal wall, to postpone entering the peritoneal cavity until the bleeding tendency could be corrected. One gram of vitamin K_1 oxide[2] was dissolved in 25 cc. of 95% alcohol, mixed with 500 cc. of isotonic sodium chloride solution, and given fairly rapidly, starting at 9 p. m. This was followed by the administration of 525 cc. of citrated whole blood.[3] Blood for the determination of prothrombin time was taken at 9 p. m., and at 10:30 p. m., frozen overnight, and reported the following day as 15% and 24% of normal, respectively. The operation was begun at 10:45 p. m., with cyclopropane, ether, and oxygen anesthesia after intravenous thiopental sodium induction. Curare (12 mg.) aided in producing adequate relaxation. An acute gangrenous appendix without perforation was found and removed without drainage, the operation being completed at 11:50 p. m. No unusual bleeding was encountered, and no further blood was given.

Recovery from operation was relatively uneventful. The temperature reached 101.6 F within the first 24 hours after operation and gradually fell to normal during the next six days. A mixture of 400,000 units of penicillin and 0.5 gm. of dihydrostreptomycin was given intramuscularly every 12 hours for six days after operation. On the morning after operation the prothrombin time was 68% of normal. On the three following days the prothrombin times were 55%, 63%, and 83% of normal, respectively. Anticoagulant therapy was resumed on the third morning after operation, with ethyl biscoumacetate (tromexan® ethyl acetate). Despite the use of large (single) daily doses of this drug, the prothrombin time did not fall to an effective anticoagulant level during the next 10 days (see the accompanying figure). When bishydroxycoumarin was substituted, it was found that doses much larger than average were needed to produce effective prothrombin levels. This seems significant in view of the less than average doses of this same drug needed to produce the same levels preoperatively.



Serum prothrombin time (percentage) in patient T. G., before and after operation. Each square of diagonal lines indicates 50 mg. of bishydroxycoumarin. Each cross hatched square indicates 300 mg. of ethyl biscoumacetate. The broken lines mark the therapeutic anticoagulant zone.

COMMENT

The problem of surgery in a patient receiving full anticoagulant (bishydroxycoumarin) therapy (prothrombin time, 15% to 20% of normal) was handled empirically and without the benefit of immediate prothrombin time determinations. The good results obtained may have been at least partially due to the fact the patient had had no anticoagulant drug for three days prior to operation. Nevertheless, it would seem that the vitamin K (synthetic), Vitamin K_1 oxide, and citrated whole blood, which were given just prior to surgery, were effective in preventing any abnormal bleeding. The relative efficacy

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From the Medical and Surgical Services of the Bryn Mawr Hospital, Bryn Mawr, Pa.

[1] Dr. Julius Margolis provided the data for this patient and gave permission to publish the report.

[2] The vitamin K_1 oxide used was furnished by Merck & Company, Inc., Rahway, N. J.

[3] The blood was obtained from the hospital blood bank and was approximately 48 hours old.

of these different substances cannot be determined in this instance. Our previous experience leads us to believe that the use of the vitamin K_1 oxide was probably an effective measure. The fact that citrated whole blood was given just before the operation must not be overlooked, however.

Prior to operation, somewhat less than average doses of bishydroxycoumarin were needed to produce an effective prothrombin depression. After operation anticoagulant therapy was again resumed, with ethyl biscoumacetate. Unusually large doses were used, and an effective level was not reached in 10 days. Resumption of bishydroxycoumarin therapy demonstrated the fact that the doses needed to produce equal prothrombin depression were much greater than in the preoperative period. The question may be raised whether the vitamin K_1 oxide therapy and/or the blood transfusions may have been operative in producing the relative resistance to the anticoagulant drugs postoperatively.

SUMMARY

Acute appendicitis developed in a patient receiving anticoagulants for an acute myocardial infarction. The methods utilized in the management of this problem, including preoperative and postoperative care, are described.

330 S. 9th St.

SHORT-TERM USE OF CORTICOTROPIN AS ADJUNCTIVE THERAPY

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and
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As experience with corticotropin (ACTH) accumulates, it is becoming increasingly apparent that its greatest usefulness is less as a cure in disease states than as an antitoxic agent, bolstering the defense mechanism and promoting remission. This quality may assert itself by the liberation of substances that absorb toxins by strengthening the cell membrane to prevent entry of damaging agents or by preventing disorganization of enzymatic processes within the cell itself. Bacterial growth is not inhibited, and specific antibody formation is neither accelerated nor arrested. The action may be presumed to be mainly one of blocking the action of toxins and allowing an adequate build-up of the defensive processes.

Viewed in this light, the potentialities of the short-term use of corticotropin for conditions in which there is poor response to other therapy becomes readily evident. Systemic infections, shock states, and acute toxemias, when the patient is overwhelmed or floundering, may be brought under control by the addition of this protective agent, as is illustrated in the following instances.

CASE 1.—J. S., a 63-year-old man, was admitted to the hospital on May 16, 1951, with a diagnosis of acute appendicitis and peritonitis. At operation a ruptured gangrenous appendix with purulent peritonitis was found. The appendix was removed, the wound closed, and a cigarette drain placed in the fascial layer. One-half gram of streptomycin and 300,000 units of procaine penicillin were given intramuscularly twice daily. The abdomen remained distended, and the patient started hiccoughing on the second day. The temperature was elevated to 100 F, and the pulse rate ranged from 110 to 120 per minute. Attempts at passing a Miller-Abbott tube were unsuccessful, and the patient was becoming increasingly toxic and exhausted. While administration of the antibiotics was continued and the daily salt intake reduced, 20 mg. of corticotropin was injected at 10 a. m. on the third day and 10 mg. given every 4 hours, for a total of 12 doses in 48 hours. Six hours after the initial dose, flatus was passed for the first time, and the abdomen was less distended. The temperature dropped to 98.8 F, the pulse rate to 100, and the patient was more comfortable. On the following morning he had a liquid bowel movement, with further reduction in the distention; the temperature remained normal, and the pulse rate dropped to 86. The patient took food with relish and appeared much less toxic. The next day he was even more improved, had another liquid movement, and was passing flatus freely; the distention was practically gone. The hiccoughing had now stopped. Improvement continued from this day on, and he was discharged on the 14th day, having made an uncomplicated recovery.

CASE 2.—R. J., a white man, 61, was admitted to the hospital May 18, 1951, with a diagnosis of acute diverticulitis with perforation and generalized peritonitis. Severe pain in the left lower quadrant of the abdomen and temperature of 101.4 F had occurred the evening before. The fever had persisted, and the abdominal tenderness was now marked over the entire lower part of the abdomen.

Conservative therapy was instituted. Continuous tube suction was started, succinylsulfathiazole (sulfasuxidine®) and terramycin were given by mouth, and penicillin and streptomycin injected intramuscularly. The temperature remained elevated during the next week, and it became evident that an abscess had developed in the left lower part of the abdomen. While this area was palpated, the abscess ruptured, and the patient was taken to the operating room, where the abscess was drained and three Penrose drains left in situ.

In spite of seemingly adequate drainage and continued treatment with antibiotics, the temperature and pulse rate remained elevated, and the patient was making no progress. On the fifth day an injection of 20 mg. of corticotropin was given, followed by a 10 mg. dose every four hours for a total of 12 doses. Six hours after the first injection the temperature dropped to normal, and the patient reported an increased sense of well-being. He improved rapidly from this point and was discharged on June 4th, nine days after operation.

CASE 3.—A. B., a white man, 65, was admitted to the hospital April 20, 1951, because of severe knife-like pain in the right side of the chest of one week's duration. The night before admission he had a slight chill, and the pain became much severer. The temperature was 100.2 F, the pulse rate 100, and there was evidence of pleural effusion over the right lung base. There was very little cough and no hemoptysis. The white blood cell count was 10,600, with 79% polymorphonuclears, 9% lymphocytes, 9% mononuclears, and 3% eosinophils. X-ray study on April 21 showed evidence of right lower lobe pneumonia with associated effusion.

On April 21 and 22 the pleuritic pain became more intense, dyspnea was marked, and there was slight cyanosis. Strapping of the chest made the pain worse, and intercostal nerve block produced only temporary relief. Thoracentesis was done on April 23d, and only 15 cc. of cloudy orange-colored fluid was obtained. This showed principally mature polymorphonuclears, with many very large and often multinucleated histiocytes. Blood and pleural fluid culture showed no growth. Dulness was now present over both lung bases, and the pain, dyspnea, and cyanosis, with elevated temperature, persisted in spite of continuously administered oxygen and frequent use of anodynes. Use of penicillin and streptomycin, 300,000 units and 0.5 gm. respectively, was started twice daily.

On April 24, with the condition and treatment unchanged, the first injection of 20 mg. of corticotropin was given, and this was repeated every four hours for 12 doses. Six hours after the first dose the pain had stopped, the respirations had slowed, and their depth had increased. By the next morning improvement

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From the Departments of Medicine and Surgery, Harper Hospital.

was even more marked; the temperature was normal, the breath sounds over the lung bases were louder, and crepitant rales were heard for the first time. There was no more dyspnea or pain during the period of corticotropin administration, but five hours after the last dose was given the pain and dyspnea recurred. Corticotropin was given again, this time in a dosage of 20 mg. every six hours, and again there was dramatic disappearance of the disturbing signs. Use of the drug was continued for the next six days at the same dosage level; administration was decreased on the seventh day to 15 mg., on the eighth day to 10 mg., and on the ninth day to 5 mg. every six hours, then discontinued. This time the improvement persisted, and, while there were occasional fleeting chest pains, the signs of effusion gradually disappeared.

X-ray studies on April 28 showed increased fluid in the basilar section of the right thorax, obscuring the lower part of the right lung. Two days later an area of consolidation was evident, which surrounded pleural thickening of the right lower lung field highly suggestive of infarct. After another four days the localized area of consolidation just above the right leaf of the diaphragm again showed no change. One area of rarefaction, without definite fluid level, was seen within the center of the lesion. The left costophrenic angle was now obliterated, suggesting accumulation of fluid here. A final x-ray on May 11 showed slight progress in resolution of the lesion in the lower third of the right lung.

On May 7, four days after use of corticotropin was discontinued, marked pain and tenderness in the calf of the left leg developed, and bilateral femoral vein ligation was performed by Dr. E. A. Osius. Recovery from the operation was uneventful, the chest signs continued to improve, and the patient was discharged May 14, 1951, 25 days after admission.

SUMMARY

In the first two patients, both with purulent peritonitis, response to therapy was indifferent and little, if any, progress was being made toward recovery. Administration of corticotropin (ACTH) for 48 hours, while use of antibiotics was continued, provided the necessary stimulus that tipped the scales toward a prompt favorable outcome. The third patient, with severe pain, dyspnea, and toxemia from pulmonary infarction, responded promptly to 48 hours' treatment with corticotropin but showed return of symptoms when it was stopped. The condition improved and recovery followed when the corticotropin therapy was resumed and continued for another six days. Accordingly, in these three patients, the value of the short term use of corticotropin for conditions in which there is poor response to other forms of therapy has been demonstrated.

968 Fisher Bldg. (Dr. Reveno).

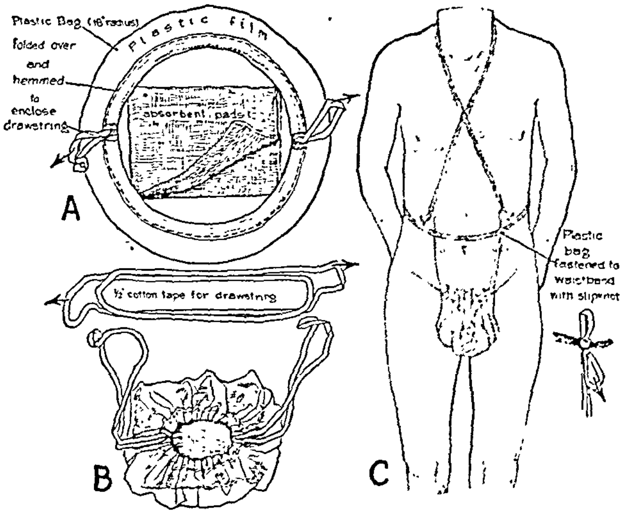
Meaningless Phrases.—In its proper proportion description of a disease entity is only the first step, and a small one at that, toward understanding of etiology, pathogenesis, and treatment. There is no disease which is thoroughly understood; often specific therapy does more to stifle progress in this respect than any other single factor. Insulin effectively stopped research on the etiology and pathogenesis of diabetes for twenty years; the antibiotics may be doing the same for infectious diseases. Medical science, still in its childhood, continues to describe new disease entities, which is progress. But when we are tempted to use meaningless phrases, or what is worse, the names of men, as descriptions, we are taking semantic refuges from the realities of ignorance. "Essential" hypertension, idiopathic thrombocytopenia, Loeffler's syndrome are examples. "Essential" means "I don't know." I have always preferred the word "agnogenic," which really says, "I don't know." Let us not be afraid to admit our ignorance, and qualify words with that humble admission, thereby making a challenge of a refuge.—Henry A. Schroeder, M.D., Knowledge, Faith and Humility, *The Centaur of Alpha Kappa Kappa*, May, 1952.

PLASTIC BAG FOR URETHRAL LEAKAGE CONTROL AFTER PROSTATECTOMY

Thomas S. Cullen, M.D., Baltimore

In March, 1950, Dr. Howard C. Smith removed the central lobe of my prostate at the Church Home and Hospital. He described it as the largest central lobe he had ever encountered in surgery; the weight was 254 gm. The posterior incision healed by first intention; nevertheless, there was a great amount of leakage from the urethral orifice, evidently due to laxness after removal of the large tumor.

As soon as I was well enough, Mrs. Cullen, a graduate of the Johns Hopkins Hospital School of Nursing, undertook control of the leakage. She selected a waterproof plastic,[1] so soft that it could hardly be felt. She made a round bag of this material sufficiently large to contain the genitalia, with a wide hem through which to draw tapes.


Plastic Bag (18" radius)
Plastic film
folded over and hemmed to enclose drawstring
absorbent pads
A
½" cotton tape for drawstring
B
C
Plastic bag fastened to waistband with slipknot

A, top view of plastic bag showing placing of drawstrings and absorbent pads. *B*, drawstrings drawn tight so that the bag would completely cover the genitalia. (The tighter the strings are drawn, the better.) *C*, bag in place. To prevent the bag from slipping down in the back, the two ends of the drawstrings should be fastened laterally, as far as possible, instead of near the front as indicated in the drawing.

Two absorbent pads were placed in the bag, the bag was placed around the genitalia, and the tapes were drawn tight enough to prevent leakage. Each tape string was then tied to the side of the abdominal girth (made of gauze bandage) by slipknots, which could be easily untied. The girth was held firmly by another gauze bandage placed around the neck, crossed over the chest, and tied on each side of the girth (see figure).

The plastic bag can be removed whenever necessary, usually every two or three hours during the day, and replaced by a fresh one with dry pads. At night, a urinal may be used when required. When the patient gains strength, he can remove and replace the bag without assistance.

20 Eager St. (2).

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[1] Acor plastic film, a product of Acor Co., New

TORULOSIS (CRYPTOCOCCOSIS) PRODUCING A SOLITARY PULMONARY LESION

REPORT OF A FOUR YEAR CURE WITH LOBECTOMY

Morris Berk, M.D.
and
Bruno Gerstl, M.D., Oakland, Calif.

Torulosis, or cryptococcosis, is an often fatal disease due to a yeast-like organism and having a predilection for the central nervous system, with the lungs as the second commonest site of involvement. It has been considered a rare disease, but many cases are probably overlooked, owing to the lack of its consideration in the differential diagnosis, failure to culture the organism on suitable mediums for sufficiently long periods of time, and frequent confusion of the organism with lymphocytes or red blood corpuscles in the spinal fluid.[1] Notwithstanding, a total of nearly 200 cases have now been reported.[2] Of this number, only five cases of solitary lung involvement are on record (see the accompanying table).[3] To this meager group we add a sixth, which is considered worthy of publication not only because of its rarity but also because it simulated tumor of the lung, eventually leading to thoracotomy. Other authors have mentioned similar experiences.[4] For this reason attention is hereby again directed to the necessity for consideration of torulosis in the differential diagnosis of circumscribed pulmonary lesions. Also, inasmuch as there is no specific therapy and since pulmonary involvement may actually precede dissemination, there is a growing tendency to consider surgical resection in all cases of pulmonary cryptococcosis. Thus, Taber[4b] has stated that "the early surgical removal of a circumscribed area of torulosis seems as logical as the early removal of carcinoma." Our case would tend to bear this out.

Cases of Solitary Pulmonary Cryptococcosis Found in the Literature

Author	History	Roentgen Findings	Method of Diagnosis	Outcome and Comment
Sheppe[3a] (1921)	Age 50; white male; 4-month illness with tonsillitis, neck stiffness, and weight loss; eventual development of signs of consolidation at right lung base	Initial x-ray normal (no additional x-rays)	Culture of pulmonary tissue at necropsy	Death; brain and meninges apparently not examined; no report of spinal fluid examination
Hardaway and Crawford[3b] (1935)	Age 33; white male; cough only symptom	Multiple nodular lesions; 15-mo. follow-up disclosed little change	Smear and culture of sputum	No essential change during 15-mo. observation
Greening and Menville[3c] (1947)	Age 24, Negro; chest pain, cough, and hemoptysis	Patchy consolidation of upper lobe of left lung and enlargement of bronchopulmonary lymph nodes; confluence of patchy areas with cavity formation and pleural effusion; fibrosis one year after onset	Smear and culture of pleural fluid	Eventual improvement with potassium iodide, antibiotic, and roentgen therapy; alive after one year
Moody[3e] (1947)	Age 38, Mexican; suspected tuberculosis; bronchoscopy revealed "tracheal tumors"	Submiliary lesions	Culture of tissue at necropsy	Asphyxial death
Froio and Bailey[3d] (1949)	Age 19; asymptomatic pulmonary lesion discovered by x-ray at Army induction; referred to tuberculosis sanitarium (negative findings); eventual pneumonectomy for possible bronchogenic cancer	"Mass-like" lesion in lower lobe of right lung	Histological examination of surgical specimen	Circumscribed, rubbery lesion, 4.5 cm. in diameter; alive 5 yr. 7 mo. after surgery without dissemination or recurrence

REPORT OF A CASE

A 30-year-old white World War II veteran, who was receiving compensation for bronchial asthma, was admitted to the Veterans Hospital, Oakland, Calif., on Oct. 31, 1947. Since 1944, when on the island of New Britain in the Southwest Pacific, he had been having infrequent episodes of cough, fever, substernal tightness, wheezing, night sweats, and malaise. He was given a medical discharge from the Army in March, 1945, with a diagnosis of bronchial asthma. After discharge and until his present hospitalization, he had several additional episodes. They were nonseasonal, and each lasted about a week. Five months prior to this hospital entry, during a health survey, a roentgenogram showed a "spot" in the left lung. He was referred to his private physician and eventually to this hospital. The patient, however, was asymptomatic until three days before hospitalization, when he noted the onset of pleural pain over the left side of the chest, malaise, feverishness, and dry cough. There were no chills, hemoptysis, or weight loss. He had had dengue while in the Southwest Pacific in 1943 and seasonal rhinitis since 1945. A maternal grandparent had asthma. There was no history of tuberculosis or of exposure to it.

Physical examination revealed a moderately ill, robust-appearing, white man with a moderate, dry, hacking cough. Respirations were not labored or wheezy. There was no cyanosis or clubbing of the fingers. Oral temperature was 100 F. The pulse rate was 96, respiratory rate 24, and blood pressure 120/88. There were no meningeal signs, skin lesions, lymphadenopathy, or hepatosplenomegaly. The percussion note was resonant throughout. Breath sounds were essentially normal, except at the left lower portion of the chest anteriorly and the lower axilla, where numerous medium and fine rales were heard. No alteration of fremitus or voice sounds was noted.

Laboratory Data.—The white blood cell count was 18,700, with 72% polymorphonuclear cells, 9% stab forms, and 4% juvenile forms. The red blood cell count was 4,700,000, with 14.3 gm. hemoglobin per 100 cc. The sedimentation rate was 95 mm. (Westergren). Results of examination of the blood for cold agglutinins were negative. Results of liver function tests were normal. Blood culture was sterile. Concentrated sputum collections and gastric wash were negative on smear and culture and in a guinea pig test for tuberculosis. The coccidioidin skin re-

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Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

[1] Gendel, B. R.; Ende, M., and Norman, S. L.: Cryptococcosis: Review with Special Reference to Apparent Association with Hodgkin's Disease, Am. J. Med. 9:343-55 (Sept.) 1950.

[2] Cook, A. W.: Cryptococcus (Torula) Meningitis, J. A. M. A. **146:** 1105-1107 (July 21) 1951.

action was negative; a test with second strength purified protein derivative yielded a positive result. Bronchoscopy disclosed a normal condition throughout, and bronchial secretions were negative for tumor cells. The initial chest x-ray (fig. 1) revealed a density occupying the anterolateral and lower portions of the upper lobe of the left lung, just above the interlobular fissure. A subsequent x-ray revealed some increase in size of this shadow, with the addition of a small amount of pleural fluid (fig. 2). Bone survey and excretory pyelograms were normal, a bronchogram of the left side showed moderate cylindrical bronchiectasis in the lingular region. However, bronchi extending to the lesion in question appeared normal.

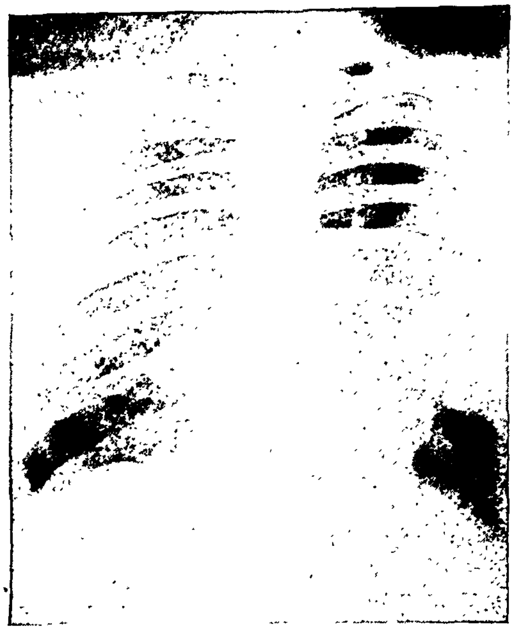
Fig. 1.—Initial roentgenogram, showing circular density in left mid-lung field.

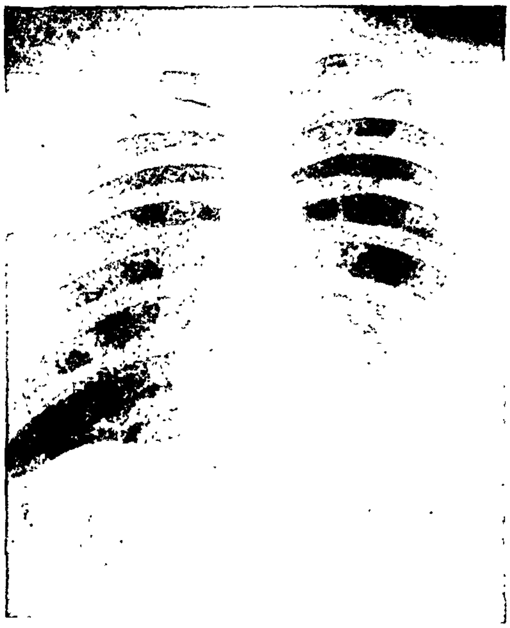
Fig. 2.—Roentgenogram taken 10 days after the first (fig. 1), with more circumscribed outline of the density and the addition of pleural effusion.

Clinical Course.—A tentative diagnosis of pneumonia was made, and the patient was treated with penicillin and sulfadiazine but without response. He continued to have fever, his temperature ranging from 99 to 101.6 F. On the eighth day after entry there was a sudden increase of pain in the left lateral portion of the chest, with physical signs of fluid, accompanied by a pleural friction rub. By thoracentesis 200 cc. of serosanguineous fluid was removed. The sediment of this fluid showed numerous cells of endothelial character. In view of the lack of response to chemotherapy with progression of the lesion seen on x-ray, the case was reviewed by the tumor board, and surgical exploration was advised. A tentative diagnosis of tumor was made. Thoracotomy was performed by Dr. David Dugan, consulting chest surgeon, five weeks after the patient's admission to the hospital, and a large tumor mass in the upper lobe of the left lung was removed by lobectomy. The tumor on frozen section was diagnosed as mycotic granuloma. The patient made an uneventful postoperative recovery and was discharged approximately two weeks later. He was still living, without evidence of recurrence or dissemination of his disease, four years later.

Description of Specimen.—Gross: The pleura of the removed upper lobe of the left lung was intact except over an oval-shaped area of the lower lateral segment, which had a dull yellow appearance. Sectioning revealed an oval-shaped, firm, tumor-like, fairly well-encapsulated, grayish yellow mass with a mucoid surface. It measured 7 by 6 by 6 cm. and corresponded in location to the pleural lesion. The remaining pulmonary parenchyma was unremarkable.

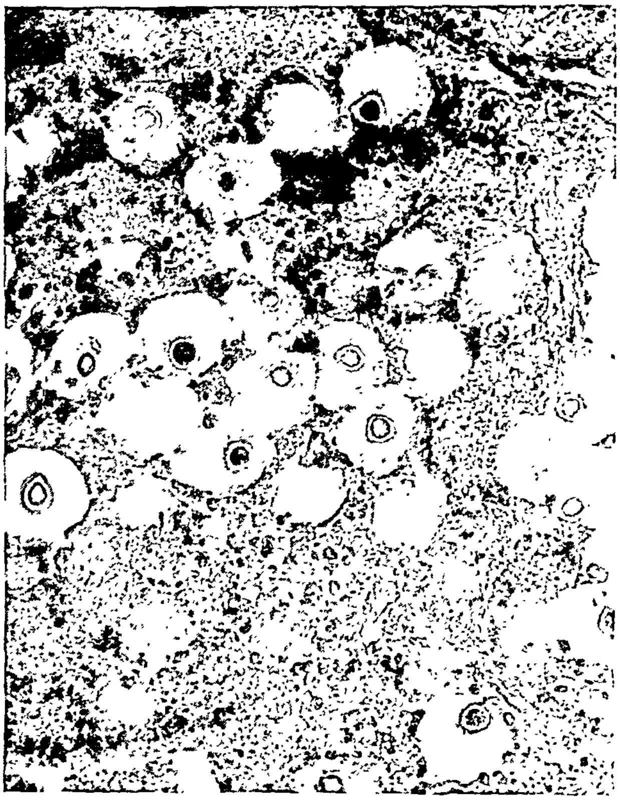
Fig. 3.—Cryptococcus organisms in lung lesion, with characteristic structure and wide refractile capsule (× 230).

Microscopic: Sections representing the peripheral areas of the lesion revealed fairly dense connective tissue infiltrated by small round cells. The adjacent alveoli were compressed. In

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3. (*a*) Sheppe, W. M.: Torula Infection in Man, Am. J. M. Sc. **167**: 91-108 (Jan.) 1924. (*b*) Hardaway, R. M., and Crawford, P. M.: Pulmonary Torulosis: Report of Case, Ann. Int. Med. **9**: 334-340 (Sept.) 1935. (*c*) Greening, R. R., and Menville, L. J.: Roentgen Findings in Torulosis: Report of Four Cases, Radiology **48**: 381-388 (April) 1947. (*d*) Froio, G. F., and Bailey, C. P.: Pulmonary Cryptococcosis: Report of Case with Surgical Cure, Dis. Chest **16**: 354-359 (Sept.) 1949. (*e*) Moody, A. M.: Asphyxial Death due to Pulmonary Cryptococcosis: Case Report, California Med. **67**: 105-106 (Aug.) 1947.

4. (*a*) Froio and Bailey.[3d] (*b*) Taber, K. W.: Torulosis in Man: Report of Case, J. A. M. A. **108**: 1405-1406 (April 24) 1937. (*c*) Dormer, B. A.; Friedlander, J.; Wiles, F. J., and Simson, F. W.: Tumor of Lung Due to Cryptococcus Histolyticus (Blastomycosis), J. Thoracic Surg. **14**: 322-329 (Aug.) 1945.

some areas the parenchyma, including blood vessels, had undergone necrosis. There were cellular debris and fine pink-staining fibers with numerous circular clear spaces. Many of the latter contained round bodies whose centers stained grayish pink and which were highly refractile. Their light pink broad peripheral zone suggested capsule formation (fig. 3). Two nearby bronchial lumina were largely occluded by granulation tissue containing a few multinucleated giant cells. With Kernohan's method,[5] the micro-organisms stained bright pink with pale yellow in the periphery. Sabouraud's medium was inoculated with fresh tissue but remained sterile. India ink preparations of imprints demonstrated a thick capsule.

COMMENT

A review of the literature shows the frequency with which pulmonary cryptococcosis presents itself as a circular, circumscribed density, usually associated with meningeal involvement, or occasionally just preceding such involvement.[6] Pulmonary lesions are said to occur in two forms: either as gelatinous masses or as fibrotic granulomatous lesions. In the first type there is massive proliferation of cryptococci into a gelatinous mass. In the second type there is a paucity of organisms. The miliary nodule, which resembles a tubercule both grossly and microscopically, is the commonest pathological lesion.[7] Greening and Menville [3c] found four cases of torulosis in 537,135 admissions to the Charity Hospital in New Orleans. In three there was pulmonary involvement, followed with serial roentgenograms. From their observations and review of the literature, they noted that the disease had a predilection for the lung bases. In early cases there were circumscribed patches of homogenous consolidation with only a small amount of surrounding reaction. These areas tended to become confluent, occasionally progressing to cavity formation. With healing there was fibrosis. Slight hilar enlargement was sometimes noted.

The portal of entry of torulosis is as yet obscure. Most observers consider it to be the lung. The disease is probably carried to other organs via the blood and lymph channels. Wade and Stevenson's [8] work indicates that the disease passes through a septicemic stage. They were unable to produce central nervous system involvement without visceral involvement (chiefly lung and kidney) when they inoculated white mice intracerebrally, intravenously, intraperitoneally, intratracheally, and subcutaneously. Intranasal inoculation caused only severe rhinitis or sinusitis. Organisms spread out beneath the cribriform plate but did not invade the central nervous system. These studies suggest initial pulmonary involvement and thus the probable importance of early resection.

SUMMARY

A case of solitary pulmonary torulosis simulating tumor is hereby added to the literature as the sixth case on record. The patient is alive, without recurrence or dissemination of the disease, four years after lobectomy. The lung may be the portal of entry of this highly fatal disease. For this reason, surgical excision is recommended as a curative measure prior to dissemination. Torulosis should be considered in the differential diagnosis of circumscribed nodular pulmonary lesions.

13th and Harrison Streets.

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5. Kernohan, J. W.: A New Staining Method to Demonstrate Pathogenic Yeasts and Fungi, J. Neuropath. & Exper. Neurol. **2**: 95-98 (Jan.) 1943.

6. Hardaway and Crawford.[5b] Greening and Menville.[3c] Froio and Bailey.[3d] Taber.[4b] Ratcliffe, H. E., and Cook, W. R.: Cryptococcosis: Review of Literature and Report of Case with Initial Pulmonary Findings, U. S. Armed Forces M. J. **1**: 957-969 (Sept.) 1950.

7. Voyles, G. Q., and Beck, E. M.: Systemic Infection Due to Torula Histolytica (Cryptococcus Hominis): Report of Four Cases and Review of Literature, Arch. Int. Med. **77**: 504-515 (May) 1946.

8. Wade, L. J., and Stevenson, L. D.: Torula Infection, Yale J. Biol. & Med. **13**: 467-476 (March) 1941.

AGRANULOCYTOSIS OCCURRING DURING PHETHENYLATE ("THIANTOIN") SODIUM THERAPY

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and
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Putnam and Merritt's method of testing the anticonvulsant activity of drugs in animals opened an era of intensive search for more effective drugs with which to control epilepsy.[1] Innumerable chemical substances were tested, and, beginning with diphenylhydantoin sodium, several were subsequently introduced into clinical practice.

Whereas some of the new anticonvulsant drugs are now considered valuable additions to the therapeutic armamentarium for epilepsy, their toxic side-effects create a certain amount of hazard in the treatment of this disease. There have been reports of the occurrence of bone marrow depression, exfoliative dermatitis, nephrosis, and hepatitis, with recoveries and fatalities. Apparently depression of the blood elements is the commonest among these serious complications. In 1950, Abbott and Schwab noted that there had been 25 fatal reactions in the previous four years, which occurred during trimethadione, methylphenylethyl hydantoin, or phenacemide administration; 15 of the patients died with aplastic anemia or pancytopenia (6 during methylphenylethyl hydantoin, and 9 during trimethadione therapy).[2] Since then we have found additional reports of pancytopenia and death from methylphenylethyl hydantoin therapy (one case)[3] and from phenacemide therapy (two cases).[4] While paramethadione therapy may also cause bone marrow depression in susceptible patients,[5] no serious cases have been reported.

Phethenylate ("thiantoin") is one of the newest drugs with anticonvulsant properties. It was prepared in 1941, and preliminary clinical trials began in 1942. In 1947, Peterman reported on 73 children treated with this drug.[5a] He observed as side-reactions hypertrophy of the

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From the Medical Service and the Neuropsychiatric Service of the Veterans Administration Hospital.

1. Putnam, T. J., and Merritt, H. H.: Experimental Determination of Anticonvulsant Properties of Some Phenyl Derivatives, Science **85**: 525 (May 28) 1937.

2. Abbott, J. A., and Schwab, R. S.: Medical Progress: The Serious Side Effects of the Newer Antiepileptic Drugs: Their Control and Prevention, New England J. Med. **242**: 943 (June 15) 1950.

3. Hofstatter, L.: Mesantoin in the Management of the Hospitalized Psychotic Epileptic: Report of Toxic Effects and One Fatality, South. M. J. **44**: 827 (Sept.) 1951.

4. Forster, F. M., and Frankel, K.: Fatal Aplastic Anemia During Anti-Convulsant Therapy, Dis. Nerv. System **10**: 108 (April) 1949. Simpson, T. W.; Wilson, E. B., Jr., and Zimmerman, S. L.: Fatal Aplastic Anemia Occurring During Anticonvulsant Therapy: Probable Idiosyncrasy to Phenurone, Ann. Int. Med. **32**: 1224 (June) 1950.

5. (*a*) Peterman, M. G.: Epilepsy in Childhood: Newer Methods of Diagnosis and Treatment, J. A. M. A. **138**: 1012 (Dec. 4) 1948. (*b*) Forster, F. M., and Borkowski, W. J.: Paradione in the Treatment of Petit Mal Epilepsy, Am. Pract. & Digest Treat. **1**: 406 (April) 1950.

gums in two cases, ataxia in four cases, and rash caused by the drug in two cases. The drug has been used in over 40 cases at the Illinois Neuropsychiatric Institute without any notable blood changes.[6] In 1950, Best and Paul reported a case of severe hypoplastic anemia with recovery occurring during diphenylhydantoin sodium, methylphenylethyl hydantoin, and phethenylate administration.[7] In 1951, Weiss and Hawley described a case of fatal pancytopenia following the use of methylphenylethyl hydantoin and phethenylate.[8] The part that phethenylate played in the production of these toxic accidents cannot definitely be stated, since in both cases this drug was given simultaneously with methylphenylethyl hydantoin, a known depressant of the hematopoietic system. In order to furnish the first known direct evidence of toxic effects of phethenylate on the bone marrow, we consider it of interest to report a case of agranulocytosis in a patient during administration of this drug.

REPORT OF A CASE

E. J., a 37-year-old divorced woman, was admitted to the neuropsychiatric service on Aug. 17, 1951. She had a history of life-long inadequacy and dependency. The onset of her psychosis was insidious and extended over a period of years. On admission, the physical examination showed nothing essentially abnormal. A routine blood cell count showed 4,500,000 red blood cells, 12.5 gm. of hemoglobin, and 5,400 white blood cells, with a differential count of 62% neutrophils, 3% eosinophils, and 35% lymphocytes. Psychiatric observation revealed that the patient was actively hallucinating; she was confused and disoriented as to place and often got lost in the ward. Her speech was incoherent, irrelevant, and showed marked delapidation of thoughts.

A course of deep-coma insulin treatment was started, which had to be discontinued because urticaria and angioneurotic edema developed. This allergic reaction to insulin also reappeared when zinc insulin was substituted for crystalline insulin. Treatment, therefore, was changed to electric shock therapy. The patient's condition became worse, and she started to complain of severe headaches. With this new symptom and a history of an old head injury, an electroencephalogram was made. The tracing showed abnormalities compatible with psychomotor epileptic discharge, idiopathic rather than of focal origin. Anticonvulsant therapy was recommended by the electroencephalographer, and on Nov. 1 treatment with phethenylate sodium, 2 grains (0.13 gm.) twice a day, was started. Soon after this treatment was instituted, definite improvement was observed in the patient's mental condition. The headaches subsided, and she seemed less confused. No routine blood cell counts were made during medication. The patient had received no other drugs since her admission to the hospital.

On Nov. 19, the 19th day of phethenylate therapy, the patient complained of being cold and aching all over. Her temperature was normal, and symptoms subsided spontaneously. On Nov. 21, she had a severe chill lasting over an hour, followed by a temperature of 105.4 F rectally. On Nov. 22, she felt comfortable except for vomiting after lunch. The maximal temperature on that day was 103 F.

On Nov. 23, the patient again had a chill, followed by a temperature of 104.8 F. At this time a medical consultation was requested. The patient had no symptoms, and she appeared cheerful and alert. Physical examination revealed a slight pallor of the skin but none of the mucous membranes. The throat was clear. There was a single area of ecchymosis of the conjunctiva of the right eye; otherwise the skin and mucous membranes were clear. No enlarged lymph nodes were palpable. The spleen was palpable on deep inspiration, but results of the rest of the physical findings were negative.

A roentgenogram of the chest was normal, and results of urinalysis and malaria smears were negative. A blood culture was taken and later reported sterile. A complete blood cell count showed 2,200 white blood cells with 51% neutrophils and a shift to the left. The red blood cell count, hemoglobin, and platelet count were normal. The diagnosis of granulocytopenia due to phethenylate sodium therapy was made.

Beginning Nov. 24, the patient was free of symptoms and felt well during the rest of the observation period, with the temperature remaining normal. Her subsequent course was characterized by changes in the blood cell count (see the accompanying table).

The total white cell count fell to its lowest level, 1,500, on Nov. 26 (the sixth day of illness), and granulocytes were completely absent in the peripheral count on Nov. 28 (eighth day). On that day a sternal puncture was done for a bone marrow study. The differential cell picture of the bone marrow showed a disturbance in maturation at the myelocyte level consistent with the diagnosis of granulocytopenia due to drug sensitivity. There was an increase in granulocytic myelocytes. The myeloid-erythroid ratio was found to be 1: 1.24. The megakaryocytes were normal and the platelets plentiful.

Neutrophils reappeared in the peripheral blood on Nov. 29 (9th day) and reached 50% in a 5,200 total white blood cell count on Dec. 4 (14th day). The peripheral red cell count did not show any abnormalities except for a transient 15% drop on the fifth and sixth days of illness. The platelet count remained normal throughout the entire period.

Phethenylate therapy was discontinued after the first blood cell count, Nov. 23 (third day), and treatment then consisted of the administration of procaine penicillin, 600,000 units once daily, and bed rest. In anticipation of pancytopenia, because of the red cell drop, blood transfusions were started on Nov. 25 and given for five consecutive days. Retrospectively, however, we do not believe that erythropoiesis was impaired in this case or that the transfusions played any role in the recovery of the patient.

Changes in the White Blood Cell Count in the Reported Case

Date (1951)	Day of Illness	White Blood Cell Count	Neutrophils, %	Lymphocytes, %
11/23	3	2,200	51	44
11/25	5	2,200	4	96
11/26	6	1,500	3	97
11/27	7	2,100	1	99
11/28	8	2,100	0	100
11/29	9	2,000	1	99
11/30	10	2,300	2	98
12/ 3	13	4,000	48	52
12/ 4	14	5,200	50	48

COMMENT

Apparently all the newer anticonvulsants are capable of depressing bone marrow in an occasional case. Only diphenylhydantoin sodium seems to be an exception to this. It is beyond the scope of this report to discuss in detail the control and prevention of the serious side-effects of the newer antiepileptic drugs, a topic comprehensively presented by others.[9] The advisability of frequent blood cell counts during the administration of the newer anticonvulsants, however, is emphasized.

SUMMARY

Agranulocytosis developed in a patient on the 20th day of administration of phethenylate ("thiantoin") sodium, 2 grains (0.13 gm.) twice a day. After cessation of treatment with the drug, the patient promptly recovered.

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[6] Gibbs, F. A., and Garvin, J., quoted by Best, W. R., and Paul, J. T.: Severe Hypoplastic Anemia Following Anticonvulsant Medication: Review of Literature and Report of a Case, Am. J. Med. 8: 124 (Jan.) 1950.

[7] Best, W. R., and Paul, J. T.: Severe Hypoplastic Anemia Following Anticonvulsant Medication: Review of Literature and Report of a Case, Am. J. Med. 8: 124 (Jan.) 1950.

[8] Weiss, H. A., and Hawley, W. D.: Pancytopenia Following the Use of Mesantoin and Thiantoin, U. S. Armed Forces M. J. 2: 83 (Jan.) 1951.

[9] Abbott and Schwab.[2] Best and Paul.[7]

INTOXICATION DUE TO CHLORDANE

REPORT OF A CASE

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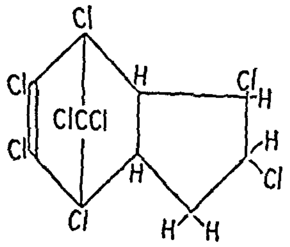
and
Commander Wilmot F. Pierce (MC), U.S.N.

Chlordane is a commonly used insecticide with the empirical formula $C_{10}H_6Cl_8$ and the following structural formula.



Stohlman, Thorp, and Smith[1] have reported that it causes acute toxicity in rabbits equal to that caused by dichlorodiphenyltrichloroethane (DDT), a similar compound, in equivalent oral dosage ($L.D._{50}$), and that it causes greater chronic toxicity than does this similar compound because of greater cumulative action. Lehman[2] reported that toxicity in rats occurred after 12 weeks' administration of chlordane, 250 parts per million in the diet, the lowest intake causing gross demonstrable defects, whereas the toxic dose of dichlorodiphenyltrichloroethane was only 100 parts per million, but this required administration for 104 weeks. In dogs, administration of chlordane, 660 parts per million, caused death in four weeks; 2,640 parts per million of dichlorodiphenyltrichloroethane was necessary over a similar period. As with dichlorodiphenyltrichloroethane, continuous administration of chlordane to laboratory animals caused focal hepatic necrosis, edema, congestion and exudates in the lungs, and degenerative changes in intestinal submucosa and in the convoluted tubules of the kidneys. Organically bound chlorine was demonstrated in the urine of these animals. Bushland, Wells, and Radeleff[3] reported that, when chlordane was applied to cattle, hogs, sheep, and goats by spraying or in dips, signs of neurological and hepatic damage and subserosal hemorrhages resulted.

Statements by some investigators indicate the possibility of human intoxication from chlordane. Lehman[2] reported that the mean lethal dose of this compound in man is half that of dichlorodiphenlytrichloroethane and that smaller amounts than those of dichlorodiphenyltrichloroethane can be dangerous to man, in both divided and single doses. Hine[4] stated, "The halogenated hydrocarbons affect, in large amounts, the heart or central nervous systems, just as does chloroform; and in small amounts, if given over a period of time, produce liver and kidney damage, as does chloroform." On the other hand, Princi and Spurbeck,[5] in a study of 22 workers exposed to chlordane, aldrin, and dieldrin (compounds similar to chlordane) in the manufacture of these compounds, found no evidence of toxic effects as indicated by history, physical examinations, or laboratory tests. The men worked in atmospheres having up to 54 mg. of chlorinated hydrocarbons per cubic meter and were protected only by rubber gloves. Poisoning by swallowing an aldrin mixture has been reported by Spiotta.[6]

A review of the available medical literature has revealed no specific instance of chlordane causing toxic reactions in man. In the following case there was evidence of injury to the lungs, liver, and hematopoietic system after inhalation of chlordane.

REPORT OF A CASE

A 33-year-old Caucasian housewife was admitted to the United States Naval Hospital, San Diego, Calif., complaining of nausea, vomiting, and cough. She had been well until approximately one week previously, when a mildly acute infection of the upper respiratory tract had developed. Two or three days later, she entered her apartment at about 9:30 p. m. and noticed strong fumes of an insecticide, with which the apartment had been sprayed several hours earlier. She never opened her windows at night, fearing burglars, and she retired without airing the rooms. About 3 a. m. she awoke with severe cough and vomiting, and these symptoms continued unabated until admission four days later. The cough produced a moderate amount of thick, white sputum, which diminished in quantity. She did not complain of pain; nausea and vomiting were the only symptoms referable to the gastrointestinal tract. Past history was significant only in that, during an admission five months previously, after cerebral concussion, she had had an epileptiform seizure, the only one of her life; no abdominal organs were palpable at that time. She had returned from a prolonged stay at Guam 18 months prior to the last admission but had not been ill while there. She admitted occasional evenings of alcoholic intake of six or seven highballs but denied having had any alcohol for some time prior to the onset of this illness. The patient's private physician gave the information that she was a periodic "binge" drinker and that he was called to see her a few hours after she became ill by a woman friend of the patient, who was herself obviously intoxicated. He did not see or talk with the patient but suspected she also was intoxicated. She steadfastly denied this. It was ascertained that the insecticide used in the apartment was 1% or 2% chlordane, probably in an oil base similar to kerosene, used in an ordinary insect spray gun.

The patient was an alert, well-developed, slightly obese woman, appearing moderately ill and coughing frequently. Temperature, pulse, respiration, and blood pressure were normal. Physical findings were entirely within normal limits except for a smooth, firm, slightly tender liver edge, which descended three fingerbreadths below the right costal margin on inspiration. Breath sounds, voice sounds, and resonance were normal throughout both lungs, and no rales or rhonchi were heard. No telangiectasia and no lymphadenopathy were noted.

On admission the urine was normal. The Kahn reaction of the blood was negative, and the heterophil agglutination (not absorbed on guinea pig tissue) was positive in a titer of 1:14. Other admission laboratory findings are included in tables 1 and 2. The chest was within normal limits according to roentgenographic examination.

The opinions or assertions contained herein are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

1. Stohlman, E. F.; Thorp, W. T. S., and Smith, M. I.: Toxic Action of Chlordan, Arch. Indust. Hyg. **1**: 13 (Jan.) 1950.

2. Lehman, A. J.: Presented at Conference on Agricultural Chemicals, California Bureau of Adult Health, July 12, 1948.

3. Bushland, R. C.; Wells, R. W., and Radeleff, R. D.: Effect on Livestock of Sprays and Dips Containing New Chlorinated Insecticides, J. Econ. Entomol. **41**: 642, 1949; cited by Princi, F., and Spurbeck, G. H.: Study of Workers Exposed to the Insecticides Chlordan, Aldrin, Dieldrin, A. M. A. Arch. Indust. Hyg. **3**: 64 (Jan.) 1951.

4. Hine, C. H.: Presented at Conference on Agricultural Chemicals, California Bureau of Adult Health, July 12, 1948.

5. Princi and Spurbeck.[3]

6. Spiotta, E. J.: Aldrin Poisoning in Man: Report of Case, A. M. A. Arch. Indust. Hyg. **1**: 560 (Dec.) 1951.

The patient was afebrile throughout and showed rapid improvement. The cough was nonproductive on admission. Three days after admission the cough and emesis had practically ceased, and five days later the liver was no longer palpable. The only treatment was the administration of fluids parenterally on admission, hepatitis diet, and codeine for the cough. She was discharged 10 days after admission and was told to continue to have blood cell counts and liver function tests as an outpatient (tables 1 and 2). Unfortunately, blood drawn on Feb. 2, 1951, for a complete blood cell count clotted in the oxalate tube, and thereafter the patient failed to return to the hospital.

COMMENT

The pulmonary exudation exhibited by this patient and the hepatic dysfunction demonstrated by sulfobromophthalein retention and moderately positive cephalin flocculation are consistent with the toxic changes found to result from chlordane poisoning in animals. Hematologic reactions to dichlorodiphenyltrichloroethane have been reported,[7] including agranulocytosis from aerosol use, so that it is entirely possible that a similar compound, chlordane, caused a like reaction in this patient, with persistent depression of granulocytes, in one determination to 19%. The anemia was normocytic and hypochronic, and normal reticulocyte count and serum bilirubin concentration militate against hemolysis; the cause of the anemia is undetermined.

It might be argued that the blood picture and liver dysfunction were simply the result of cirrhosis in an alcoholic person in view of the slightly lowered albumin-globulin ratio (1.5:1), anemia, and leukopenia. However, the anemia was not macrocytic (mean cell volume, 94) as Tumen[8] stated is the tendency in cirrhosis, and the leukopenia was actually neutropenia. The serum albumin (3.8 gm.) per 100 cc. was only slightly lowered if at all (one source[9] quotes 3.5 to 5.6 gm. as normal, while others[10] give 4 gm. as the minimum normal value), and this is hardly valid evidence of cirrhosis. It should be noted also that hepatomegaly was not observed at the time of the patient's admission five months previously. Further, the liver enlargement found on this admission was no longer discernible after a few days and the degree of sulfobromophthalein retention diminished rapidly, indicating an acute process. Spider nevi and palmar flush were not seen.

TABLE 1.—*Hematological Findings*

	1/10/51	1/12/51	1/15/51	1/17/51	1/24/51
Hemoglobin, gm.	10.5	10.5	9.5	10.5	10.5
Red blood cells, millions	3.7	3.7	3.4 *	3.6	3.7
Hematocrit, %	....	....	32	34	....
Platelets	....	....	....	190,000	....
Reticulocytes, %	....	....	....	1.2	....
White blood cells	3,600	5,200	4,700	5,400	5,050
Basophils	....	....	1	....	1
Eosinophils	....	....	....	....	5
Segmented neutrophils	47	62	18	43	47
Lymphocytes †	53	38	80	57	47
Monocytes	....	....	1	....	....

* Mean cell volume, 94 cu. microns; mean corpuscular hemoglobin, 88 micromicrogram; mean corpuscular hemoglobin concentration, 30%.
† No abnormal lymphocytes reported.

In an editorial in THE JOURNAL[11] it was indicated that fat soluble chlorinated hydrocarbons, such as chlordane, are stored in adipose tissue and in the fat of all parenchymal cells, interfering with enzymatic processes in these cells. In view of this patient's obesity and the fact that she had definite alcoholic tendencies, it might be surmised that she had at least some increase in fat deposition in the liver, which could have made her more susceptible to hepatic insult.

It is unlikely that the abnormalities in this patient could have been caused by the solvent rather than by chlordane. The Council of Pharmacy and Chemistry of the American Medical Association in a report on dichlorodiphenyltrichloroethane[7] authorized the following statement: "The signs of solvent intoxication set in promptly, in contrast to the latent period of several hours required for the development of DDT symptoms." Approximately six hours intervened between the onset of the patient's exposure and development of cough and vomiting. It has also been stated[12] that xylene, kerosene, and other solvents may cause "irritation and other local symptoms, but are of minor importance when compared with the danger of system toxicity," referring particularly to the cutaneous absorption of aldrin and dieldrin, compounds similar to chlordane. It is possible that this patient's productive cough was due to bronchial irritation from the solvent fumes but hardly likely that such irritation would have caused a cough of over a week's duration. The hepatic and hematologic reactions could hardly have been due to solvent intoxication. Unfortunately, the patient was lost to observation before laboratory studies indicated a complete return to normal hepatic and hematopoietic functions.

TABLE 2.—*Results of Liver and Kidney Function Tests*

Test	1/10/51	1/15/51	1/19/51	1/24/51	2/2/51
Serum bilirubin	0.3	0.3	..	..	..
Sulfobromophthalein retention,* %	65	60	..	25	25
Cephalin flocculation					
24 hr.	2+	2+	0	..	1+
48 hr.	2+	2+	0	..	2+
Thymol					
Turbidity, units	1	1	1	..	0
Flocculation	0	0	0	..	0
Serum protein					
Albumin	..	..	3.8	..	..
Globulin	..	..	2.5 †	..	..
Phenolsulfonphthalein ‡					
15 min.	..	..	45%	..	..
60 min.	..	..	15%	..	..

* Dose of 5 mg./kg. of body weight, 45 minute interval.
† Ratio of 1.5:1.
‡ Intravenous injection.

SUMMARY

Toxic manifestations in an adult woman after inhalation of chlordane used in an insecticide spray are described. Involvement of lungs and liver consistent with

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[7] Pharmacologic and Toxicologic Aspects of DDT (Chlorophenothane U.S.P.), Report of the Council on Pharmacy and Chemistry, J. A. M. A. **145**: 728 (March 10) 1951.

[8] Tumen, H. J.: Cirrhosis of the Liver, in Bockus, H. L.: Gastro-Enterology, Philadelphia, W. B. Saunders Company, 1946, vol. 3, p. 317.

[9] Cantarow, A., and Trumper, M.: Clinical Biochemistry, ed. 4, Philadelphia, W. B. Saunders Company, 1949, p. 80.

[10] Bing, J.; Naeser, J.; Rasch, G., and Røjel, K.: Serum Proteins in Normal People, Acta med. Scandinav. **126**: 351, 1946; cited by Hiller, G. I.; Huffman, E. R., and Levey, S.: Studies in Cirrhosis of the Liver: Relationship Between Plasma Volume, Plasma Protein Concentrations and Total Circulating Proteins, J. Clin. Investigation **28**: 322, 1949. Peters, J. F., and Eisenman, A. J.: Serum Proteins in Diseases Not Primarily Affecting Cardiovascular System or Kidneys, Am. J. M. Sc. **186**: 808, 1933.

[11] Insecticide Storage in Adipose Tissue, editorial, J. A. M. A. **145**: 735 (March 10) 1951.

[12] Aldrin and Dieldrin Poisoning, editorial, J. A. M. A. **146**: 378 (May 26) 1951.

chlordane intoxication demonstrated in animals and a hematologic reaction similar to that reported from dichlorodiphenyltrichloroethane exposure occurred The possibility of increased susceptibility in this patient to chlordane poisoning is discussed, as well as the possibility of other factors operating in the production of the symptoms and abnormal physical and laboratory findings.

Cherry and Kimbrough Sts. (Dr. Lemmon).

CUTANEOUS ALLERGY FROM LOCAL CONTACT WITH ISONICOTINIC ACID HYDRAZID

James W. Jordon, M.D., Buffalo

Isonicotinic acid hydrazid and its isopropyl derivative have recently been introduced for the treatment of tuberculosis. Because these drugs will find a widespread use, I believe the following report of a case of cutaneous sensitivity from local contact with isonicotinic acid hydrazid is of considerable interest. As far as I know, this is the first report of such a case.

REPORT OF A CASE

A white man, aged 34, a pharmaceutical chemist, was first seen on May 2, 1952, with the chief complaint of a skin eruption on his hands that had started on March 24, 1952. He stated that the only new product he had worked with prior to the onset of the eruption was isonicotinic acid hydrazid. He first worked with this drug on March 10, 1952. Because of the severity of the eruption he discontinued work from April 11 to April 21, and during this interval the rash had improved. On returning to work the eruption again became severe. When he was examined on May 2, 1952, he had an acute vesicular confluent dermatitis venenata involving the dorsal surface of both hands, the dorsal and ventral surface of both wrists, and to a slight extent the anterior aspects of the face and sides of the neck. He was asked to discontinue work because of the severity of the eruption and to bring in samples of the isonicotinic acid hydrazid as well as all other chemicals he had worked with prior to the onset of the dermatitis. He did this on May 20, and patch tests were made with the chemicals. At the end of 48 hours he showed a 4+ vesicular reaction to a tablet containing isonicotinic acid hydrazid. All other substances with which he was tested gave negative results. Because the tablet contained fillers and binders, he was asked to bring all the separate ingredients of the tablet for further patch test studies, and these were done on May 26, 1952. At the end of 48 hours he showed a vesicular reaction to the isonicotinic acid hydrazid, the other ingredients of the tablet giving negative results. Since discontinuing work and avoidance of contact with isonicotinic acid hydrazid, his dermatitis has gradually subsided. He was last seen on June 2, 1952, and was almost free of his eruption.

COMMENT

The development of the dermatitis venenata from local contact with isonicotinic acid hydrazid shows that this drug can induce epidermal sensitization if it contacts the skin. This means that in others who handle the drug, such as pharmacists, physicians, nurses, or patients, cutaneous sensitivity and dermatitis venenata may develop from local skin contact with it, and it should be suspected as a cause of unexplained dermatitis venenata in such persons.

· 71 North St. (2).

From the Department of Dermatology and Syphilology, University of Buffalo School of Medicine.

PLASTIC COMPRESSOR FOR ENUCLEATION AND EVISCERATION

Conrad Berens, M.D., New York

An "enucleation compressor" devised by Anthony,[1] was found to be most practical in controlling bleeding during enucleation and evisceration. The modification of the Anthony compressor herein described [2] is lighter and, because of the wicket shape, is easily manipulated. The instrument is made of lucite® (polymerized methyl methacrylate), with two arms bent at an angle of 45 degrees to the central grip. The central grip, which is 10 cm. long and 16 mm. in diameter, affords a comfortable hold. The arms of the plastic compressor are 9 cm. in length and taper in diameter from 16 mm. to 4 mm. The arms terminate in ovoid compressor tips, one 14 mm. and the other 11 mm. in diameter (fig. 1). The compressor is light in weight, inexpensive and may be sterilized in the usual manner for other plastic materials.

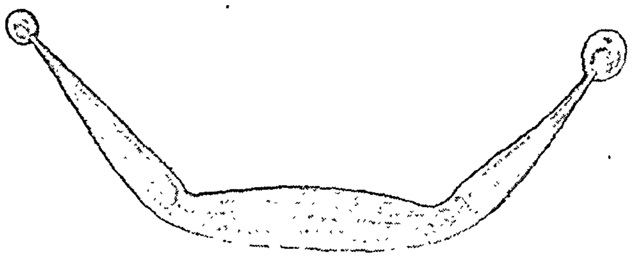

Fig. 1.—Plastic compressor for enucleation and evisceration.

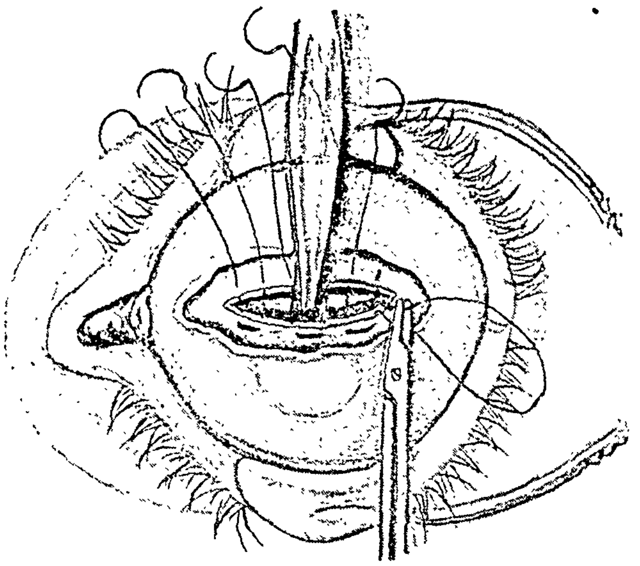

Fig. 2.—Compressor in scleral shell controls bleeding while sutures are being inserted.

The large ovoid compressor tip (for adults) or the smaller tip (for children) is introduced into Tenon's capsule immediately after the optic nerve is severed for enucleation or into the scleral shell after evisceration. Moderate constant pressure usually controls bleeding. The use of a compressor is more satisfactory for hematosis than is filling of the orbit or scleral shell with tampons and the use of digital pressure. Furthermore, with the use of tampons, the surgical procedure is necessarily halted

Aided by a grant from The Ophthalmological Foundation, Inc.

From the Department of Plastics, New York Eye and Ear Infirmary.

[1] Anthony, D. H.: An Enucleation Compressor, Arch. Ophth. 18: 274 (Aug.) 1937.

[2] The lucite® compressor described was made by R. O. Gulden, Philadelphia.

until the bleeding is stopped and the tampons removed from the orbit. In evisceration the scleral wound may be closed with the compressor in place (fig. 2), and the same is true of the suturing of Tenon's capsule in implantations following enucleation.

In several instances extrusion of an implant has been reported, which was apparently due to the fact that bleeding had not been adequately controlled before the implant was inserted. The compressor is of great assistance in controlling hemorrhage.

708 Park Ave.

COUNCIL ON PHARMACY AND CHEMISTRY

NEW AND NONOFFICIAL REMEDIES

The following additional articles have been accepted as conforming to the rules of the Council on Pharmacy and Chemistry of the American Medical Association for admission to New and Nonofficial Remedies. A copy of the rules on which the Council bases its action will be sent on application.

R. T. Stormont, M.D., *Secretary.*

Cortisone Acetate (See New and Nonofficial Remedies 1952, p. 322).

Merck & Company, Inc., Rahway, N. J.

Ophthalmic Ointment Cortone Acetate: 3.5 gm. tubes. An ointment containing 15 mg. of cortisone acetate in each gram.

Ophthalmic Suspension Cortone Acetate 0.5%: 5 cc. dropper bottles. An isotonic, buffered solution containing 5 mg. of cortisone acetate in each gram. Preserved with 0.02% benzalkonium chloride.

Ophthalmic Suspension Cortone Acetate 2.5%: 5 cc. dropper bottles. An isotonic, buffered solution containing 25 mg. of cortisone acetate in each gram. Preserved with 0.02% benzalkonium chloride.

Tablets Cortone Acetate: 5 mg.

Heparin Sodium-U. S. P. (See New and Nonofficial Remedies 1952, p. 208).

Abbott Laboratories, North Chicago, Ill.

Solution Heparin Sodium: 10 cc. vials. A solution containing 5,000 U. S. P. units of heparin sodium in each cubic centimeter; 5 cc. vials. A solution containing 10,000 U. S. P. units of heparin sodium in each cubic centimeter. Preserved with 0.18% methylparaben and 0.02% propylparaben.

Aminophylline-U. S. P. (See New and Nonofficial Remedies 1952, p. 294).

The Bowman Bros. Drug Company, Canton, Ohio.

Tablets Aminophylline: 0.1 gm. plain and 0.2 gm. enteric coated.

Aureomycin Hydrochloride-U. S. P. (See New and Nonofficial Remedies 1952, p. 117).

Lederle Laboratories Division, American Cyanamid Company, Pearl River, N. Y.

Capsules Aureomycin Hydrochloride: 100 mg.

Methamphetamine Hydrochloride-U. S. P. (See New and Nonofficial Remedies 1952, p. 185).

Rexall Drug Company, Los Angeles.

Tablets Methamphetamine Hydrochloride: 2.5 mg. and 5 mg.

COUNCIL ON FOODS AND NUTRITION

Owing to the current interest in sodium-restricted diets, the Council on Foods and Nutrition has authorized the publication of the following article.

James R. Wilson, M.D., *Secretary.*

NUTRITIONAL IMPLICATIONS OF SODIUM RESTRICTION

In the preparation of diets restricted to less than 0.5 gm. of sodium daily, alterations from the diet usually considered optimum may be necessary. Thus, a reduction in animal protein and the nutrients usually associated with it may be required and certain other foods specifically prepared to remove naturally occurring sodium may be added to the diet. These alterations in the usual dietary customs of persons may sometimes compromise optimum nutrition, a matter to be seriously considered by physicians prescribing a low-sodium diet.

Such changes in the diet as the omission of salt for seasoning, using sweet butter, low-sodium bread, and omitting foods naturally high in sodium should involve no nutritional difficulties, except for any attendant decrease in taste appeal. The same is also true for processed foods packed without added sodium salts.

Limitations in the amount of meat and eggs allowed per day will sometimes be necessary, particularly if the daily sodium intake is to be 200 mg. or less. This involves a decrease in protein of high biological value and of the concomitant vitamins of the B complex.

The processing of foods to remove natural sodium may decrease the content of certain other nutrients. Products such as these are appearing on the market in increasing numbers and may be highly desirable because of the low levels of sodium they provide. At present, the extent of the reduction in nutrients other than sodium in these foods is not known, in many instances, but efforts will be made to procure this information and make it available to physicians.

Thus, the physician prescribing a low-sodium diet must weigh carefully the therapeutic benefits to be expected against the consequences of a possible reduction in the intake of essential nutrients. This may be particularly true of persons already malnourished or with conditioning factors such as defects in digestion and absorption or abnormalities in intermediary metabolism.

The Council will consider for acceptance foods packed or prepared without added sodium provided the food is otherwise acceptable and provided the sodium content appears on the label (in milligrams per 100 gm.) in addition to a statement such as "Packed without added sodium." The Council will also consider foods processed to remove naturally occurring sodium. When significant loss of nutrients, other than sodium, is shown to have occurred, a statement to this effect must also appear on the label and detailed information concerning this reduction in specific nutrients must be made available to the Council and to physicians likely to use the product.

ACCEPTED FOODS

The following product has been accepted as conforming to the rules of the Council.

James R. Wilson, M.D., *Secretary.*

B. M. Mishler Company, Pasadena, California.

Bradford Brand Dietetic Pack Tuna. Ingredients: Light meat tuna and distilled water.

Analysis (submitted by distributor).—Total solids 30.6%, moisture 69.4%, protein (N × 6.25) 28.3%, fat (ether extract) 0.75%, carbohydrates (by difference) 0.15%, sodium 40 mg./100 gm., cholesterol 70 mg./100 gm.

Calories.—1.22 per gram; 35 per ounce.

Use.—A useful adjunct for the planning of low fat, low sodium dietaries.

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PREVENTION OF RABIES

The physician who treats a patient exposed to the bite or saliva of an animal is confronted now with a more difficult problem than in the past. Current developments in the field of rabies prophylaxis have led to conflicting opinions concerning the indications for the Pasteur treatment, the degree of efficacy of this form of treatment, and the incidence of postvaccinal neurological sequelae. In December, 1951, Koprowski and Cox [1] noted in a review that until recent years it was customary to give the Pasteur treatment to all persons exposed to the saliva of an animal that behaved abnormally. There has been a trend, however, toward greater conservatism in using the Pasteur treatment. Behind this change in attitude lie two major factors: the questionable value of the Pasteur treatment in cases in which severe wounds have been inflicted by obviously rabid animals, and the occurrence of a significant number of serious postvaccinal neurological sequelae.

Pait and Pearson,[2] using data collected in the county of Los Angeles for 1940 to 1946, report an incidence of neuroparalytic reactions of 1:600 among persons receiving Semple vaccine, while the chances of acquiring rabies following a known dog bite in the city of Los Angeles varied from 1:1400 to 1:2100. Similarly, in Manhattan among 3,305 patients who received the Pasteur treatment during the period 1946 to 1949, in 6 postvaccinal paralysis developed, an incidence of 1:551.[3] On the other hand, Sellers [4] found 7 cases of post-treatment paralysis among about 50,000 patients treated, an incidence of 1:7000. The exact incidence of these postvaccinal reactions is not known, but a significant number of neuroparalytic sequelae, including some fatalities, do occur. Thus in deciding whether to administer antirabies vaccine the physician must balance the risk of a potential serious postvaccinal reaction against the possibility of the development of rabies.

Indications for vaccine treatment in cases of animal bites were considered by the first session of the WHO Expert Committee on Rabies, held in April, 1950. The committee published [5] detailed indications for the use of vaccine treatment that are designed to avoid, as much as possible, indiscriminate use of the Pasteur treatment. Use of the Pasteur treatment was favored only in cases of bites and licks of uncaptured, wild, or rabid animals and in cases of multiple bites of the head, neck, and shoulders. Similar conservatism has been advocated by Sellers,[6] who considers antirabic treatment contraindicated in the following circumstances: (1) when there is no broken skin anywhere on the body, including face or mouth; (2) if previous wounds are known to be over 24 hours old or are covered with an unbroken scab; (3) if the tooth wounds were made through untorn clothing (such wounds are usually bruises); (4) if exposure is limited to handling only the dog or objects contaminated with the saliva, or to drinking the milk of rabid cows or goats; (5) if the wounds were inflicted not less than seven days prior to the detection of visible signs of the disease; and (6) if the animal remains normal for as long as seven days after inflicting the wounds. Reimmunization should be avoided for borderline exposure regardless of the time elapsed since the last treatment, according to Sellers. He recommends that re-treatment be limited in any case to a short booster series of five or six injections.

The use of antirabies serum, although known since 1889, has never gained prominence; however, since 1948, concentrates of antirabies serum prepared in sheep or rabbits have been available for clinical trial in the United States. The series of human patients treated with antirabies serum is not yet sufficiently large to draw definite conclusions, but Koprowski reports that no person who has received antiserum has died of rabies.[1] At the time of Koprowski's report,[1] in December, 1951, antiserum had been given to 48 persons. Of these, 29 were bitten by animals shown to be rabid. He recommended the administration of the serum as soon as possible after exposure, preferably within 24 hours. He used it in conjunction with vaccine, which may be administered 24 hours after administration of the antiserum, or possibly somewhat later, since rabies antibodies remain present for 7 to 10 days after passive immunization. Serum sickness was noted in about 20% of those treated with rabbit serum and in a much larger number of those treated with sheep serum. Therefore, the production of serum in sheep has been discontinued in favor of horse or rabbit serum. Although the usual precautions to be observed with the use of serums apply here, serum sickness would be less serious than rabies or a serious neuroparalytic complication. Antiserum is particularly suited for therapy of those patients with severe bites of the head or neck in which the Pasteur treatment has been least efficacious. If further clinical studies confirm the successful results obtained in experimental work [7] with the use of antiserum, it may be possible to reduce the number of injections of phenolized vaccine when antiserum is used adjunctively.[1] It should thus be possible to reduce the incidence of neurological sequelae, since it is known that paralytic complications occur more frequently when an increased number of injections of vaccine are given.

A recent departure from the presently used phenolized antirabies vaccine is the development of the chick-embryo-adapted Flury strain of live rabies virus vaccine.[8] This vaccine has been used widely and with apparent effectiveness in the immunization of dogs and gives promise of eliminating the postvaccinal neuroparalytic sequelae, which are thought by most investigators to be due to sensitization to brain tissue. A recent communication [9] from Israel reports that since the start of the antirabies campaign in October, 1950, carried on with the assistance of WHO, the number of rabies cases in animals has dropped from an average of 100 a year, before the start of the campaign, to 10 in the 15 months prior to the report. This program includes a mass vaccination of dogs with egg-adapted vaccine (Flury strain). There were no deaths in humans from rabies in Israel in 1951, whereas there were 20 such deaths between 1948 and 1950. Although it is too early to determine to what extent vaccine has brought about the decline in rabies incidence, it is believed that the mass vaccination has played an important part. The egg-adapted vaccine has proved harmless to dogs and seems to give a greater and longer immunity than other vaccines, according to this communication. This vaccine has also been administered to about 50 persons without undue reactions resulting.[10] It appears to offer possibilities for use in humans but further study of the problem is required.

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[1] Koprowski, H., and Cox, H. R.: Recent Developments in the Prophylaxis of Rabies, Am. J. Pub. Health **41**: 1483-1489 (Dec.) 1951.

[2] Pait, C. F., and Pearson, H. E.: Rabies Vaccine Encephalomyelitis in Relation of the Incidence of Animal Rabies in Los Angeles, Am. J. Pub. Health **39**: 875-877 (July) 1949.

[3] Koprowski, H., and Le Bell, I.: The Presence of Complement-Fixing Antibodies Against Brain Tissue in Sera of Persons Who Had Received Antirabies Vaccine Treatment, Am. J. Hyg. **51**: 292-299 (May) 1950.

[4] Sellers, T. F.: Complications of Antirabic Treatment, J. M. A. Georgia **36**: 30-35 (Jan.) 1947.

[5] Expert Committee on Rabies: Report on the First Session, World Health Organization Technical Report Series, 3-17 (Nov.) 1950.

The ultimate solution to the problem of rabies in human beings is complete eradication of the disease in animals. It has been demonstrated that the disease may be controlled by mass vaccination in dogs and thus prevented in human beings when dogs are the sole source of infection. In view of the widespread geographic distribution of rabies and the many species of animals involved, however, complete eradication in animals is extremely difficult. Therefore, continued research to improve agents for prevention of this disease in exposed persons is indicated. Current developments in rabies prophylaxis, although they do not yet offer a final solution to the problem of rabies prevention, seem to justify an optimistic outlook for the future.

WHY MEDICAL GROUPS DISSOLVE

The Bureau of Medical Economic Research has recently published Bulletin 90, "Discontinuance of Medical Groups, 1940-1949." [1] Of the 441 medical groups in existence at the start of the 1940's, 82 were discontinued during the decade; of these 82, 40 were dissolved entirely, 27 were still in existence but had ceased to meet the requirements of the Bureau's definition of a group, and 15 were reorganized. By restricting their study to the groups that were discontinued and to the reasons therefor, the authors have avoided any discussion of the pros and cons of group practice, particularly from the standpoint of the patient. They have taken a leaf out of research publications in the field of marketing and have tried to set forth the reasons why some groups fall by the wayside, in the hope that such an analysis will be helpful to a physician who is contemplating organizing, joining, or reorganizing a medical group or choosing individual or nongroup partnership practice.

GRANTS BY NATIONAL FUND FOR MEDICAL EDUCATION PAST $2,500,000 MARK

With the awarding of its third series of grants to the medical schools this month, the National Fund for Medical Education has passed the $2,500,000 mark in the funds distributed to the medical schools in the past 12 months.

Included in this total is the $1,417,752 that has been raised by the American Medical Education Foundation to date. The important contributions of the medical profession to this nationwide program to raise funds for medical education has been formally recognized by the National Fund for Medical Education by the following resolution adopted unanimously by the Trustees of the fund at their last meeting.

"The Board of Trustees extend a vote of appreciation to the American Medical Association and the American Medical Education Foundation for the fine progress that they have made, and for their cooperation in turning over their collections to the Fund for distribution by it to the medical schools."

It is thus clear that the medical profession is setting an example of self-help that should stimulate industry and other groups in American society to make their contributions to this important undertaking.

The American Medical Education Foundation has set an annual goal of 2 million dollars to be raised by the medical profession. It is hoped that this goal can be reached for the first time during 1952. Prospects are reasonably good that this can be accomplished. Already 3,255 individual contributions have been received, which is 42.3% more than the total number of contributions received during the entire year 1951. Several state societies are just getting their campaigns into full operation. The Woman's Auxiliary, which has made two handsome contributions from its own funds, is embarking on an active campaign to: (1) stimulate gifts from individual physicians, (2) seek the financial support of over 1,000 auxiliaries, and (3) carry on a vigorous public education program on behalf of the Foundation.

When the medical profession achieves its goal of $2,000,000 annually the National Fund will be well on the way to its goal of raising $5,000,000 in unrestricted funds each year. When this has been accomplished, the more acute financial threats to the stability and continuing high quality of the medical schools should be substantially relieved. Physicians who have not yet contributed to the American Medical Education Foundation are urged to make their contributions promptly either through their state organizations or directly to the Foundation at 535 N. Dearborn Street, Chicago 10.

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6. Cautions Care in Antirabic Vaccine Use, *Selected Reports*, Pub. Health Rep. **66**: 1117-1118 (Aug. 31) 1951.

7. Koprowski, H.: Experimental Studies on Rabies Virus, Canad. J. Pub. Health **40**: 60-67 (Feb.) 1949.

8. Koprowski, H., and Cox, H. R.: Studies on Chick Embryo Adapted Rabies Virus: Culture Characteristics and Pathogenicity, J. Immunol. **60**: 533-554 (Dec.) 1948.

9. Rabies Campaign, Foreign Letters, J. A. M. A. **149**: 1153 (July 19) 1952.

10. Starr, L. E.: What the Physician Should Know About Rabies, South. M. J. **45**: 370-374 (April) 1952.

1. Dickinson, F. G., and Bradley, C. E.: Discontinuance of Medical Groups, 1940-1949, Chicago, Bureau of Medical Economic Research, American Medical Association, 1952. A copy of Bulletin 90 may be obtained by writing to the Bureau, 535 North Dearborn Street, Chicago 10.

MEDICAL NEWS

CALIFORNIA

Discussion of the Normal Child.—The Southern California Society for Mental Hygiene on July 15 held a discussion at the Institute of Aeronautical Sciences in Los Angeles on guides to understanding the growth and development of the normal child. In addition to a community panel, the following physicians participated: Reynold A. Jensen, Minneapolis; Martha G. MacDonald, Palo Alto; C. Morley Sellery, Los Angeles; and Hale F. Shirley and Emmy Sylvester, San Francisco. Dr. Nathan K. Rickles of Beverly Hills, neuropsychiatric consultant, VA Hospital at Sawtelle, was moderator.

Personals.—Dr. Robert Wartenberg, clinical professor of neurology, University of California School of Medicine, San Francisco, has been elected an honorary member of the Italian Neurological Society.——Dr. John H. Aldes, Los Angeles, presented a paper entitled "The Use of Ultrasonic Therapy in the Treatment of Hypertrophic Arthritis with and Without Radiculitus—Geriatric Group" at the International Congress of Physical Medicine in London, England, July 14-19. He also attended the orthopedic association's meeting in London and planned to tour the rehabilitation centers in the Scandinavian countries before returning to the United States.

COLORADO

New Research Head at National Jewish Hospital.—Dr. Gardner Middlebrook, associate in the department of pathology and microbiology at the Rockefeller Institute for Medical Research in New York, has been appointed director of research and laboratories at the National Jewish Hospital in Denver to succeed Dr. Harry J. Corper, who has retired after 32 years of service as the institution's research head. The National Jewish Hospital, opened in 1899, is a nonsectarian tuberculosis center.

CONNECTICUT

Cerebral Palsy Study.—The cerebral palsy study conducted by the Connecticut State Department of Health in 1950 included determinations of the prevalence; the medical, social, psychological, and educational aspects; and the medical, educational, and institutional needs of cerebral palsy patients less than 21 years of age. The rates for children 5 to 9 and 10 to 14 years of age were much higher than for younger and older children. These differences in rates were considered to have been due to poor reporting or recognition of patients among infants and to the eventual adjustment to the diseases, favorable or unfavorable, among older persons. Specific needs of cerebral palsy patients were determined by detailed studies of sample groups of 130 Connecticut children. Less than half the Connecticut patients (34.3%) were considered as having potentialities for improving on an ambulatory system of therapy, and 51.7% of the children were recommended for inpatient care.

DISTRICT OF COLUMBIA

Freedmen's Hospital Interns and Residents.—At the annual session of the Association of Former Internes and Residents of Freedmen's Hospital, Dr. R. Frank Jones, professor of urology at Howard University College of Medicine, was elected president; Dr. Peyton R. Higginbotham, Bluefield, W. Va., president-elect; Dr. Charles R. Chephas, New Brighton, Pa., secretary; and Dr. L. Greeley Brown, Elizabeth, N. J., treasurer. The 31st session will be a memorial to Dr. William A. Warfield Sr., who was surgeon-in-chief of the hospital for 35 years.

ILLINOIS

Test for Insecticide Poisoning.—Testing of blood specimens for the detection of poison from exposure to insecticides is now offered by the diagnostic laboratories of the State Department of Public Health. This new service is made possible by the development in the department's laboratories of a specially prepared heparinized tube for collecting the blood samples. The special tubes may be had on request by physicians who need them for immediate use. Requests should be addressed to Howard J. Shaughnessy, Ph.D., Deputy Director in Charge of Laboratories, 1800 W. Fillmore St., Chicago 12.

The new test is for detection of poisoning with organic phosphates. As poisoning from such phosphates is cumulative, the test may be used best as a preventive, but it is helpful also in confirmation of diagnosis.

Chicago

Gift for Polio Research.—A check for $7,350 to further polio research was presented recently to Michael Reese Hospital by the Merton Davis Memorial Fund. This brings the total contributions of the Merton Davis Memorial Club to $24,450. This club was organized three years ago by a group of high school students in memory of a classmate who died of polio.

Dr. Frank Goes to North Carolina.—Dr. Edward Colman Frank has been appointed associate professor of psychiatry at the University of North Carolina School of Medicine, Chapel Hill, and will assume his new duties Sept. 1. Dr. Frank, who is adjunct psychiatrist at the Psychosomatic Institute of Michael Reese Hospital, is also consultant to Portal House Outpatient Clinic, Chicago; the Family Service Organization, Louisville, Ky.; and the alcoholic unit of the mental health centers of the state of Illinois. Dr. Frank was formerly psychiatrist for the David B. Rotman Research Laboratory of the Psychiatric Institute of the Municipal Court of Chicago.

Dr. Soskin Resigns as Director of Research Institute.—Dr. Samuel Soskin will resign as director of the Medical Research Institute of Michael Reese Hospital and dean of the hospital's postgraduate school, Sept. 1, to enter private practice in his specialty (diabetes and other metabolic and endocrine disorders) in Los Angeles. Dr. Soskin has been a full-time staff member of the hospital since 1929, when he was appointed director of metabolic and endocrine research, and from 1943 to 1950 he served as medical director of the institution. For 20 years he served first as assistant professor and later as professorial lecturer in physiology at the University of Chicago. Recently he has been an associate professor of medicine at Northwestern University Medical School. Dr. Soskin is a past president of the Chicago Society of Internal Medicine and of the Chicago Diabetes Association and a former chairman of the Illinois section of the Society for Experimental Biology and Medicine. He will continue as editor-in-chief of *Metabolism*, a scientific journal, and will devote time to research, teaching, and private practice.

INDIANA

Hospital Fund Drive.—Dr. Lawson J. Clark, Indianapolis, heads a 16 physician committee, which will serve among the physicians for the $12 million hospital fund drive. Pledges will be sought on a five year basis. The Hospital Development Association has already received in pledges a total of $1,400,000.

Anniversary of Medical Foundation.—An invitational open house followed by a dinner marked the 40th anniversary of the South Bend Medical Foundation, Inc. Dr. Alfred S. Giordano has been director of the foundation since 1922. Dr. Robert A. Moore, professor of pathology and dean of the school of medicine, Washington University School of Medicine, St. Louis, addressed the guests.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

NEW YORK

Dr. Simpson Honored.—Dr. Leo F. Simpson of Rochester was recently awarded an honorary degree of doctor of science by St. Bonaventure University as "an outstanding physician and surgeon who has won the universal admiration of colleagues, assistants, patients and the public." Dr. Simpson is a past president of the Monroe County Medical Society, the Rochester Academy of Medicine, and the Medical Society of the State of New York.

New York City

Society News.—The New York Roentgen Society announces election of the following officers: Dr. John L. Olpp, Tenafly, N. J., president; Dr. Irving Schwartz, vice-president; Dr. Harold W. Jacox, secretary; and Dr. Jacob R. Freid, treasurer.

Personals.—Dr. Norman H. Joliffe, director of the Bureau of Nutrition of the New York City Department of Health, was recently elected president of the National Vitamin Foundation, Inc., an industry-supported institution for financing research in nutritional fields.——Dr. H. Houston Merritt, professor of neurology at Columbia University College of Physicians and Surgeons, has been appointed to the advisory committee on virus research and epidemiology of the National Foundation for Infantile Paralysis.

Palsy Center Affiliates with Medical College.—Nassau County's new $500,000 Cerebral Palsy Diagnostic and Treatment Center, recently completed at Roosevelt, L. I., has become affiliated with New York Medical College, Flower-Fifth Avenue Hospitals. The affiliation will make available to the center the services of specialists and at the same time will provide training for medical students and physicians. Dr. Milton J. Wilson, professor of orthopedics, Dr. Lawrence B. Slobody, professor of pediatrics, and Dr. Robert V. Martin, director of the new center, will act with Dr. J. A. Werner Hetrick as an advisory committee. The new center, built through public contributions and with the assistance of local trade unions, which donated thousands of free man-hours of labor, went into full operation July 1. At capacity, it will provide outpatient treatment for 295 children with palsy at a time.

NORTH CAROLINA

Dr. Warren Joins Duke Faculty.—Dr. James V. Warren, president of the American Federation for Clinical Research, has been appointed professor of medicine at The Duke University School of Medicine, Durham. Formerly a professor of medicine and physiology at Emory University School of Medicine, Atlanta, Dr. Warren recently received a grant-in-aid from the American Heart Association for research in heart and blood diseases at Duke. In addition to his duties at Duke, he will help enlarge the medical department of the new Veterans Administration Hospital in Durham. Dr. Warren recently was given a brief leave of absence to serve on a U. S. Navy medical project in Africa.

OHIO

Research in Leukemia.—A foundation to support research in leukemia has been established at Ohio State University College of Medicine through a gift of $18,946 by Dr. and Mrs. James O. Beavis of Dayton, whose son, James Ewing Beavis, was a victim of the disease last summer.

Course in Heart Disease of Children.—A course in heart disease of children will be offered at the Cincinnati Children's Hospital and allied institutions Sept. 16-19. There will be opportunities for examination of patients and observation of new techniques of diagnosis. The course will be restricted to 10 physicians. A fee of $50 will be charged. For information, address the Children's Heart Association, 3231 Burnet Ave. (General Hospital), Cincinnati 29.

State Society Encourages Rural Practice.—The committee on rural health of the Ohio State Medical Association recently inaugurated a series of informal lectures to encourage senior medical students to take up the general practice of medicine as a career. The series of lectures was the culmination of several years of effort on the part of the committee to bring forth practical talks that would present the facts on rural medical practice.

Topics covered in the series included: "Selecting the Place to Practice"; "Type of Practice Encountered"; "Hospital Connections and Emergencies"; "The Economics of a Rural Practice"; and "The Physician and His Community." Dr. J. Martin Byers, Greenfield, chairman of the committee on rural health, and Dr. Edmond K. Yantes, Wilmington, a member of that committee and of the Committee on Medical Service Plans of the Association, served as volunteer lecturers. The committee will meet during the summer to plan with the Ohio State University medical faculty the program for next year. The possibilities of making similar arrangements with both of the other medical schools in the state will be explored.

WEST VIRGINIA

Dr. Kemper Retires as Health Officer.—Dr. Americus Kemper of Clarksburg retired July 1 as health officer for Harrison County after 21 years of service. On the day before his retirement, the staffs of the Harrison County Health Department and the Harrison tuberculosis office, and the school nurse presented him with a camera, and the Harrison County Medical Society gave him a gold watch at a banquet.

Personals.—Dr. David C. Prickett of Weston, health officer for district no. 4, has accepted appointment as director of the Marion County Health Department to succeed Dr. A. Glenn Evans, resigned.——Dr. Benjamin M. Drake of Charleston, deputy state director of health, has resigned to accept appointment as head of the division of preventive medical services of the Kentucky State Department of Health with headquarters in Louisville.——Dr. Frank J. Holroyd of Princeton, past president of the West Virginia State Medical Association, has been reappointed by Governor Patteson as a member of the medical licensing board for the term ending June 30, 1957.——Dr. Hu C. Myers of Philippi has been reappointed by Governor Patteson as a member of the advisory board of the hospital licensing program of the State Department of Health. His term will expire June 30, 1959.

GENERAL

Group Psychotherapy Seminars.—The Group Psychotherapy Institute, New York, has announced the addition of three seminars for the 1952-53 academic year, each to run for 30 consecutive two-hour sessions beginning October, 1952: (1) "Survey Seminar in Group Psychotherapy," (2) "Activity Group Therapy with Children," and (3) "Analytic Group Psychotherapy." Among the instructors will be Drs. Nathan W. Ackerman, Aaron Stein, James J. Thorpe, Joseph J. Geller, and Hyman Spotnitz, New York, and Samuel B. Hadden, Philadelphia. Information may be obtained from the Institute, 105 E. 22nd St., New York 10.

Research Fellowships.—The American College of Physicians announces that a limited number of fellowships in medicine, carrying a stipend of $3,000-$3,500, will be available from July 1, 1953 to June 30, 1954. The fellowships are designed to provide an opportunity for research training in the basic medical sciences or in the application of these sciences to clinical investigation. Assurance must be provided that the applicant will be acceptable in the laboratory or clinic of his choice and that he will be provided with the facilities necessary for the proper pursuit of his work. Application forms, supplied on request to the American College of Physicians, 4200 Pine St., Philadelphia 4, must be submitted in duplicate not later than Oct. 1.

Applications for Grants in Cancer Research.—The Committee on Growth of the National Research Council, acting for the American Cancer Society, is accepting applications for grants in support of cancer research. Applications for new grants received before Oct. 1 will be considered during the winter, and grants recommended at that time will become effective July 1, 1953. Investigators now receiving support will be notified individually regarding application for renewal of these grants. The research program includes, in addition to clinical investigations on cancer, fundamental studies in the fields of cellular physiology, morphogenesis, genetics, virology, biochemistry, metabolism, nutrition, cytochemistry, physics, radiobiology, chemotherapy, endocrinology, and environmental cancer. During the past year the American Cancer Society, on recommendation of the Committee on

Growth, has awarded approximately 250 grants totaling about $1,700,000. Application blanks may be obtained from the Executive Secretary, Committee on Growth, National Research Council, 2101 Constitution Ave., Washington 25, D. C.

Rocky Mountain Radiological Society Conference.—The annual Mid-Summer Conference of the Rocky Mountain Radiological Society will be held in Denver at the Shirley-Savoy Hotel Aug. 7-9. Dr. Philip J. Hodes, Philadelphia, will discuss the roentgen manifestations of (1) pancreatic and periampullary disease and (2) cerebellopontine angle tumors; Dr. Harold O. Peterson, St. Paul, will speak on the roentgen diagnosis of benign gastric ulcer and unusual neurological conditions with diagnostic roentgen findings; Dr. George H. Ramsey, Rochester, N. Y., will present cinefluorographic analysis of (1) some movements of the cervical spine and (2) the causes of dysphagia; and Dr. A. Justin Williams, San Francisco, will speak on the roentgen diagnosis of intra-abdominal hernia.

A symposium on diseases of animals transmissible to man will be presented. Evening meetings will include a joint session with the Denver Medical Society, followed by Dutch lunch, and an informal banquet. The annual picnic on Saturday afternoon will conclude the conference.

National Medical Association.—The National Medical Association, Inc., will meet in Chicago, Aug. 11-15, under the presidency of Dr. Joseph G. Gathings of Washington, D. C. Headquarters of the meeting will be at the DuSable High School, Wabash Ave. and 50th St. On Monday morning there will be a panel discussion on "The President's Committee on the Health Needs of the Nation." Tuesday afternoon "Late Syphilis" will be presented as a symposium and illustrated lecture. The Wednesday morning session will open with the annual Stubbs Lecture on "Interesting Lesions of the Gastrointestinal Tract" by Dr. Ellsworth E. Hasbrouck, Chicago. On Wednesday afternoon there will be symposiums on modern obstetrical procedures and leukorrhea. A symposium on gastritis will be held Thursday afternoon, and on Friday time will be given to psychiatric problems that confront the general practitioner. Another symposium, to be held at the same time, will deal with common fractures of the upper and lower extremities.

Photography in Medicine and Biology.—How to make photography serve more effectively in medicine and biology is the subject of a symposium at the Hotel New Yorker, New York. Aug. 12 as part of the convention of the Photographic Society of America. Among many others, Dr. David S. Ruhe of New York, director of the Audio-Visual Institute of the Association of American Medical Colleges, will speak on "The Changing Ideology of Medical Teaching Motion Pictures."

Techniques and equipment for the types of photography carried on in a general hospital will be described by Leonard A. Julin, of the Mayo Clinic, Rochester, Minn. Dr. Dan H. Gordon, professor of ophthalmologic surgery at Cornell University Medical College, will review the rapid advances in techniques for photographing the eye. Co-chairmen of the symposium are Dr. Milton G. Bohrod of Rochester, N. Y., General Hospital, and H. Lou Gibson of the Medical Division of Eastman Kodak Company. Registration fee for the day is $2. The Photographic Society of America has headquarters at 2005 Walnut St., Philadelphia 3.

Physical Medicine Congress.—The annual scientific session and instruction seminar of the American Congress of Physical Medicine will be held Aug. 25-29 at the Roosevelt Hotel, New York, under the presidency of Dr. Robert L. Bennett of Warm Springs, Ga. Lecturers for the instruction seminar include: Drs. Charles O. Bechtol, San Francisco; Robert L. Bennett, Warm Springs, Ga.; Frances A. Hellebrandt and Meyer A. Perlstein, Chicago; Frank H. Krusen, Rochester, Minn.; Allen S. Russek and Joseph D. Sullivan, New York; and Malford W. Thewlis, Wakefield, R. I. The charge for the eight lectures is $15.

On Monday there will be a luncheon of the Advisory Committee on Education, Council on Physical Medicine and Rehabilitation, American Medical Association. This will be followed by an educational conference, at which the following topics will be discussed: "University Sponsored Graduate Fellowship Training in the Medical Specialties," "Method of AMA Residency Approval," "Undergraduate Instruction in Physical Medicine and Rehabilitation," and "Report of the Advisory Committee of the Council on Physical Medicine and Rehabilitation of the American Medical Association." On Wednesday Dr. Frank H. Krusen, professor of physical medicine and rehabilitation, Mayo Foundation, will deliver the second John Stanley Coulter Memorial Lecture on "Report on the International Congress of Physical Medicine." Wednesday evening the annual congress dinner will be held.

On Thursday at 10 a. m. there will be a rehabilitation symposium, with the following presentations:

Howard A. Carter, M.E., Chicago, Rehabilitation of the Hard of Hearing.

Samuel S. Sverdlik, New York, Evaluation of Speech Training in the Rehabilitation of the Aphasic.

Everill W. Fowlks, Portland, Ore., Patient Evaluation by Wechsler Bellevue Test.

Charles H. Reagan, Tuscaloosa, Ala., Rehabilitation of 120 Lobotomized Patients in a VA Hospital.

Edith L. Kristeller, New York, Work Program for the Disabled Housewife.

Marion Strauss Lesser, M.A., and Robert C. Darling, New York, Some Follow-Up Studies in Rehabilitation at the Institute for the Crippled and Disabled.

Adam Peylan, Tel-Aviv, Israel, Combined Use of Hydrotherapy and Sweating Cure in Certain Arthritis Problems.

Heart Association Seeks Research Applicants.—New research applications in cardiovascular and related fields are being accepted by the American Heart Association for studies to be conducted during the year beginning July, 1953, according to Dr. Louis N. Katz, Chicago, chairman of the association's scientific council. Research fellowships are open to graduates of approved medical and graduate schools who are interested in research and who plan to follow an academic career. Established investigatorships are open to persons of proved ability who have the degree of Doctor of Medicine, Doctor of Philosophy, Doctor of Science, or the equivalent, and who are interested in a career in research. Grants-in-aid are available to nonprofit institutions for a specified program of research, including work in the basic sciences, under the direction of an experienced investigator.

The research committee recently voted to increase the stipends granted to research fellows and established investigators. Under the new scale, research fellowships, which are granted for a one year period, range from $3,500 to $5,500. Established investigatorships, which may be extended annually for a five year period, range from $6,000 to $9,000. Grants-in-aid are awarded in varying amounts, usually not exceeding $10,000, and the period covered is variable, depending on the particular program of study. Applications for research fellowships and established investigatorships should be submitted by Sept. 15. Applications for research grants-in-aid may be filed up to Dec. 1. Information may be obtained from the Medical Director, American Heart Association, 1775 Broadway, New York 19.

International Surgeons Meeting.—The annual assembly of the U. S. and Canadian chapters of the International College of Surgeons will take place at the Conrad Hilton Hotel in Chicago, Sept. 2-5. Dr. Henry W. Meyerding, Rochester, Minn., is president of the U. S. chapter and Dr. Lyon H. Appleby, Vancouver, B. C., president of the Canadian chapter. The assembly will open with a gallbladder symposium Tuesday morning. During the luncheon in the North Ballroom (11:45 a. m. to 1:45 p. m.), there will be a panel on the symposium, with Dr. Arnold S. Jackson of Madison as moderator. The Tuesday evening session will be devoted to a symposium on antibiotics. The clinic, Wednesday morning, will be followed by a roundtable discussion on treatment of duodenal ulcers, and the evening session will be devoted to a film forum. The Thursday morning program will feature recent developments in clinical research. During this session the Herbert Acuff Memorial Address on "Feasibility of Prolonged Survival Following Total Pelvic Exenteration for Advanced Cancer" will be delivered at 9 a. m. by Dr. Alexander Brunschwig, New York, and the Custis Lee Hall Memorial Address on "Volkman's Ischaemic Contraction" will be given at 10 a. m. by Dr. Meyerding. On Thursday at 4:10 p. m. there will be a roundtable discussion on surgery of the colon, rectum, and anal canal.

The speaker at the banquet, Thursday evening, is Mr. Arthur Dickson Wright, F.R.C.S., surgeon at St. Marys Hospital and

Prince of Wales Hospital, London, and a member of the council of the Royal College of Surgeons of England.

The convocation will be held at the Civic Opera House at 7 Friday evening. The Right Hon. Lord Horder, G.C.V.O., M.D., F.R.C.P., F.R.C.S., of London, England, will deliver an address on "Freedom in Medicine."

Two special features of the meeting will be the program on Tuesday of a new section devoted to the basic sciences allied to surgery, and a special program for operating room nurses.

Among the foreign surgeons who are expected to make presentations during the session are the following: Dr. Bernhard Zondek, head of the hormone research laboratory, Hadassah Medical School of the Hebrew University of Jerusalem; Dr. Raymond Darget, professor of urology at the University of Bordeaux; Dr. Lucien Leger, assistant professor of surgery, University of Paris; and Dr. Antonio Puigvert Gorro, director of the Institute of Urology, University of Barcelona.

Prevalence of Poliomyelitis.—According to the National Office of Vital Statistics, the following number of reported cases of poliomyelitis occurred in the United States and its territories and possessions in the weeks ended as indicated:

Area	July 12, 1952	July 5, 1952	July 14, 1951
New England States			
Maine	3	2	1
New Hampshire	..	..	..
Vermont	1	1	..
	6	2	17
	..	..	..
	2	2	2
Middle Atlantic States			
New York	26	24	26
New Jersey	10	2	7
Pennsylvania	8	4	11
East North Central States			
Ohio	41	45	24
Indiana	5	2	8
Illinois	33	4	40
Michigan	19	13	24
Wisconsin	5	3	15
West North Central States			
Minnesota	17	4	7
Iowa	72	27	12
Missouri	5	10	13
North Dakota	..	1	4
South Dakota	15	2	2
Nebraska	35	16	7
Kansas	19	17	8
South Atlantic States			
	..	..	..
	2	..	6
	5	..	..
Virginia	13	10	4
West Virginia	17	5	3
North Carolina	11	8	6
South Carolina	..	..	3
Georgia	18	11	32
Florida	38	10	18
East South Central States			
Kentucky	18	2	1
	21	12	16
	13	17	27
	38	26	16
West South Central States			
	9	10	12
	60	19	29
	47	26	22
Texas	296	186	92
Mountain States			
Montana	5	1	1
Idaho	3	1	..
	1	..	2
	10	6	20
	22	16	3
	7	7	5
Utah	..	1	2
Nevada	..	..	..
Pacific States			
Washington	18	14	5
Oregon	7	9	4
California	46	43	58
Territories and Possessions			
Alaska	..	..	..
Hawaii	11	18	..
Puerto Rico	2	..	3
Total	1,060	639	618

CORRECTION

Florida Medical Officers.—In The Journal, July 12, page 1038, Dr. Samuel M. Day, Jacksonville, was listed as editor of the *Journal of the Florida Medical Association.* Dr. Day was re-elected secretary-treasurer of the association, and Dr. Shaler A. Richardson, Jacksonville, was re-elected editor of the journal.

EXAMINATIONS AND LICENSURE

BOARDS OF MEDICAL EXAMINERS

Alabama: *Examination.* Montgomery, June 23-25. Sec., Dr. D. G. Gill, 537 Dexter Ave., Montgomery.

Alaska:* *Examination.* Juneau. On application in other towns where there are board members. *Reciprocity.* On application. Sec., Dr. W. M. Whitehead, Box 140, Juneau.

Arkansas:* *Examination.* Little Rock, Nov. 6-7. Sec., Dr. Joe Verser, Harrisburg.

California: *Written.* Los Angeles, Aug. 18-21; Sacramento, Oct. 20-23. Application must be received at least two weeks prior to date of examination. *Oral for Reciprocity Applicants.* Los Angeles, Aug. 16; San Francisco, Nov. 8. Applicants are requested not to arrange to come to an oral examination until they receive notice from the Credentials Committee. *Oral and Clinical for Foreign Medical School Graduates.* Los Angeles, Aug. 17; San Francisco, Nov. 9. Sec., Dr. Frederick N. Scatena, 1020 N St., Sacramento.

Colorado:* *Reciprocity.* Denver, Oct. 14. Exec. Sec., Mrs. B. H. Hudgens, 831 Republic Bldg., Denver.

District of Columbia:* *Reciprocity.* Washington, Sept. 8. Sec., Dr. Daniel L. Seckinger, 4130 E. Municipal Bldg., Washington.

Florida:* *Examination.* Jacksonville, Nov. 23-25. Sec., Dr. Homer L. Pearson, 701 Dupont Bldg., Miami 32.

Georgia: *Reciprocity.* Atlanta, Oct. 14-15. Sec., Mr. R. C. Coleman, 111 State Capitol, Atlanta 3.

Guam: The Commission on Licensure will meet whenever a candidate appears or submits his credentials. Ex. Sec., Dr. Austin W. Matthis, Agana.

Illinois: *Examination.* Chicago, Sept. 23-25. Supt. of Regis., Mr. Charles F. Kervin, Capitol Bldg., Springfield.

Kansas: Topeka, Dec. 10-11. Sec., Dr. O. W. Davidson, 864 New Brotherhood Bldg., Kansas City.

Michigan:* *Examination.* Lansing, Oct. 8-10. Sec., Dr. J. Earl McIntyre, 202-4 Hollister Bldg., Lansing.

Minnesota:* Minneapolis, Oct. 21-23. Sec., Dr. J. F. Du Bois, 230 Lowry Medical Arts Bldg., St. Paul 2.

Mississippi: *Reciprocity.* Jackson, December. Asst. Sec., Dr. R. N. Whitfield, State Board of Health, Jackson 113.

Montana: Helena, Oct. 6-8. Sec., Dr. S. A. Cooney, 214 Power Block, Helena.

Nebraska:* *Examination.* Omaha, June 1953. Director, Mr. Husted K. Watson, Room 1009 State Capitol Bldg., Lincoln 9.

Nevada:* Carson City, Aug. 4. Sec., Dr. George H. Ross, 112 Curry St., Carson City.

New Hampshire: Concord, Sept. 10. Sec., Dr. John S. Wheeler, 107 State House, Concord.

New Jersey: *Examination.* Trenton, Oct. 21-24. Sec., Dr. E. S. Hallinger, 28 W. State St., Trenton.

North Carolina: *Reciprocity.* Durham, Oct. 13. Sec., Dr. Joseph J. Combs, 419 Professional Bldg., Raleigh.

Ohio: *Reciprocity.* Columbus, Oct. 7. Sec., Dr. H. M. Platter, 21 W. Broad St., Columbus.

Oregon:* *Reciprocity.* Portland, July 25-26. Final date for filing application was June 16. Exec. Sec., Mr. Howard I. Bobbitt, 609 Failing Bldg., Portland.

South Carolina: *Reciprocity.* Columbia, Sept. 3. Sec., Mr. N. B. Heyward, 1329 Blanding St., Columbia.

Tennessee:* *Examination.* Memphis, Oct. 1-2. Sec., Dr. H. W. Qualls, 1635 Exchange Bldg., Memphis.

Texas: *Reciprocity.* Fort Worth, Aug. 3. Sec., Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth.

Wyoming: Cheyenne, Oct. 6. Sec., Dr. Franklin D. Yoder, State Office Bldg., Cheyenne.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

Alaska: *Examination.* On application. Juneau or other towns in Territory as decided by Board. *Reciprocity.* On application. Sec., Dr. C. Earl Albrecht, Box 1931, Juneau.

Arizona: *Examination.* Tucson, Sept. 16. Sec., Mr. Herbert D. Rhodes, Room 423, Liberal Arts Bldg., University of Arizona, Tucson.

Arkansas: *Examination.* Little Rock, Oct. 1-2. Sec., Mr. Louis E. Gebauer, 1002 Donaghey Bldg., Little Rock.

Colorado: *Examination.* Denver, Sept. 10-11. Sec., Dr. Esther B. Starks, 1459 Ogden St., Denver 18.

District of Columbia: *Examination.* Washington, Oct. 20-21. Sec., Dr. Daniel L. Seckinger, 4130 E. Municipal Bldg., Washington.

Florida: *Examination.* Gainesville, Oct. 25. Sec., Mr. M. W. Emmel, Box 340, University of Florida, Gainesville.

Michigan: *Examination.* Detroit and Ann Arbor, Oct. 10-11. Sec., Mrs. Anne Baker, 423 W. Michigan Ave., Lansing.

Minnesota: *Examination.* Minneapolis, Oct. 7-8. Sec., Mr. Raymond N. Bieter, 105 Millard Hall, University of Minnesota, Minneapolis.

Nebraska: *Examination.* Omaha, Oct. 7-8. Director, Bureau of Examining Boards, Mr. Husted K. Watson, 1009 State Capitol Bldg., Lincoln.

New Mexico: *Examination.* Santa Fe, Sept. 21. Sec., Mrs. Marguerite Cantrell, Box 1592, Santa Fe.

Oklahoma: *Examination.* Oklahoma City, Sept. 13. Sec., Dr. Clinton Gallaher, 813 Braniff Bldg., Oklahoma City.

Oregon: *Examination.* Portland, Sept. 6 and Dec. 6. Sec., Dr. Charles D. Byrne, State Board of Higher Education, University of Oregon, Eugene.

Rhode Island: *Examination.* Providence, Aug. 13. Administrator of Professional Regulations, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

Texas: *Examination.* Austin, October. Sec., Brother Raphael Wilson, 407 Perry-Brooks Bldg., Austin.

Wisconsin: *Examination.* Madison, Sept. 13, Milwaukee, Dec. 6. Sec., Mr. W. H. Barber, 621 Ransom St., Ripon.

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* Basic Science Certificate required.

MEETINGS

Alaska Territorial Medical Association, Anchorage, Aug. 21-23. Dr. William P. Blanton, P. O. Box 2569, Juneau, Secretary.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, The Homestead, Hot Springs, Va., Sept. 4-6. Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Texas, Secretary.

American College of Surgeons, Waldorf-Astoria, New York, Sept. 22-26. Dr. Michael L. Mason, 40 East Erie St., Chicago 11, Secretary.

American Congress of Physical Medicine, Roosevelt Hotel, New York, Aug. 25-29. Dr. Frances Baker, One Tilton Ave., San Mateo, Calif., Secretary.

American Hospital Association, Philadelphia, Sept. 15-18. Mr. George Bugbee, 18 East Division St., Chicago 10, Executive Director

American Medical Association Public Relations Institute, Edgewater Beach Hotel, Chicago, Sept. 4-5. Mr. Leo E. Brown, 535 N. Dearborn St., Chicago 10, Director.

American Physiological Society, New Orleans, Sept. 4-6. Dr. E. F. Adolph, University of Rochester School of Medicine and Dentistry, Rochester, N. Y., Secretary.

American Roentgen Ray Society, Shamrock Hotel, Houston, Texas, Sept. 23-26. Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Secretary.

American Society of Maxillofacial Surgeons, Hotel del Coronado, San Diego, Calif., Sept. 10-13. Dr. Casper M. Epsteen, 25 East Washington St., Chicago 2, Secretary.

Biological Photographic Association, Hotel New Yorker, New York, Sept. 10-12. Mr. Lloyd E. Varden, Pavelle Color, Inc., 533 W. 57th St., New York 19, Secretary.

Colorado State Medical Society, Stanley Hotel, Estes Park, Sept. 9-12. Mr. Harvey T. Sethman, 835 Republic Bldg., Denver 2, Executive Secretary.

Medical Society of Delaware, Rehoboth, Sept. 8-10. Dr. Andrew M. Gehret, 822 North American Bldg., Wilmington, Secretary.

Michigan State Medical Society, Book-Cadillac Hotel, Detroit, Sept. 24-26. Dr. L. Fernald Foster, 606 Townsend St., Lansing 15, Secretary.

Montana Medical Association, Florence Hotel, Missoula, Sept. 18-21. Mr. L. R. Hegland, 104 North Broadway, Billings, Executive Secretary.

National Medical Association, Chicago, Aug. 11-15. Dr. John T. Givens, 1108 Church St., Norfolk 10, Va., General Secretary.

Neurosurgical Society of America, The Cloister, Sea Island, Ga., Sept. 18-20. Dr. C. D. Hawkes, 22 North Manassas St., Memphis 5, Tenn., Secretary.

Nevada State Medical Association, Reno, Aug. 21-23. Dr. William A. O'Brien III, 505 Chestnut St., Reno, Secretary.

New Hampshire Medical Society, Mt. Washington Hotel, Bretton Woods, Sept. 7-9. Dr. Deering G. Smith, 44 Chester St., Nashua, Secretary.

North Pacific Pediatric Society, Challenger Inn, Sun Valley, Idaho, Sept. 19-20. Dr. S. Gorham Babson, 1107 S.W. 13th Ave., Portland 5, Ore., Secretary.

North Pacific Society of Internal Medicine, Sun Valley, Idaho, Sept. 19-20. Dr. Robert L. King, 1115 Terry Ave., Seattle 1, Wash., Secretary.

North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, Texas, Sept. 17. Dr. E. Aubrey Cox, Hamilton Bldg., Wichita Falls, Texas, Chairman.

Reno Surgical Society Conference, Reno, Nevada, Aug. 21-23. Dr. Olin C. Moulton, 130 N. Virginia St., Reno, Nevada, Chairman.

Rocky Mountain Radiological Society, Denver, Aug. 7-9. Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Neb., Secretary.

U. S. Chapter, International College of Surgeons, Conrad Hilton Hotel, Chicago, Sept. 2-5. Dr. Arnold S. Jackson, 1516 Lake Shore Drive, Chicago, Executive Secretary.

Utah State Medical Association, Salt Lake City, Sept. 4-6. Dr. T. C. Weggeland, 42 S. Fifth East St., Salt Lake City, Secretary.

Vermont State Medical Society, Mt. Washington Hotel, Bretton Woods, N. H., Sept. 7-9. Dr. James P. Hammond, 128 Merchants Row, Rutland, Secretary.

Washington State Medical Association, Olympic Hotel, Seattle, Sept. 13-17. Dr. Bruce Zimmerman, 338 White-Henry-Stuart Bldg., Seattle, Secretary.

INTERNATIONAL

Aero Medical Association Interim Meeting, Paris, France, Sept. 26-28. Dr. Armand Robert, 2 Rue Marbeuf, Paris 8e, France, Secretary.

American Congress of Industrial Medicine, Rio de Janeiro, Brazil, Sept. 20-28. Prof. Dr. Jose Pedro Reggi, Calle Arenales No. 981 Buenos Aires, Argentina, Director General.

Australasian Medical Congress, Melbourne Victoria, Australia, Aug. 22-30. Dr. C. H. Dickson, Medical Society Hall, 426 Albert St., East Melbourne, Victoria, Australia, General Secretary.

European Congress of Cardiology. University of London, Bloomsbury, W.C.1, England, Sept. 10-12. Dr. K. Shirley Smith, 35 Wimpole St., London, W.1, England, Secretary.

European Society of Cardiovascular Surgery, Strasbourg, France, Oct. 3-4. Sir James Learmonth, Department of Surgery, University New Buildings, Edinburgh 8, Scotland.

Interamerican Cardiological Congress, Buenos Aires, Argentina, Aug. 31-Sept. 7. Dr. Blas Moia, Larrea 1132, Buenos Aires, Argentina, Secretary-General.

Inter-American Congress of Public Health, Havana, Cuba. Sept. 26-Oct. 1. Pan-American Sanitary Bureau, 2001 Connecticut Ave., Washington, D. C.

Inter-American Congress of Radiology, Mexico City, Mexico, Nov. 2-8. Dr. Guido Torres Martinez, Marsella No. 11, Mexico, D.F., Secretary-General.

International Congress of Anthropology and Ethnology, Vienna, Austria, Sept. 1-8. For information write: Secretaries, Royal Anthropological Institute, 52 Upper Bedford Place, London, W.C.1, England.

International Congress on Diseases of the Chest, Rio de Janeiro, Brazil, Aug. 24-30. Mr. Murray Kornfeld, 112 East Chestnut St., Chicago 11, Illinois, Executive Secretary.

International Congress of Haematology, Mar del Plata, Argentina, Sept. 21-26. Dr. Carlos Reussi, Anchorena 1710, Buenos Aires, Argentina, Secretary.

International Congress of the History of Medicine, Nice-Cannes, France, and Monaco, Sept. 8-14. Dr. F. A. Sondervorst, 124 Avenue des Allies, Louvain, Belgium, Secretary-General.

International Congress on Hydatid Disease, Santiago, Chile, Nov. 21-24. Organizing Committee, P. O. Box 9183, Santiago, Chile.

International Congress of Internal Medicine, Friends House, London, N.W.1, Eng. Sept. 15-18. Sir Harold Boldero, 12 Pall Mall East, London, S.W.1, England, Secretary.

International Congress of Medical Press, Venice, Italy, Oct. 4-5. Dr Bruno de Biasio, S. Marco 4646, Venice, Italy, Secretary.

International Congress on Medical Records, London, England, Sept. 7-12. Miss Gwen Perkins, 120 C Street S.E., Washington, D. C., Chairman.

International Congress of Military Medicine and Pharmacy, Montevideo, Uruguay, March 1-7, 1953. Dirreccion General del Servico de Sanitad Militar, 8 de Octubre y Mariano Moreno, Montevideo, Uruguay, Secretariat of the Congress.

International Congress on Neuropathology, Rome, Italy, Sept. 8-13. Dr. Armando Ferraro, 722 W. 168th St., New York, N. Y., U. S. A., Secretary General.

International Congress of Otorhinolaryngology, Amsterdam, Netherlands, June 8-15, 1953. Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam, Netherlands, Secretary.

International Congress of Therapeutics, Madrid, Spain, Oct. 16-18. Professor Armijo, Instituto de Farmacologia, Cuidad Universitaria, Madrid, Spain, Secretary.

International Congress of Urology, The Waldorf-Astoria, New York, N. Y., U. S. A., Sept. 15-18. Dr. John A. Taylor, 2 East 54th St., New York, N. Y., U. S. A., Secretary-General.

International Hospital Congress, London, England, May 25-30, 1953. Capt. J. E. Stone, 10 Old Jewry, London, EC2, England, Hon. Secretary.

International Study Conference on Child Welfare, Bombay, India, Dec. 5-12. For information write: All India Save the Children Committee, 5 Carmichael Road, Bombay, India.

International Union Against Tuberculosis, Rio de Janeiro, Brazil, Aug. 24-27. Prof. Etienne Bernard, 66 Blvd. St. Michel, Paris 6e, France, Secretary-General.

Medical Women's International Association, Vichy, France, Sept. 12-16. Dr. Margaret M. Wurts, 27 East Wellesley Road, Montclair, N. J., U. S. A., National Secretary.

Neuroradiologic Symposium, Stockholm, Sweden, Sept. 17-20. Docen Ake Lindbom, Symposium Neuroradiologicum, Serafimerlasarettet, Stockholm K., Sweden, Secretary.

World Congress of Jewish Physicians, Jerusalem, Israel, Aug. 10-14 Secretariat: % Israel Medical Association, Rehov Mesilat Yesharim Beth Kovatz, Jerusalem, Israel.

World Congress of Medicine, Montecatini, Italy, Oct. 16-18. Secretariat: Corso Trieste, 65, Rome, Italy.

World Federation of Mental Health, Brussels, Belgium, Aug. 25 Sept. 1. Dr. J. R. Rees, 19 Manchester St., London, W.1, Englanc Secretary.

World Medical Association, Athens, Greece, Oct. 12-16. Dr. Louis Bauer, 2 East 103d St., New York 29, N. Y., Secretary General.

DEATHS

Beck, Carl ⊕ Sayre, Pa.; born in Milin, Austria, March 26, 1864; Deutsche Universität Medizinische Fakultät, Prague, Austria, 1889; at one time assistant in surgery and gynecology in the clinics at Prague; made several trips to America as steamer surgeon in 1889-1890, settling in Chicago in the latter year to follow the general practice of medicine; served as volunteer in the 11th Infantry Regiment, Austrian Army, and later first lieutenant of the 75th Infantry Regiment; member of the military council and teacher of plastic surgery to military surgeons during World War I; for many years professor of surgical pathology and clinical pathology at the University of Illinois College of Medicine; formerly professor of surgery at the Post Graduate Hospital and Medical School and the Chicago College of Dentistry; fellow and a founder of the American College of Surgeons; past president of the Chicago Surgical Society; a founder of the German Medical Society and the Bohemian Medical Society of Chicago; member of the Illinois State Medical Society, German Society of Surgeons of Berlin, Society of Physicians of Prague, and the Royal Academy of Rome; founder and chief of staff of surgery, North Chicago Hospital; founded St. Anthony Hospital, with Sisters of Joliet; and was attending surgeon at Cook County and St. Joseph hospitals; originator of bismuth paste treatment for chronic sinusitis under name of Beck's paste; author of "Principles of Surgery," 1905, and "Crippled Hand and Arm," 1925, as well as of many articles in medical journals; died July 21, aged 88.

Brodhead, George Livingston ⊕ New York; born in New Orleans Oct. 14, 1869; College of Physicians and Surgeons, medical department of Columbia College, New York, 1891; an Associate Fellow of the American Medical Association; past president of the Medical Association of the Greater City of New York; member of the New York Academy of Medicine and New York Obstetrical Society; specialist certified by the American Board of Obstetrics and Gynecology; instructor in obstetrics at his alma mater from 1895 to 1897 and the New York University and Bellevue Hospital Medical College from 1899 to 1917; professor of obstetrics at the New York Post Graduate Medical School and Hospital from 1899 to 1916; served on the staff of St. Luke's Hospital in Newburgh, Letchworth Village in Thiells, United Hospital in Port Chester, Jamaica (N. Y.) Hospital, St. Agnes Hospital in White Plains, and Knickerbocker, Jewish Maternity, Lutheran, Union, and Bronx Maternity hospitals all in New York; author of "Approaching Motherhood"; died May 13, aged 82, of bronchopneumonia.

Kemper, John Willard ⊕ Ann Arbor, Mich.; born in Richmond, Ind., Sept. 14, 1891; University of Michigan School of Dentistry in Ann Arbor in 1917 and University of Michigan Medical School in 1927; joined the university staff as instructor in oral surgery in 1929; the following year became assistant professor, and in 1934 was elevated to the rank of associate professor; promoted to full professorship in 1935, and in the same year became head of the department of oral surgery and consulting dental surgeon to the University Hospital; for many years affiliated with St. Joseph's Mercy Hospital; specialist certified by the American Board of Plastic Surgery; fellow of the American College of Surgeons; first vice-president and second president of the American Society of Maxillofacial Surgeons; served during World War I; associate editor of the *American Journal of Orthodontics and Oral Surgery*; died in the University Hospital May 8, aged 60, of dissecting aneurysm of the aorta with cardiac tamponade.

Jones, Marvin Fisher ⊕ New York; born in Spencer, N. Y., March 10, 1889; University of Buffalo School of Medicine, 1913; specialist certified by the American Board of Otolaryngology; at one time on the faculty of New York Post Graduate Medical School of the New York University-Bellevue Medical Center; served in France during World War I; member of the American Academy of Ophthalmology and Otolaryngology, American Laryngological Association, American Ophthalmological Society, and the American Otological Society; fellow of the American College of Surgeons; consultant, Fitkin Memorial Hospital in Neptune, N. J., Tioga County General Hospital in Waverly, New York Hospital of New York, and United Hospital in Port Chester; since 1936 surgeon-director, department of otolaryngology at Manhattan Eye, Ear and Throat Hospital, where he was in charge of its conservation of hearing service, and where he died May 26, aged 63, of multiple myeloma and cardiac failure.

Gillick, David Walter ⊕ Talihina, Okla.; born in St. Louis, Aug. 9, 1895; St. Louis College of Physicians and Surgeons, 1919; fellow of the American College of Physicians; since Sept. 21, 1921, physician in the Indian Service, Department of Interior; medical officer in charge of Talihina Indian Medical Center, and during 1951-1952 was designated acting area medical officer for Eastern Oklahoma; served as superintendent and physician in charge of Shawnee Indian Sanatorium in Shawnee, and as district medical director of states, including Oklahoma, Kansas, Mississippi, North Carolina, and Florida; died in Muskogee March 29, aged 56, of cerebral hemorrhage.

Reeves, Thaddeus Benjamin ⊕ Greenville, S. C.; University of Virginia Department of Medicine, Charlottesville, 1914; member of the founders group of the American Board of Surgery; for two years instructor in anatomy at his alma mater; member of the Southern Surgical Association and the Alumni Association of the Mayo Foundation; fellow of the American College of Surgeons; formerly fellow in surgery at Mayo Foundation, Rochester, Minn.; attending surgeon at the Greenville General Hospital and the St. Francis Hospital; consulting surgeon at the Shriners Hospital for Crippled Children; orthopedic surgeon for the Southern Railway and the Piedmont and Northern Railway; died June 9, aged 66.

Carlisle, Chester Lee, Palo Alto, Calif.; Western Reserve University Medical Department, Cleveland, 1898; specialist certified by the American Board of Psychiatry and Neurology; member of the American Psychiatric Association and the Association for Research in Nervous and Mental Diseases; retired in July, 1946, after 15 years as clinical director of the Veterans Administration Hospital; served in state and federal hospitals for a period of 45 years; a captain in the medical corps of the U. S. Army during World War I; chief of division of mental defect and delinquency, New York State Board of Charities, from 1917 to 1920; formerly connected with the U. S. Public Health Service; died April 29, aged 75.

Kennedy, William Aloysius ⊕ St. Paul; born in Minneapolis Aug. 1, 1893; University of Minnesota Medical School, Minneapolis, 1918; member of the American Academy of Ophthalmology and Otolaryngology; specialist certified by the American Board of Ophthalmology; formerly secretary-treasurer of the Minnesota Academy of Ophthalmology and Otolaryngology; served during World War I; at one time a fellow in ophthalmology at Mayo Foundation; affiliated with Children's Hospital, Gillette State Hospital, and St. Joseph's Hospital, where he died April 30, aged 58, of right amyotrophic lateral sclerosis, cerebral hemorrhage, and terminal pneumonia.

Urie, James William ⊕ Wilmington, Del.; Hahnemann Medical College and Hospital of Philadelphia, 1929; served on Leyte and Luzon in the Philippines during World War II; during the campaign on Luzon was awarded the Bronze Star for heroic action in rescuing several wounded men; physician to Conrad High School football teams, and in 1950 health officer for the Memorial Hospital, where he served as secretary to hospital medical staff, and registrar for its school of nursing; affiliated with Wilmington General Hospital; died April 17, aged 46, of acute coronary thrombosis.

⊕ Indicates Member of the American Medical Association.

Beer, James Anderson ⊕ Columbus, Ohio; Ohio Medical University, Columbus, 1906; formerly assistant professor, university health service, Ohio State University College of Medicine; for many years city bacteriologist and at one time city health commissioner; served as treasurer of the Ohio State Medical Association; member of the American Association for the Advancement of Science; honorary member of the Columbus Academy of Medicine; died in White Cross Hospital April 30, aged 72, of cerebral hemorrhage.

Betts, George Seymour ⊕ Banner, Ill.; Medical Department of Omaha University, 1898; died April 27, aged 82, of heart disease.

Breese, Floyd Pinckney ⊕ Elmira, N. Y.; University of Buffalo School of Medicine, 1915; fellow of the American College of Surgeons; served during World War I; member of the honorary staff of the Arnot-Ogden Hospital; died in Coral Gables, Fla., March 16, aged 58, of acute myocardial infarction.

Brewer, Asbury Manton, Tunnel Hill, Ga.; Chattanooga (Tenn.) Medical College, 1901; died April 24, aged 79, of myocardial failure and hypertension.

Bunch, Morrell McKinsey, Muncie, Ind.; Bennett Medical College, Chicago, 1911; died April 25, aged 63.

Byram, James Waldamor, Willmar, Minn.; University of Minnesota Medical School, Minneapolis, 1931; affiliated with Willmar State Hospital; formerly affiliated with Hastings (Minn.) State Hospital; at one time a surgeon in the police department of St. Paul; died in Northwestern Hospital, Minneapolis, April 28, aged 49.

Charpentier, Albert Arthur, St. Paul; School of Medicine and Surgery of Montreal, Canada, 1899; died in Ancker Hospital April 12, aged 78, of coronary occlusion.

Creighton, Samuel Smith ⊕ Colonel, U. S. Army, retired, Cambridge, Md.; University of Buffalo School of Medicine, 1909; Army Medical School, 1911; entered the medical corps of the U. S. Army in 1911, and retired Dec. 31, 1939; fellow of the American College of Surgeons; died April 3, aged 71.

Daniels, Henry Manford, Venango, Pa.; Jefferson Medical College of Philadelphia, 1887; died recently, aged 92, of uremia and arteriosclerosis.

Dimm, Charles Hill ⊕ Mifflinburg, Pa.; University of Pennsylvania Department of Medicine, Philadelphia, 1889; during World War I chairman of the Union County Draft Board and served as its medical examiner; died in George F. Geisinger Memorial Hospital, Danville, April 24, aged 88, of arteriosclerotic heart disease and prostatic hypertrophy.

Feldaker, Lipa M. B., St. Louis; University of St. Vladimira Faculty of Medicine, Kiev, Russia, 1922; died April 20, aged 68.

Fish, Julia F., Erie, Pa.; Pulte Medical College, Homeopathic, Cincinnati, 1901; died in Cambridge Springs, April 26, aged 86, of chronic arteriosclerotic heart disease.

Grant, John Henry, Tyler, Texas (licensed in Texas by years of practice); formerly practiced in Corpus Christi, and in 1918 was president of the Nueces County Medical Society; died April 19, aged 88.

Harris, John Simpson, McMinnville, Tenn.; University of Nashville Medical Department, 1894; Vanderbilt University School of Medicine, Nashville, 1894; died in the Vanderbilt University Hospital, Nashville, April 25, aged 84, of undiagnosed disease of biliary system with jaundice.

Harrison, Hugh ⊕ Detroit; Michigan College of Medicine and Surgery, Detroit, 1896; served on the staffs of Harper and St. Joseph Mercy hospitals; died April 27, aged 80, of arteriosclerotic heart disease.

Hazen, Charles Morse, Bon Air, Va.; Medical College of Virginia, Richmond, 1897; emeritus professor of physiology at his alma mater; died April 22, aged 86, of cancer.

Heid, George Joseph ⊕ Bushkill, Pa.; Western Pennsylvania Medical College, Pittsburgh, 1906; died April 5, aged 67.

Hennemeyer, Rudolph J. ⊕ Chicago; Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin, 1923; affiliated with Woodlawn Hospital; died in the Albert Merritt Billings Hospital May 23, aged 56, of malignant hypertension.

Hottenstein, Daniel Edgar ⊕ Millersburg, Pa.; University of Pennsylvania Department of Medicine, Philadelphia, 1903; past president of the Dauphin County Medical Society; for many years county medical director; died in Harrisburg (Pa.) Hospital April 30, aged 76, of pulmonary embolism and bronchopneumonia.

Huckaby, A. H., Griffin, Ga.; University of Georgia School of Medicine, Augusta, 1905; died March 18, aged 69, of mesenteric thrombosis.

Hudnutt, Orrin Dean ⊕ Plainwell, Mich.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1910; member of the Radiological Society of North America; past president of the Allegan County Medical Society; served during World War I; died in William Crispe Hospital April 25, aged 71.

Hurley, William Cyril Rowe ⊕ Sunapee, N. H.; Tufts College Medical School, Boston, 1916; member of the Massachusetts Medical Society; fellow of the American College of Surgeons; for many years practiced in Quincy, Mass., where he was affiliated with Quincy Hospital; died in Sarasota, Fla., April 26, aged 63, of dissecting aortic aneurysm and coronary artery disease.

Hyde, Allan Pearson ⊕ Sharon, Pa.; Cleveland Homeopathic Medical College, 1901; specialist certified by the American Board of Radiology; member of the Radiological Society of North America and the American College of Radiology; served during World War I; affiliated with Sharon General Hospital; a director of the First National Bank in Sharpsville; died April 30, aged 73, of coronary thrombosis.

Jones, Henry ⊕ Coolidge, Ga. (licensed in Georgia in 1895); director of Moultrie Banking Company; affiliated with Vereen Memorial Hospital in Moultrie, where he died April 30, aged 82, of heart disease.

McNamee, John Ellis, Madison, S. D.; Barnes Medical College, St. Louis, 1902; died April 1, aged 85, of hypertensive heart disease.

Matzke, Samuel E., Warsaw, Ill.; Kansas City (Mo.) Homeopathic Medical College, 1897; affiliated with St. Joseph's and Graham hospitals in Keokuk, Iowa, and Memorial Hospital in Carthage, where he died April 2, aged 79, of carcinoma.

Moore, William Lee ⊕ Madisonville, Ky.; Barnes Medical College, St. Louis, 1896; physician for the Louisville and Nashville Railroad and Illinois Central Railroad; member of the city council, 1950-1951; for many years member of the county board of health; on the staff of the Hopkins County Hospital; died recently, aged 85.

Mueller, Walter Edmund, Green Bay, Wis.; University of Illinois College of Medicine, Chicago, 1914; past president of the Brown-Kewaunee-Door Counties Medical Society; served during World War I; president of the medical staff of Bellin Memorial Hospital for many years; affiliated with Veterans Ad- ministration; died April 23, aged 61, of cirrhosis of the liver and coronary occlusion.

Murawsky, William Joseph ⊕ Burlington, Wis.; Marquette University School of Medicine, Milwaukee, 1929; member of the American Academy of General Practice; health officer of Lyons; affiliated with Burlington Memorial Hospital, where he died April 23, aged 47, of cerebral hemorrhage.

Murphey, Eugene Edmund ⊕ Augusta, Ga.; University of Georgia School of Medicine, Augusta, 1898; past president of the Medical Association of Georgia; fellow of the American College of Physicians; professor emeritus of clinical medicine at his alma mater; served during World War I; for many years chairman of the board of health, and city health officer; an associate member of the American Ornithologists Union; died April 13, aged 76, of pneumonia.

Nichols, James Lutellus ⊕ Alligator, Miss.; Vanderbilt University School of Medicine, Nashville, Tenn., 1911; twice president of the Clarksdale and Six Counties Medical Society; served in France during World War I; died in the Kennedy General Hospital, Memphis, Tenn., April 28, aged 65, of cerebral hemorrhage.

Phillips, Floyd C. ⊕ Arthur, Ill.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1902; also a graduate in pharmacy; served as president of the Douglas County Medical Society; died April 13, aged 74, of cardiorenal vascular disease.

Pieroth, John Philip, Seattle; Rush Medical College, Chicago, 1925; served during World War I; on the staff of the Virginia Mason Hospital; died April 14, aged 57, of pontine hemorrhage.

Piper, Clark Culbertson ⊕ Highland Park, Mich.; Rush Medical College, Chicago, 1915; died in Highland Park General Hospital April 22, aged 65.

Portoghese, Nunzio, San Diego, Calif.; Regia Università degli Studi di Palermo. Facoltá di Medicina e Chirurgia, Italy, 1906; died April 11, aged 73, of coronary occlusion.

Price, Nathaniel Gordon ⊕ Newark, N. J.; Bellevue Hospital Medical College, New York, 1897; served on the staff of Beth Israel Hospital; died in Daytona Beach, Fla., April 22, aged 75, of coronary occlusion.

Quirk, Frank John, Chicago; Bennett College of Eclectic Medicine and Surgery, Chicago, 1895; served during World War I; died in Wesley Memorial Hospital April 15, aged 82, of uremia and carcinoma of the prostate.

Ragsdale, Harrison Curtis ⊕ Bedford, Ind.; Indiana University School of Medicine, Indianapolis, 1920; fellow of the American College of Surgeons; served during World War II; affiliated with Veterans Administration Hospital in Dwight, Ill.; formerly on the staff of Dunn Memorial Hospital; died in Indianapolis April 26, aged 56.

Reichbaum, Theodore ⊕ Easton, Pa.; Harvard Medical School, Boston, 1925; specialist certified by the American Board of Internal Medicine; from 1929 to 1940 coroner of Northampton County; served during World War I; on the staff of Easton Hospital; died April 16, aged 55, of acute coronary thrombosis.

Rymer, Thomas Edward, Ripley, W. Va.; Starling Medical College, Columbus, 1903; for many years a member and president of the county board of education; served during World War I; died April 6, aged 76, of heart disease.

Simmons, Colon L., Hazlehurst, Miss.; Memphis (Tenn.) Hospital Medical College, 1900; past president of the Tri-County Medical Society; served as medical adviser for Selective Service during World War II; for a number of terms member of the board of aldermen of the city of Hazlehurst; on the staff of Hardy Wilson Memorial Hospital; died April 10, aged 75, of Hodgkin's disease.

Simpson, James Asa, Dallas, Texas; Vanderbilt University School of Medicine, Nashville, Tenn., 1900; fellow of the International College of Surgeons; member of the Industrial Medical Association; actively identified in National Guard units for many years; served in the Philippine Insurrection, the Spanish-American War, the Mexican Border campaign, and in France during World War I; affiliated with the War Relocation Authority in Topaz, Utah, during World War II; died April 20, aged 78, of coronary disease.

Sinkler, Samuel, Chicago; Chicago College of Medicine and Surgery, 1908; also a dentist; died in the Albert Merritt Billings Hospital April 25, aged 74, of hydronephrosis and bronchopneumonia.

Smith, French Miles ⊕ Lyndon, Kan.; University Medical College of Kansas City, Mo., 1909; secretary of the Osage County Medical Society; died in Kansas City, Kan., April 19, aged 73, of carcinoma of the pancreas.

Starks, John Charles ⊕ Gower, Mo.; Missouri Medical College, St. Louis, 1897; an Associate Fellow of the American Medical Association; died March 21, aged 77, of coronary occlusion and arteriosclerosis.

Stein, Frederick ⊕ Lyndhurst, N. J.; L.R.C.P., Edinburgh, L.R.C.S., Edinburgh, and L.R.F.P. & S., Glasgow, 1941; associate dermatologist, St. Mary's Hospital in Passaic; died April 21, aged 40, of coronary occlusion.

Stine, Harvey Andrew ⊕ Harrisburg, Pa.; Baltimore Medical College, 1912; consultant in obstetrics, Harrisburg Polyclinic Hospital; died April 4, aged 73, of myocardial infarction and arteriosclerosis.

Van Doren, Raymond Fleming ⊕ East Lynn, Ill.; Northwestern University Medical School, Chicago, 1911; an Associate Fellow of the American Medical Association; died May 5, aged 65, of cardiorenal disease.

Van Gundy, Clyde Rogers ⊕ Detroit; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1912; served overseas during World War I; on the staff of the Lincoln Hospital; died May 5, aged 65, of coronary thrombosis.

Vella, Salvatore I., Chicago; Chicago College of Medicine and Surgery, 1915; died in St. Luke's Hospital April 22, aged 64, of cerebral thrombosis, hypertension, and diabetes mellitus.

Vincil, Frank Hendricks, Portland, Ore.; Barnes Medical College, St. Louis, 1903; served during World War I; died in Veterans Administration Hospital April 30, aged 72, of coronary sclerosis and diabetes mellitus.

Vinson, Charles Samuel ⊕ Columbus, Ohio; Ohio State University College of Medicine, Columbus, 1941; served overseas during World War II; resident in radiology at Ohio State University Hospital, where he died April 28, aged 35, of coronary infarction.

Vogt, Elkin ⊕ Lithonia, Ga.; University of Georgia School of Medicine, Augusta, 1939; member of the American Academy of General Practice; died May 5, aged 49, of coronary thrombosis.

Wagner, Louise Dorothy ⊕ Santa Barbara, Calif.; Rush Medical College, Chicago, 1920; for many years practiced in Pasadena, where she was affiliated with Huntington Memorial Hospital; died in Santa Barbara Cottage Hospital April 2, aged 56, of carcinoma of the sigmoid.

Wagner, Nicholas Bacon, Platteville, Wis.; Rush Medical College, Chicago, 1910; died May 19, aged 73.

Wentz, Frank Riegel ⊕ Allentown, Pa.; Jefferson Medical College of Philadelphia, 1913; affiliated with Sacred Heart Hospital, where he died April 25, aged 60, of myocardial infarction.

Whelan, Joseph David ⊕ Manchester, N. H.; Georgetown University School of Medicine, Washington, D. C., 1934; served during World War II; chief of staff, Sacred Heart Hospital; died in the Veterans Administration Hospital, Boston, May 6, aged 44, of leukemia.

Whitaker, Leslie Stow ⊕ San Jose, Calif.; University of Louisville (Ky.) School of Medicine, 1926; affiliated with Santa Clara County Hospital and O'Connor Hospital; died May 10, aged 52, of coronary occlusion.

Williams, Dell E., Laguna Beach, Calif.; State University of Iowa College of Medicine, Iowa City, 1897; died April 29, aged 80, of cardiac decompensation.

Willis, David Arthur ⊕ Chicago; University of Illinois College of Medicine, Chicago, 1923; associate professor of surgery, Chicago Medical School; specialist certified by the American Board of Surgery; fellow of the American College of Surgeons; affiliated with Mount Sinai Hospital; died in the Garfield Park Community Hospital May 19, aged 52, of myocardial infarction.

Ziegler, Samuel Edgar, Piqua, Ohio; Starling-Ohio Medical College, Columbus, 1910; died March 8, aged 78.

GOVERNMENT SERVICES

ARMY

Promotions in the Surgeon General's Office.—Stars were pinned on two officers of the Medical Service in a ceremony July 7 in the Surgeon General's Office. Major Gen. Silas B. Hays, Deputy Surgeon General, was awarded his second star and Col. Rawley E. Chambers, newly appointed Chief of Professional Divisions in the Surgeon General's Office, became a brigadier general.

General Hays became Deputy Surgeon General last year after serving as surgeon of the Japan Logistical Command since early in the Korean conflict. In this assignment he was responsible for management of Army hospitals in Japan, evacuation of casualties from Korea, and furnishing of medical supplies and equipment to both Japan and Korea. During World War II General Hays served as chief of medical supply in the European Theater of Operations. He was awarded the Legion of Merit with Oak Leaf Cluster, the Croix de Guerre with Palm, and Order of Public Health from France for his work in that country.

In addition to being coordinator of seven professional divisions in the Surgeon General's Office, General Chambers will serve as chief of the psychiatry and neurology consultants division. He has served as chief of the neuropsychiatric service at Brooke Army Hospital, Fort Sam Houston, Texas, and director of the department of neuropsychiatry at the medical field service school there. Under his direction the child guidance clinic was created at the hospital. During World War II he was commanding officer of the 12th Evacuation Hospital, 805th Hospital Center, 77th Station Hospital, 7th General Hospital, and 7th Hospital Group in the European Theater. Among his awards are the Legion of Merit, World War II Victory Medal, and Commendation Ribbon.

NAVY

Physicians Needed for Marine Reserve Unit.—The First Engineer Battalion, Marine Corps Reserve, at Fort McHenry, Baltimore 30, offers an exceptional opportunity to physicians of e Navy Reserve. This unit is authorized to have two reserve ysicians. Membership in the unit will afford physicians an opportunity to earn retirement and promotion credit points and will not effect their status in regards to active duty. However, for physicians who do not desire to attend the two week summer training camp at Camp Lejeune, N. C., but who desire to affiliate with this unit, the status of appropriate duty is offered. In an appropriate duty status a physician receives the same benefits, but is not considered a member of the unit and will not attend summer camp. Navy Reserve physicians interested should telephone Lieut. Col. E. P. Moses Jr., Marine Corps, at PLaza 4132 or see him personally at Fort McHenry in Building 1.

Reserve Officers Receive Retirement Credit for Attending Convention.—The chief of naval personnel announces that Reserve Medical Department Officers will receive retirement point credits for attending the annual convention of the Association of Military Surgeons, meeting in Washington, D. C., Nov. 17-19, 1952. Reserve officers of the Medical, Dental, Medical Service, Nurse, and Hospital Corps, not on the inactive status list (Public Law 810, 80th Congress), will receive one retirement credit point for each of the three sessions attended, provided that the duration of the meeting is at least two hours. An officer designated by the chief of the Bureau of Medicine and Surgery will attend the convention and record the names of those attending, the sessions attended and the duration of each session.

Personal.—Rear Adm. Lamont Pugh, Surgeon General of the Navy, has been notified of his recent election to active fellowship in the American Surgical Association.

PUBLIC HEALTH SERVICE

Regular Corps Examination for Medical Officers.—On Oct. 7, 8, and 9, 1952, the U. S. Public Health Service will hold competitive examinations for physicians interested in appointment in the Regular Corps. Completed applications, accompanied by a birth certificate and transcripts, must be received in the Washington office no later than Aug. 26. Appointments are for career service and will be made in the grades of Assistant and Senior Assistant Surgeon, with salary and allowances totaling $6,017 and $6,918, respectively. Provisions are made for periodic promotion, liberal retirement benefits, sick and annual leave, and other benefits. Applicants must be citizens of the United States and graduates of a recognized school of medicine. Interns are eligible for admission to the examination. Additional information should be requested immediately from the Chief, Division of Commissioned Officers, Public Health Service, Federal Security Agency, Washington 25, D. C.

Survey of Equipment and Supplies for Hospitals.—Half of the nation's hospitals have completed and returned their questionnaires in the nation-wide survey of the needs of civilian hospitals for vital equipment and supplies, it was announced by the Surgeon General. The survey is being conducted by the American Hospital Association and the Public Health Service to provide information on the supply and equipment needs of hospitals as a basis for determining what production is required to meet these needs. With this information, the equipment and supply requirements of the nation's medical care facilities can be planned more accurately as military requirements rise. Wesley E. Gilbertson, chief of the Public Health Service's Division of Civilian Health Requirements, urges that hospitals that have not yet returned the questionnaire do so immediately.

It was emphasized that reliability of the survey will be helped if hospital administrators will (*a*) verify that the items reported are exactly the same as those requested on the questionnaire form, (*b*) make sure that each box in the heading on the form contains an entry, (*c*) convert information respecting quantities to the units of measure requested, and (*d*) carefully review the completed questionnaire before mailing, to eliminate typographical and other inadvertent errors.

Division of Occupational Health.—New staff appointments have been announced by the Division of Occupational Health, Public Health Service, coincident with the reorganization of the division that went into effect on July 1. The environmental investigations branch and the clinical investigations branch have been merged into a single investigations branch at Cincinnati, which will be headed by Dr. W. Clark Cooper. Dr. Cooper was formerly Deputy Chief of Medical Services in the Public Health Service Hospital, San Francisco. Dr. Dale C. Cameron, who had previously served as chief of the cooperative health services branch, is now in charge of the new program developments branch of the division. Mr. Henry Doyle, who was formerly in charge of the division's Salt Lake City field station, will direct the activities of the state aid branch. Both of these branches are located in the Washington office. Dr. Lewis J. Cralley has been assigned to direct the activities of the Salt Lake City field station. Prior to this new assignment, Dr. Cralley had served as chief of the environmental investigations branch in Cincinnati.

Field Work for Foreign Health Workers.—The Division of International Health of the Public Health Service recently assisted the Rockefeller Foundation and the World Health Organization in orienting into the field of public health two groups of foreign health workers who just graduated from various schools of public health in the United States. The students, who represented 18 different countries, were given field work in Mississippi, Georgia, and Tennessee. Following this they were placed in county health departments in five states for further training during the summer.

FOREIGN LETTERS

ARGENTINA

Congress of University Teachers.—From May 14 to 17 the first Congress of University Teachers was held in Buenos Aires. In Argentina there are only governmental universities, private ones not being allowed. In one of the initial speeches, President Perón was mentioned as the "archetype of the University professor." At the final meeting, the congress declared that the mission of Argentine universities is to promote and diffuse a national culture related to the Christian occidental civilization and in accordance with the national constitution. The congress requested that the rank of full professor be considered at the same economical rank of a judge and that the function of professor be incompatible with any other official position. The congress established 169 technical committees for the study of university problems. The congress asked that May 15 be declared the "day of the University teacher" and that the Confederation of University Teachers be called for an international university congress under the economic support of the government. President Perón delivered a speech at the final meeting of the congress.

Prizes Awarded.—The prize of the Severo Vaccaro Foundation (1951-1952) was awarded to Dr. Oscar Orias, director of the Mercedes and Martin Ferreyra Institute for Medical Research, a private scientific nonprofit laboratory in Cordoba, supported by public gifts and in great part by resources granted by many Rotary Clubs of Argentina. Dr. Orias was a pupil of Professors C. Wiggers (Western Reserve), W. B. Cannon (Harvard), and B. A. Houssay (Buenos Aires), and was visiting professor of physiology in Long Island College of Medicine from 1948 to 1949. His principal researches have been on heart dynamics in coronary ligation, experimental and clinical branch blocks, gallop rhythm, and hypertension. He wrote, with E. Braun-Menendez, a book on heart sounds, which has been translated into English. He has developed modern scientific methods for all the cardiologists of Argentina. His researches on hypophysis are very important, including studies of action on heart and blood pressure and regulation of sexual function in batrachians. With his pupil Ines de Allende he recently wrote a book "Cytology of Human Vagina," translated by Dr. George Corner. Dr. Orias has been a hard-worker, a brilliant and stimulating teacher, and a man of the highest integrity and energy, who has devoted full time to research and teaching.

The annual prize of the Circulo Medico de Rosario was awarded to Drs. J. J. Staffieri and L. J. Cardonnet for clinical work on NPH 50 (modified protamine) insulin. The Argentine Medical Association awarded the Guillermo Rawson prize to Dr. J. C. Loza Colomer for his paper on "Organization of General Hospitals" and the Pedro O. Bolo prize to Drs. A. Lanari, M. Molins, and O. C. Croxatto for their experimental work on pulmonary graft. The prizes for the best medical thesis in 1951 were awarded to Drs. R. R. Rodriguez, M. A. Pico, and Gil Marino by the School of Medicine of the University of Buenos Aires.

Finlay Commemoration.—The Ministry of Public Health has organized many ceremonies in commemoration of the historical discovery by Carlos Finlay from Havana, Cuba, of the mosquito as a transmitter of yellow fever. In a ceremony attended by Pres. Juan Perón, held on May 20, a bust of Finlay was unveiled in the Policlinico Central. Moreover, one street in Buenos Aires has been named Carlos Finlay.

ISRAEL

Superinfection of Leishmania Tropica in Leishmaniasis.—Successful experimental superinfection of Leishmania tropica in patients with relapsing cutaneous leishmaniasis has been performed by Dostrovsky, Sagher, and Zuckermann of the dermatological department of the Hadassah University Hospital in Jerusalem. L. tropica in a susceptible human subject ordinarily produces a lesion that heals totally and spontaneously, leaving no residual parasites. The cured host generally remains immune for life and is refractory to natural reinfection. A small percentage of patients, however, are unable to eradicate the parasite, and their initial lesions continue to grow for years owing to successive relapses, with active peripheral foci surrounding a central scar. This lesion is known as leishmaniasis recidiva.

The investigators attempted to determine whether such patients with relapsing cutaneous leishmaniasis were susceptible to experimental superinfection with cultures of a strain of L. tropica capable of inducing typical primary nodular cutaneous lesions in subjects not previously exposed to leishmania organisms. Six patients with leishmaniasis recidiva, therefore, received intradermal inoculations consisting of 1 to 10 million washed culture flagellates of L. tropica over the left and right deltoid regions, respectively. Aspirated juice from the lesions developing at the inoculation sites yielded positive cultures in five of the six patients 1 to 15 weeks after inoculation, proving the subjects susceptible to superinfection.

Superinfection lesions obtained in these patients closely resembled relapse lesions and not initial (nodosa) lesions, as shown by the following lines of evidence. 1. Superinfection and relapse lesions consisted of yellowish-brown infiltrates with peripheral "apple jelly" nodules of granulation tissue revealed by diascopic pressure, as contrasted with typical nodosa lesions, which are reddish-blue and have no apple jelly nodules. 2. Superinfection lesions, like relapse lesions, were composed of tuberculoid tissue from their inception until cicatrization, as contrasted with the histocytic structure of the typical nodosa lesion. 3. As in relapse lesions, very few parasites were found at the superinfection sites. Microscopic examination was regularly negative, and not all tubes seeded with tissue juice yielded positive cultures. In contrast, parasites are generally numerous enough in the primary nodosa type lesion to be demonstrated microscopically, and all tubes seeded from such lesions are regularly positive. This phenomenon consisting in the development of a lesion resembling the relapse type rather than the primary type at the site of superinfection in patients suffering from relapsing cutaneous leishmaniasis has been designed as an "isophasic reaction."

Diagnosis of Leprosy.—The long incubation period of leprosy is the greatest obstacle in the exact diagnosis of this disease. Through the experimental studies of Sagher, Kocsard, and Liban, it has now become possible to answer the question of whether there is an acute infection. The first observation brought the result that histological examinations following tuberculin, leishmanin, milk, and peptone injected in the skin of leprosy patients showed the development of a granulomatous reaction indistinguishable from lepromatous leprosy. Furthermore, inoculations of BCG vaccine in leprous patients produced clinically typical lesions. The histological examinations from these lesions, however, revealed a granulomatous tissue reaction indistinguishable from true lepromas. It seems, therefore, that lepromatous leprosy produces only its own histopathological response notwithstanding the cause of the lesion produced. These facts may be of importance for criteria of cure from this disease, since the response was still present in patients after disappearance of

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The items in these letters are contributed by regular correspondents in the various foreign countries.

clinical signs and bacilli. Further, early cases may be detected even in the incubation period by performance of those tests and histological findings of lepromatous granulation tissue. So far, no other known disease reacts in this way.

Antimalaria Work.—According to Dr. J. Saliternik, the annihilation of Anopheles mosquitoes is found to be the most effective method of controlling malaria in this country. The most important malaria vectors today are the Anopheles sacharovi and Anopheles sergentii. Till 1945 Anopheles sacharovi, a domestic mosquito, was considered the main malaria vector. Since then it has decreased in number as well as in importance, as a result of the use of D.D.T. sprayed systematically in all malarious districts. The most dangerous mosquito in the country today is the Anopheles sergentii. Being a half domestic mosquito it cannot be sufficiently affected by residual sprayings.

A survey of all the water courses, potential Anopheles mosquito breeding places, in this country showed: 17,878 wells, 558 natural water courses of 1,236 km., 7,500 dunams of swamps (not including the Hule), and 23,467 dunams of fish ponds. In 1950, 28,000 working days were invested for chemical and technical treatment.

The fight against the adult Anopheles mosquito is carried on by residual D.D.T. spraying (5% D.D.T. solution in gasoline). In 1950, 412 tons of D.D.T. solution were used in spraying 344 settlements in malarious districts, comprising 105,714 rooms and a population of 152,690. Antimalaria work is carried on in accordance with climatic changes, population movements, the biology of the various Anopheles species, and periodicity of the malaria seasons.

As a result of the systematic antimalaria work, there has been not only a marked reduction in the number of new malaria cases (in 1950 only 842 cases were registered), but the whole epidemiological picture was completely changed. Formerly, there were two peaks in the malaria curve, one in spring in the coastal regions in particular, caused by Anopheles sacharovi, and the second in autumn in the interior of the country, where the main vectors are Anopheles sergentii and Anopheles superpictus. Now with the reduction of Anopheles sacharovi the first peak has nearly completely disappeared, and in the second there has been a considerable decrease, too.

New Department of Anatomy.—The Hebrew University-Hadassah Medical School formally inaugurated its new department of anatomy in the presence of a large number of teachers and students. The dean of the school, Professor Dostrovsky, explained that exceptional prominence was being given to the in- auguration of the courses in anatomy not only because it is a basic subject in the study of medicine but also because its in- clusion in the curriculum is a vital matter for the school itself. The many difficulties inherent in giving anatomy courses in Israel have been surmounted owing to the help extended by the ministries of health and justice and by the Anatomy Law. The opening lecture was given by Dr. M. Izkovitz, lecturer in anatomy, who described the history and development of that science through the ages. Just three years after the opening of the Hebrew University-Hadassah Medical Faculty in Jerusalem, the first class of 62 students received their Doctor of Medicine degrees in May, 1952.

New Tuberculosis Center Near Jerusalem.—The formal opening of the Eytanim tuberculosis center at Deir Amr in the Jerusalem Corridor took place in May, 1952. The 225 bed hospital has a children's section, which already cares for 100 patients. It is named in the memory of the late King Christian X of Denmark. Most of the funds necessary to equip the section were contributed by the Danish government and Danish citizens. The Danish minister to Israel, Mr. Hans P. Hoffmeyer, and representatives of the Danish Jewry attended the opening ceremony.

During the inspection of the hospital, Dr. Y. Chasis, head of the health ministry's tuberculosis department, disclosed that about 4,000 persons suffered from tuberculosis in Israel, al- though, as in every country, no exact figure is available since not all ill persons report to the authorities. Israel has at present 108 beds per 100,000 population, but it plans to raise the number of beds to 130. This figure can be considered very favorable when compared with most other countries in the world.

The Eytanim center will be expanded to hold 300 beds. Using an idea developed successfully in Switzerland, a number of prefabricated huts have been set up for former adult patients who wish to work for the hospital after their discharge as patients. By this method it is hoped that the shortage in personnel will be overcome. At present, the personnel numbers 0.5 per bed, while the international standard is 0.7.

NORWAY

Laboratory Control of BCG Vaccine.—Last May the University of Bergen conferred an M.D. degree on Dr. E. F. Krohn for his work there on the laboratory control of BCG vaccine. His work is published as "Supplement 30" to *Acta tuberculosea Scandinavica.* The cost of his investigations, which were prompted by the director of the laboratory, Dr. J. Böe, was borne by the Norwegian National Association Against Tuberculosis. The effects of the vaccines on human beings examined by Krohn were controlled by the tuberculosis department of the Oslo Public Health Service and by the state photofluorographic service. The first BCG culture was brought to Bergen by Birkhaug in 1937 from the Pasteur Institute in Paris, and both the strains employed by Dr. Krohn were derived from a culture obtained in 1945 from the same institute. The Bergen laboratory issues two types of vaccine, one concentrated and containing 20 mg. BCG moist weight per ml., and one diluted to contain only 1 mg. BCG per ml. Krohn's investigations were concentrated on the latter, which is intended for intracutaneous injection.

Since the success of the BCG campaign throughout the world must ultimately hinge on the reliability of the laboratory control of the vaccine, Krohn's work may be said to be of vital importance to millions. Yet it is so highly technical that most of his book will be understood by only a few of his fellow workers. But even for persons to whom mathematical formulas and the finesse of laboratory technique mean little, there is much in this book to stimulate and interest them. Dr. Krohn has come to the conclusion that no definite correlation can be established between the number of viable bacillary particles and the effects of the vaccine on human beings. "Such effects would seem, accordingly, to be determined in the main by qualitative properties, the demonstration of which is not possible by ordinary laboratory methods, but will require biological tests." A series of investigations of the immunizing effects of Norwegian BCG vaccine on mice is being planned as part of the present study. They are still at a preparatory stage and will therefore be published separately. In the present work a summary after each chapter and a comprehensive summary at the end of the book are helpful in giving a bird's-eye view of Dr. Krohn's main conclusions. His bibliography contains 172 references.

Fewer Days in Hospital.—There is a general impression, not always substantiated by reliable statistical evidence, that in countries with a high cultural level patients spend fewer days in the hospital than they did a few years ago. Herr E. W. Østberg, who is attached to a statistical bureau run by the municipal authorities of Oslo, has recently provided such evidence on the basis of 11,548 patients treated in certain hospitals in Oslo during the years 1928, 1937, and 1947. During this 20 year period, the average duration of treatment in a hospital was reduced from 28.9 days in 1928 to 20.6 in 1937 to 19.9 in 1947. It will thus be seen that this reduction was much greater in the first than in the second decade under review. Since it can be assumed that the duration of treatment in the hospital rises with the average age of the patients and since the average age of the patients has risen in this 20 year period, this reduction in the duration of treatment in hospitals is, indeed, a remarkable achievement. The relative upward shift of old persons in a given community was estimated in 1948 to be such that in 1920 only

3.3% of the total population of Oslo was 70 years old or older, whereas the corresponding figure in 1948 was 5.8%. In 1928, approximately one-third of the male patients studied by Østberg were under the age of 30, whereas in 1937 they represented only 20.1% of all the male patients, and in 1947 only 13.9%. A classification of the patients according to the diseases for which they were treated in the three years under review showed that there was a relative rise in the number of cases of heart disease. This rise may well reflect the greater average age of the patients. Between 1937 and 1947 there was a marked rise in the relative frequency of all forms of pneumonia.

Education in Mental Hygiene.—The Norwegian Mental Hygiene Association, whose chairman is Dr. Gordon Johnsen, last May launched a mental hygiene education campaign with two main interests. One of them is the child of preschool age and its home. The other is the employer and the employee and the parts they play in industry. Speaking on the first subject, Dr. Johnsen pointed out that the only mental hygiene polyclinic for children in Oslo has so many requests for service and so long a waiting list that up to three months have to pass before some cases can be dealt with. Yet this polyclinic is threatened by closure for want of the necessary funds. Dr. Johnsen said that the Mental Hygiene Association is planning the creation of several more polyclinics along similar lines. With the help of such powerful voluntary organizations as the National Association Against Tuberculosis and for Health, as well as the Red Cross, he hopes to organize mental hygiene teams composed of psychiatrists, psychologists, and social workers. He assured his audience that psychic disturbances were just as common in children of the preschool age as among adults.

Speaking on the same occasion, Dr. Karen Margrethe Simonsen, who was recently imported from Denmark to take charge of the new psychiatric department for children at the Rikshospital in Oslo, compared the mentalities of Norwegian and Danish children. She has come to the conclusion that in Norway the most common cause of faulty mental development in the child is the strait-laced and inhibited outlook of the parents. Inhibited by moral scruples in their emotional lives, they are apt to impose an excess of moral responsibility on their children who develop compulsion neuroses in consequence. She has found cases of this type to be much commoner in Norway than in Denmark whose population has a more open and less inhibited emotional outlook. At the Rikshospital about half of all her patients are children under school age. She is convinced that treatment given early enough, during the first years of life, has an influence on the subsequent development of the personalities of such children.

Opening of Audiological Institute in Oslo.—On May 21 the new Audiological Institute within the framework of the Rikshospital in Oslo was opened formally by the university's rector, Herr F. Castberg. It is the only institute of its kind in Norway, and it owes its creation to the generosity of a whale oil magnate, Herr Anders Jahre, whose donation of 150,000 k. has been supplemented by the sum of 75,000 k. for working expenses during the first five years. The institute is linked up with the ear, nose, and throat department of the Rikshospital, in the new surgical block. Professor Opheim, who is in charge of this department, pointed out at the opening ceremony that there are some 60,000 persons in Norway who are completely deaf, and that about 10% of the whole population suffer from defective hearing. Yet in spite of the enormous social problems raised by this form of invalidism, it was only in recent years that attempts had been made to solve them on a medical and scientific basis. The nucleus of the institute consists of a laboratory and three soundproof rooms.

The publicity given to the problems of noise and deafness in both the lay press and the medical press in recent years reflects the active interest now being taken in the prevention of defective hearing. Dr. S. Quist-Hanssen has recently published studies on this subject and has shown how much the success of ear plugs depends on the care with which they are fitted. Audiometric control of such fitting requires only a few minutes and, when supplemented by instruction in the use of such plugs, should do much to assure their success. Glass fiber wool has gained favor since an account of its use was given in May, 1951, in Helsingfors at the Scandinavian Congress for Industrial Medicine. Made from melted glass, the diameter of its fibers is only about one-tenth of that of cotton wool fibers. Dr. Quist-Hanssen has found no great difference in the effects of glass fiber wool according to whether it was lightly or tightly compressed, but other observers disagree with him on this point. A drawback to this preparation is, in his opinion, its tendency to deposit some dust in the ear passages. But they do not become irritated even when the glass fiber wool is used daily for several months. Its cheapness is also a point in its favor. It is with this problem and many others that the institute in Oslo will now be working.

TURKEY

Schistosoma Haematobium Investigation.—The Ministry of Health and Social Assistance appointed Prof. Zühdi Berke and microbiologist Dr. Tahsin Berkin of the Refik Saydam Central Institute of Hygiene, Ankara, to report on Schistosoma haematobium in the southern provinces that border Syria and Iraq. In 1939 an increase in schistosomiasis in Syria was noticed. French regiments from the Senegal, who had been stationed there from 1936 to 1941, and 60 to 70% of the Syrian population along the Syrian-Turkish border became infected. As a result of an investigation made by specialists from the Syrian Ministry of Health and in cooperation with experts from the American Medical School in Beirut, tentative control was instituted in 1948. Investigation by Prof. Zühdi Berke and Dr. Tahsin Berkin revealed the presence of two Schistosoma haematobium centers near the Turkish border. One center was in the Kuburelbeyd village, population about 1,000, 15 miles south of the Turkish border, and also in the Tel Hasnak village, 3 miles from the Turkish border. The second center was in the Tel Abyad village, 125 miles east of Halep (Aleppo) and south of the Turkish district of Akça Kale, population about 76,000, and in almost 1,000 other villages. Three streams traverse this territory. Along the Djerrahi River, from 8 to 34% of the population were affected, along the Sublah River 15 to 24%, and along the Katraniye River 10 to 40%. All patients were given treatment. Under the direction of an expert from Egypt, a schistosome prevention and control unit was equipped with a mobile hospital and laboratory to take up its activities in this territory. Investigations along the Turkish-Iraqian border confirmed that urinary schistosomiasis is endemic in Iraq. During the hot summer season, June to October, the population bathes in the streams and rivers spreading infection. Ponds and swamps, resulting from inundations, water plants along slowly moving rivers, and rice fields favor the propagation of infected snails.

Hospitals, dispensaries, and health propaganda in Iraq have resulted in a reduction of schistosomiasis and prevented more serious outbreaks. The economic aspects of the damage caused by snails has been investigated by Mr. John Watson, an expert who recommended more radical measures, and the World Health Organization has decided on an effective program for the control of the schistosomiasis scourge.

According to the report the Ministry of Health received from Prof. Berke and Dr. Berkin, there are no persons affected with schistosomiasis in the Turkish border districts of Akça Kale, Idil, and Nusaybin, or in the surrounding areas. Random urinalyses on hundreds of adults and children had negative results. Investigation at the Central Institute of Hygiene, Ankara, of mollusks from the streams and rivers traversing the three territories revealed that they were not Schistosoma haematobium hosts, Bulinus contortus, nor, though they were found in the Suruc Stream, were they infected. This corroborated the findings of Dr. Fehmi Derbent, who, in cooperation with local municipal health officers, had previously made an investigation for the Ministry of Health.

CORRESPONDENCE

TOUR TO WORLD MEDICAL ASSOCIATION MEETING IN ATHENS

To the Editor:—To facilitate travel to the Sixth General Assembly of The World Medical Association, in October, 1952, World Travel Plan has arranged a guided tour by airplane charted through an internationally known scheduled airline, namely, Sabena Airlines.

The trip will include tours of Rome, Geneva, and Paris, as well as a stay in Athens during the period of the General Assembly. The total cost is $789.00 per person, which is about $250.00 per person less than the cost of a round trip by air alone. The $789.00, however, does not include expenses in Athens, which will be in addition to the above amount. The proposition would seem to be advantageous to those who are contemplating the trip and who would like to make the tours of the other cities at the same time.

The itinerary for this tour is as follows.

DATE	PLACE	ITINERARY
Wed., Oct. 8	At New York	Accommodation provided at Hotel Roosevelt, Madison Ave. at 45th St., New York City.
Thu., Oct. 9	Leave New York	After breakfast at hotel, final check of documents and discussion. Transfer by private motor direct to Idlewild International Airport. Check in at airport and leave early afternoon by first-class D.C.-6 plane of Sabena Belgian Airlines. All meals with beverages included.
		GREECE
Fri., Oct. 10	Arrive Athens	Arrive afternoon. Transfer by private motor to hotel.
Sat., Oct. 11 to Fri., Oct. 17	At Athens	Attending the Sixth General Assembly of the World Medical Association.
Sat., Oct. 18	Leave Athens	After breakfast, transfer by motor direct to airport. Leave Athens by plane.
		ITALY
	Arrive Rome	Arrive about noon. Transfer by motor to hotel. Afternoon: A general drive and survey of the city, returning to the hotel for dinner.
[illegible], Oct. 19	At Rome	This morning is available in Rome for individual action. Afternoon: Special drive to Tivoli, proceeding along the Via Tiburtina to Acqua Albule (sulfur baths), Ponte Lucano, Tomb of Plautius Lucanus to Hadrian's Villa. Continue to the Villa d'Este (visit the grounds with many fountains, artificial grottos, and pavilions); drive to the Temples of Vesta, Sybil, and Hercules and to the waterfalls of the Anio. Return to Rome for dinner.
Mon., Oct. 20	At Rome	This morning is free in Rome. Afternoon: A general drive and survey of the city.
Tue., Oct. 21	At Rome	This day available for individual action.
Wed., Oct. 22	Leave Rome	After breakfast, transfer by motor direct to airport. Leave Rome by plane.
		SWITZERLAND
	Arrive Geneva	Arrive early afternoon. Transfer by motor to hotel. Balance of day free.
Thu., Oct. 23	At Geneva	Survey of the city, then by lake steamer on Lake Geneva (Swiss and French side) including Montreux (Chillon Castle) and Lausanne.
Fri., Oct. 24	Leave Geneva	After breakfast, transfer by motor direct to airport. Leave Geneva by plane.
		FRANCE
	Arrive Paris	Arrive about noon. Transfer by motor to hotel. Afternoon by motor for a special tour of this fabulous city.
Sat., Oct. 25	At Paris	After breakfast, by motor for additional city touring. Continue to Versailles for luncheon and visit the famous palace. Back to the hotel for dinner.
Sun., Oct. 26	At Paris	This day available for individual action.
Mon., Oct. 27	At Paris	Full day free for shopping (expert guide provided). Fashion show, additional local visits, or special interest program. Special luncheon provided at famous Parisian restaurant.
Tue., Oct. 28 and Wed., Oct. 29	At Paris	Days free. Tickets provided for Folies Bergère or similar show, the opera, etc.
Thu., Oct. 30	Leave Paris	Transfer by motor direct to airport for overnight flight home. All meals provided.
		UNITED STATES
Fri., Oct. 31	Arrive New York	Arrive International Airport. Motor transportation provided into New York City.

Louis H. Bauer, M.D.
Secretary-Treasurer
World Medical Association
2 E. 103rd St., New York 29.

"TELEVISION NECK"

To the Editor:—For many years, I have had a special interest in musculoskeletal disorders. As part of every physical examination, I have measured joint mobilities according to the methods described in my monograph "The Common Form of Joint Dysfunction" (Brattleboro, Vt., E. L. Hildreth & Co., 1949).

Recently I have observed a clinical syndrome in persons who, in watching television programs, maintain strained postures of the head and neck, often for prolonged periods. The manifestations of this syndrome, which for want of a better name can be called "television neck," include measurable increased limitation in ranges of neck movement and pain or discomfort in the posterior nuchal region (with or without radiation of pain or discomfort into the shoulders, upper back, or occiput). In many of these patients, the faulty sitting and erect posture that is maintained when they are not viewing television is a result of the tendency to flex the neck on the trunk and extend the head on the neck to maintain the face roughly parallel to the frontal plane of the body and to the television screen. There is also some concomitant exaggeration of the upper dorsal curvature, suggesting a mild or moderate upper dorsal kyphosis. When television neck occurs in both husband and wife and each has a favorite chair and position for viewing television, the limitation in movement and the discomfort occur mainly on one side in the husband and on the other side in the wife.

When several persons view the same screen, there is also sustained lateral bending of the neck. The relatively fixed position of the head, neck, and upper trunk is associated with symptom-producing neck muscle hypertonia and increased static postural traumatization of the articular surfaces of the cervical and upper thoracic vertebrae.

The remedy—aside from not viewing television—is to raise the television set so that the screen can be comfortably viewed by each person without assumption of this awkward sustained posture. Sometimes a chair with a lower or higher seat permits less strained posture.

Since television viewing in the home and elsewhere is likely to increase, physicians should be alert to recognize the syndrome of television neck and to prescribe proper treatment. Television equipment and furniture designers should be encouraged to develop home equipment that will make it possible for the viewer to enjoy television programs without maintaining strained posture for prolonged periods.

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A SIMPLE EVALUATION OF FOODS

To the Editor:—A question frequently asked, mostly by laymen, pertains to the relative value of different foods: "Are carrots a better food than potatoes?" "Do beets have much food value?" "How do spinach and kale compare in food value?" Obviously, if one were completely or largely on a potato diet, carrots in the diet would have a far greater value than an equal amount of potatoes. Yet conceivably there are diets that could not be improved by the addition of carrots.

It is realized that such questions have something in common with the question "Which is the more important, the hen or the egg?" The completely evasive, though inescapable, answer to the hen and egg question is that without one there cannot be the other. It is possible, however, to give a better answer in a comparative evaluation of different foods. The first requisite in such comparisons is to have representative analytical figures as to the content of the critical dietary essentials in any one food. Counting the essential amino acids, there are about 50 dietary essentials. Not all are critical in the sense that there is any danger of our not receiving an adequate supply, regardless of what our diet may be.

The Nutrition Board of the National Research Council, in setting up the recommended daily dietary allowances, gave recognition to what are commonly regarded as the critical dietary essentials. The recommended amounts naturally vary with the person, being affected primarily by age and activity and, to a lesser extent, by size and sex. For the purpose of the comparative evaluation of foods in the accompanying table the values for a 154 lb. (70 kg.) sedentary man and a moderately active 123 lb. (56 kg.) woman are used, since they are alike for most of the nutrients, and, in cases in which they differ, the median value has been used. These values are as follows: calories 2,400; protein 65 gm. (median, between 70 for the man and 60 for the woman); vitamin A 5,000 I. U.; thiamine 1.2 mg.; riboflavin 1.65 mg. (median between 1.8 for the man and 1.5 for the woman); niacin 12 mg.; and ascorbic acid 72.5 mg. (median between 75 for the man and 70 for the woman). It may be added that the use of these median values instead of the exact figure

Ratio of Daily Requirement of Nutrients to Daily Requirement of Calories in Foods *

	Protein (65 Gm.)	Ca (1,000 Mg.)	Fe (12 Mg.)	Vitamin A 5,000 I. U.)	Vitamin B_1 (1.2 Mg.)	Vitamin B_2 (1.65 Mg.)	Niacin (12 Mg.)	Vitamin C (72.5 Mg.)	Average
Canned Vegetables and Fruits									
Asparagus, white	3.1	1.8	8.8	1.2	4.9	4.2	6.9	24.0	6.9
Asparagus, green	3.5	2.1	10.8	12.0	6.6	6.7	8.1	24.0	10.0
Beans, baked	1.7	1.1	4.4	0.2	0.6	0.5	0.9	0.7	1.3
Beans, green cut	2.1	4.2	12.3	7.3	2.9	2.7	3.3	4.7	4.9
Beans, lima green	2.2	0.9	7.1	1.1	1.0	0.9	1.6	3.4	2.3
Beets, cubed	1.0	1.0	1.1	0.2	0.5	1.0	0.9	4.4	1.3
Carrots, cubed	0.9	2.1	5.0	208.5	1.6	1.0	0.3	2.1	27.7
Corn, white cream style	0.9	0.1	1.3	0.1	0.7	0.8	2.4	2.3	1.1
Corn, yellow cream style	0.9	0.1	1.3	0.7	0.7	0.8	2.0	2.2	1.1
Corn, white whole grain	1.0	0.2	1.4	0.1	0.6	0.9	2.6	2.5	1.2
Corn, yellow whole grain	1.0	0.1	1.1	1.0	0.7	0.9	2.2	2.3	1.2
Kraut	1.8	4.1	64.8	1.2	3.2	2.8	1.0	27.4	13.3
Mushrooms	5.7	0.8	5.3	0.4	1.7	14.7	16.9	3.9	6.2
Peas, Alaska	2.1	0.8	5.6	3.7	3.1	1.3	2.8	5.3	3.1
Peas, sweet	2.3	0.9	5.7	4.1	4.2	1.6	3.8	6.1	3.6
Pimentos	1.4	0.6	10.9	46.2	2.1	3.7	3.3	157.0	28.1
Peppers, sweet	0.9	0.6	11.5	51.4	2.5	3.8	4.5	124.3	24.9
Spinach	3.5	9.9	20.4	128.0	1.7	7.0	3.1	20.4	24.3
Sweet potatoes	0.5	0.5	1.1	43.2	1.0	0.6	0.9	4.9	6.6
Tomatoes	1.7	0.7	4.5	21.0	4.8	1.8	6.3	24.8	8.2
Turnip greens	2.9	12.9	4.9	113.2	1.6	7.0	6.2	34.8	22.9
Apricots, syrup	0.2	0.2	1.6	12.1	0.4	0.3	0.8	1.6	2.2
Blackberries, syrup	0.3	0.5	6.2	1.0	0.3	0.3	0.5	2.4	1.5
Blueberries, syrup	0.5	0.6	22.5	0.4	0.6	0.5	0.8	9.8	4.5
Cherries, red sour	0.6	0.5	10.5	7.8	1.0	0.4	0.7	3.9	3.2
Grapefruit segments	0.3	0.4	0.8	0.1	0.9	0.4	0.6	10.7	1.8
Peaches, cling	0.2	0.1	0.7	2.7	0.2	0.4	1.5	1.7	0.9
Peaches, Freestone	0.2	0.1	0.7	1.9	0.2	0.3	1.3	1.3	0.8
Pears, syrup	0.2	0.2	1.3	0.1	0.3	0.4	0.5	0.8	0.5
Pineapple, sliced	0.2	0.5	1.4	0.2	1.4	0.3	0.3	1.6	0.7
Plums, purple	0.2	0.2	2.2	6.3	0.5	0.3	0.8	0.6	1.4
Grapefruit juice	0.4	0.4	1.8	0.2	1.5	0.6	0.8	24.6	3.8
Orange juice	0.6	0.5	2.0	1.6	3.0	0.6	1.0	25.1	4.3
Pineapple juice	0.3	0.6	1.0	0.4	1.8	0.4	0.6	5.1	1.3
Tomato juice	1.5	0.8	8.6	19.9	5.3	2.0	7.4	23.6	8.6
Average	1.3	1.5	7.3	20.0	1.8	2.1	2.8	17.0	...
Canned Fish									
Mackerel	3.6	2.7	1.9	0.3	0.3	1.5	7.9	0	2.3
Salmon, pink	5.3	3.0	0.8	0.2	0.3	1.6	10.5	0	2.7
Sardines in oil	2.6	3.1	2.5	0.4	0.2	0.7	4.0	0	1.7
Sardines in tomato sauce	3.1	4.3	3.9	...	0.1	1.7	4.8	0	2.2
Shrimp, dry pack	8.2	2.1	5.8	0.3	0.2	0.4	3.7	0	2.6
Shrimp, wet pack	8.2	2.0	6.5	0.4	0.2	0.7	4.0	0	2.8
Tuna	3.5	0.1	0.9	0.1	0.3	0.8	8.0	0	1.7
Average	4.9	2.5	3.2	0.3	0.2	1.1	6.1	...	...
Noncanned Staple Foods									
Bread, white enriched	1.2	0.5	1.5	0	1.9	0.8	1.5	0	0.9
Milk, whole	1.9	4.1	0.2	1.1	1.2	3.5	0.2	0.4	1.7
Beef, cooked, all methods	2.9	0.1	2.3	0	0.5	0.8	3.7	0	1.3
Egg	2.2	0.8	3.4	3.4	1.4	3.1	0.1	0	1.8
Average	2.1	1.4	1.9	1.1	1.3	2.1	1.4	0.1	...

* Daily requirement is given in parentheses under each nutrient.

for a man or for a woman has no significant effect on the figures in the accompanying table.

The literal meaning of the figures may be illustrated with canned white asparagus, since it stands at the head of the list. It is not intended to suggest that any sedentary man or moderately active woman is ever going to eat enough white canned asparagus in a single day to provide the entire recommended daily requirement of 2,400 calories. But, if one supposes that this amount were consumed in one day, and it alone, 3.1 times the recommended protein would be supplied, 1.8 times the recommended calcium, 8.8 times the iron, 1.2 times the vitamin A, 4.9 times the thiamine, 4.6 times the riboflavin, 6.9 times the niacin, and 24 times the ascorbic acid (vitamin C).

In a similar manner, the meaning of the average figures at the bottom of the list of canned foods becomes clear. If the daily recommended calories were obtained by taking an equal number of calories from each of the 35 canned foods listed (a vegetarian diet), those canned foods would supply 1.3 times the recommended protein, 1.5 times the calcium, 7.3 times the iron, 20 times the vitamin A, 1.8 times the thiamine, 2.1 times the riboflavin, 2.8 times the niacin, and 17 times the ascorbic acid.

There is good reason for evaluating foods on the basis of the amount of the protein and noncalory nutrients supplied in proportion to the calories. Of all the factors for which the Nutrition Board has made recommendations, only calories are directly related to the amount of food consumed. If we eat less food than will supply our normal requirement of calories, we will lose weight; if we eat more, we will be burdened with overweight, and obesity is a major problem in nutrition. But no suggestion has ever been made of our getting harmful amounts of any of the other nutrients from our foods; the concern has been entirely with our getting the recommended amounts, and it has been intimated that greater amounts are all to the good.

The average figures in the last column of the table serve as rough comparative value of individual foods in their ability to supply critical nutrients; for example, 0.5 for pears as against 10.7 for green asparagus leaves little doubt about the comparative value of the two. But the figure 27.7 for carrots is largely due to the very high value (208.5) for vitamin A. All the other values for carrots, with the exception of that for calcium, being *materially lower than for green asparagus.*

The values for the canned foods have been calculated from the data in "Canned Foods in Human Nutrition" (Washington, . C., National Canners Association, 1950). This represents the st comprehensive analytical survey of any class of food. undreds of samples from the entire country were collected, and the analyses were made in the nutrition departments of nine of our great universities. The values for the noncanned staple foods are from data in the U. S. Department of Agriculture Handbook no. 8, June, 1950.

Since the data in the table are based on chemical and microbiological technique they reveal nothing regarding availability. When there are data on availability, the figures in the table will assume far greater accuracy. It should also be remarked that nitrogen content is not a true measure of protein value; also, the high calcium content of spinach is known to be of little value nutritionally. In spite of these shortcomings some definite conclusions are evident. The vegetables as a group are outstanding in providing an abundance of all the critical nutrients in proportion to their calory value. They outrank the so-called staple foods and the fruits. As a class, fruits and vegetables supply vitamins A and C in great abundance. Since the ratio of vitamins A and C to calories is 20 and 17 for the canned vegetables and fruits, respectively, our vitamin A requirement would be met if they supplied but 5% of our calories and our vitamin C requirement would be met if they supplied 6% of our calories. While the data pertain more specifically to canned vegetables and fruits, they no doubt apply to other foods, irrespective of form.

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CONTRIBUTION BY INDUSTRY TO MEDICAL EDUCATION

To the Editor:—The efforts to persuade industry of its share in the responsibility for the financial support of medical schools is well known to us all and is an endeavor of the greatest importance. This problem is so immediate and so vital that it tends to overshadow a second problem, of which I am writing you today.

The rapidly rising costs to the student in medical school have placed an extraordinarily heavy financial burden on each candidate, while at the same time the demands of the current-day curriculum make the time available for outside earnings insignificant for the most part. During this same time, as you well know, the income from scholarships and endowments has diminished. Were it not for the fact that the future of the medical school itself is at stake, this current dilemma of its students would assume much greater prominence.

Recently one of the pharmaceutical companies has recognized this as a special problem and has seen fit to give to every approved medical school in the country $1,000 to be disbursed to students in need. This action by a pharmaceutical company seems to me to deserve recognition by the members of the American Medical Association, who are so interested in seeing problems solved through the efforts of private enterprise.

If the board of trustees of such companies as the Charles Pfizer Company, which made the above-mentioned donation, can be encouraged by appropriate recognition to increase and to generalize their donations to medical education they could provide a large part of the total needs of the medical schools. Although $70,000 is not large in terms of the total need, it does seem to indicate a recognition by a pharmaceutical company of a direct responsibility for the needs of medical education. It would be regrettable if this sense of responsibility were not encouraged.

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CHICAGO MEDICAL BOOK COMPANY

To the Editor:—We wish to inform your readers that the Chicago Medical Book Company has had nothing to do with nor did we know anything about the printing, publication, or distribution of a pamphlet, "The Hospital Superintendent: What Makes Him Tick?" This is reported to be a study in human behavior, but it is unsigned, and there appears to be no way of identifying the author or sponsor. Unfortunately, at the end of the document appears this statement: "Additional copies of this book may be obtained from the Chicago Medical Book Co., Jackson and Honore Streets, Chicago, Illinois."

The Chicago Medical Book Company has never been contacted by any person or persons requesting the use of our name with regard to this book or a book of this nature, nor have we ever given our permission to any person or persons allowing them the use of our name for the sale, distribution, or procurement of this book. We are completely ignorant as to what person or persons would use our company's name on the last page of this book as a source of supply. Until the post office called us about postage due on 120 of the booklets, we knew nothing whatever about it. We have had many requests, emanating from various sections of the United States, requesting additional copies of this booklet.

We are most desirous of correcting this unfortunate incident and take this opportunity to inform your readers of the above facts. In the event that they have any information regarding this publication, we would be more than happy to be advised of the same.

A. C. Bowers
Advertising Manager
Chicago Medical Book Company
Jackson and Honore Streets
Chicago 12.

MISCELLANY

MEDICAL LIBRARY OF A NON-UNIVERSITY GENERAL HOSPITAL

A Five-Year Progress Report on the Medical Library of the New Britain General Hospital

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and
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The New Britain General Hospital is a general, nonprofit association hospital of 267 beds. It is the only hospital of its type in an industrial community that, with its suburbs, has a population of approximately 100,000. The active, courtesy, and general practice staffs consist of 84 physicians, of whom 35 have been certified by various specialty boards. An approved residency training program is conducted in surgery, gynecology, obstetrics, internal medicine, and pathology.

The Hospital Medical Library was reorganized in 1946, pending completion of its present quarters, which it occupied in June, 1947. The area presently assigned to the library consists of a main reading room with tables and chairs accommodating about 15 persons, an adjacent stack room, and a nurses' reading room, located in the dormitory of the School of Nursing. An earlier report[1] outlined the developmental background of the library and discussed the guiding principles under which it operates. During the past five years there has been ample opportunity to test the original thinking on the organization and administration of the library against actual experience. Many ideas have been altered and revised in the light of this experience, and new thoughts have been incorporated in the program. The present five-year progress report has been prepared in the hope that it may be helpful to other non-university hospital groups interested in establishing a medical library in their institutions.

THE LIBRARY COMMITTEE

From its inception, the professional supervision and control of the library has been vested in a committee of the medical and nursing staffs. This committee, appointed by the board of directors of the hospital, is directly responsible to the board. The library is a recognized department of the hospital, and the librarian, as a hospital department head, is administratively responsible to the managing director.

The library committee[2] is composed of five members, two of whom, the director of nursing service and the school of nursing and the pathologist, are full-time hospital employees. The other three members, selected from the active medical staff, are each appointed for staggered three-year terms. Regular meetings of the committee are held each month, the librarian acting as secretary. Minutes of the meeting are recorded, and copies are distributed to each member of the committee, to the managing director, and to the librarian. Special meetings to consider urgent problems may be called by the chairman of the committee. The appointment to the library committee of the director of nursing service and the school of nursing is in accord with the originally stated policy of combining the library of the School of Nursing with the medical library.

Each member of the committee has recognized the great responsibilities of his office, and rarely does a member fail to attend the scheduled meetings. These are largely concerned with reviewing suggestions presented in writing by members of the medical staff for the purchase of textbooks or journals and acting on these recommendations. Such decisions are usually based on the general usefulness of the textbook or journal to a staff of varied interests and specialty training and always with the keen awareness of a relatively limited budget. In actual practice, the total annual budget is determined by the managing director, but the distribution and allocation of funds within the budget rest largely on the recommendations of the library committee. Other matters under discussion are such items as coverage during the librarian's vacations and other absences, the purchase of additional book shelves and large equipment, methods for increasing the usefulness of our facilities to the professional staff, and continuing thinking on ways and means of enhancing the attractiveness of the library quarters. The library committee has disapproved the use of the library reading room for small meetings or classes. The library committee believes that these activities would deprive members of our professional staff of the use of the library facilities during the meetings and would thus defeat a primary objective of the library, namely, round-the-clock accessibility.

FINANCES

Since a hospital library is a nonincome producing unit of the institution, its financial support must be derived from endowments, from contributions, or from general funds. Originally it was hoped that our library would be at least partially sponsored by specific bequests, but this hope has not been realized and we have had to depend on the other two sources to secure our operating funds. The members of the medical staff, at the inception of the program, in 1946, unanimously approved a self-imposed library tax of $15 each per *annum*, and without dereliction this amount has been subscribed each year. The treasurer of the staff transfers these library dues to the hospital, which ear-marks them for library use. In addition, small library fees paid by student nurses are allocated to the library fund. These monies comprise less than one-fourth of our total annual expenditures, the remainder being provided by the board of directors from other sources. The action of our directors in so generously supporting a hospital activity that is all too frequently by-passed or ignored is a tribute to their deep interest in developing and maintaining high medical standards.

New Britain General Hospital Library
Original Budget, Actual Expenditures from 1946 Through 1951 and Anticipated Budget for 1952

	Journals	Binding	Textbooks	Miscellaneous and Salaries	Total
Original Budget	$1,000.00	$1,000.00	$500.00	$2,500.00	$5,000.00
Actual Expenditures					
Nov., 1946-Sept., 1947	999.43	397.32	601.90	2,438.54	4,437.19
Oct., 1947-Sept., 1948	969.99	487.61	537.50	3,033.38	5,028.48
Oct., 1948-Sept., 1949	1,081.81	264.86	227.15	3,048.76	4,622.58
Oct., 1949-Sept., 1950	1,210.79	756.55	168.47	3,361.99	5,497.80
Oct., 1950-Sept., 1951	1,181.42	543.42	375.26	3,547.63	5,647.73
Anticipated Budget, 1951-1952	$1,200.00	$ 550.00	$500.00	$3,897.00	$6,147.00

LIBRARY BUDGET

Actual experience has produced two significant changes in our original tentative annual budget, as shown in the table. We overestimated the cost of binding our journals—all our journals are permanently bound promptly on the completion of a volume—and we did not foresee the general inflationary trends that would require upward adjustment of the salary paid to our full-time librarian. In spite of these developments, the total expenditure for the five-year period exceeded the anticipated operating expenses of $25,000 by only $233.78. Approximately 25% of the total was derived from staff dues and nurses' library fees. During the current fiscal year, the hospital will spend out of general funds about $5,000 for library purposes.

LIBRARY CONTENT AND SERVICES

The conviction of the original library committee that the library should concentrate on journals rather than on textbooks has been reaffirmed not only by newly appointed members of the committee but also by staff approval. We are now receiving 126 periodicals, of which 6 are gifts to the library. Represented among our journals, in addition to the recognized

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Pathologist (Dr. Rosahn) and Medical Librarian (Mrs. Karpman), New Britain General Hospital.

[1] Rosahn, P. D., and Olmstead, M. V. O.: The Medical Library of the New Britain General Hospital, Connecticut M. J. **12**: 23, 1948.

[2] The following persons have served on the library committee: Dr. H. Bellach, Dr. A. J. Canzonetti, Miss A. E. Mooth, R.N., Dr. P. D. Rosahn, Dr. D. E. Wilson, Miss V. Duxbury, R.N., Dr. H. M. Clarke, and Dr. V. J. Squillacote. The first five listed here constitute the library committee at this writing.

periodicals covering the various clinical specialties of medicine, nursing, and dentistry, and also of hospital administration, are many others in the various basic sciences. An attempt has been made to supplement our journals with accredited textbooks covering the different medical specialties, but preference has consistently been given to journals. Accessions for the year ending Sept. 30, 1951, comprised 199 bound volumes of periodicals and 52 textbooks. Our inventory on this date included 1,677 bound volumes of periodicals and 1,239 textbooks. All our journals now have running files of bound volumes dating back for a minimum of five years.

The books and journals in our own library have been supplemented by interlibrary loan material borrowed from many generously cooperating organizations. Among these are the Yale Medical Library, the American Medical Association, the Armed Forces Medical Library, the Hartford Medical Society Library, and the New Britain Institute Library. In the year ending Sept. 30, 1950, for example, the following interlibrary loans were made: 91 bound journals, 52 current periodicals, 13 textbooks, 84 films and slides, 10 pamphlets, 2 microfilms, and 4 photostats and 12 paintings for display on the library walls.

All the libraries mentioned have been extremely helpful in supplying us with material not in our inventory, but special mention should be made of the Yale Medical Library, our principal source of interlibrary loan matter. We early profited by the counsel of the librarian and staff of that great institution, and they have continued to provide us generously with help and guidance. Through our institutional annual contribution to the Associates of the Yale Medical Library, we have attempted to give tangible if modest expression of our appreciation for the interlibrary loan service that the Yale Medical Library has given us.

CLASSIFICATION SYSTEM

It has been our experience that current periodicals, bound or unbound, are easily located on the shelves when arranged in alphabetical order according to the first important word of the title. We have not found it necessary to classify journals according to subject, but this can easily be done in the future if expediency demands such a classification.

At the time of organization of the library, all the major classification systems were reviewed in order that we might determine which would most suit our requirements. These systems have their individual merits, but it was decided that the Cunningham System was the best for our needs, and it was therefore adopted. Subsequent experience has indicated that the Cunningham System, because of its elasticity and simplicity, is ideally designed for a library of our size and type.

ATTENDANCE AND CIRCULATION

The value of a library is not in the amount and variety of material it contains, in the spaciousness of its quarters, or in the cost of its maintenance, but rather in the use to which it is put. Our library has become the foundation on which a vigorous residency training program has been built. Moreover, it has provided a stimulus to our medical and nursing staffs in their desire to maintain familiarity with the current literature. During the 1950-1951 fiscal years there were 867 recorded visits by members of the medical staff, 784 visits by nursing personnel, and 666 visits by others. In addition, there was an unknown number of visits by persons who did not sign our visitors' daily record. In the same year, the following items were borrowed: 421 current periodicals, 169 bound journals, and 657 textbooks. These visits and loans were without doubt important factors in maintaining high medical standards in the hospital.

But more specific benefits accrued to those of our medical staff who were preparing for specialty board examinations. Twelve members of our resident and general staff successfully took these examinations within the past three years. All of them prepared for the ordeal with the help of material in our own library and through our interlibrary loan arrangements. In no instance was it necessary for these physicians and surgeons to spend valuable time and energy traveling to distant places for their library needs.

In the earlier report already mentioned, it was indicated that there are extant two basic conflicting attitudes in library policy. With rigid custodial supervision, and restricted circulation of textbooks and journals for outside reading, a minimum of losses may be expected to accrue. On the other hand, where custodial care is only incidental, and where free withdrawal of library material is permitted and encouraged, the library content receives wide circulation, but losses of many items may be anticipated. The library committee was of the opinion that it was more desirable to have wide circulation with its accompanying probable losses than to have limited circulation and no losses. Accordingly, a liberal policy was adopted, by which access to the library was permitted every day round the clock, even in the absence of the librarian, and at all times library material other than reference books and encyclopedias could be withdrawn. This policy has been happily rewarded, for we have incurred no losses during our five years of operation.

COMMUNITY SERVICE

The only medical library in the city of New Britain is located at the General Hospital. Since many professional persons active in the community in medical and allied fields do not have hospital appointments, it was early decided to invite them to use the facilities of the hospital library. Retired physicians, city-employed physicians, industrial physicians, dentists, nurses, and the faculty and students of the State Teachers College have been encouraged to use the resources of our library. This doctrine of a community medical library, organized and maintained at the local community hospital, is another example of the many services other than the care of the hospitalized sick that a hospital can render to the community in which it is located.

MEDICAL LIBRARY POLICIES

The following library policies are being pursued at this writing:

1. The medical library consists of a main library and a library annex. The main library is located in the basement of the main hospital building and consists of a reading room and stack room, with additional storage space across the hall from the main quarters. General reference books, medical journals and books, and bound or unbound periodicals to be issued on a loan basis are housed in the main library. The library annex is located in the nurses' residence and comprises reference volumes and other material that may not be borrowed. The annex also provides space for storing bound journals dating back more than 10 years.

2. The main library is intended for use by all professional personnel connected with the hospital. This includes physicians, dentists, nurses, dietitians, physical therapists, technicians, and social workers. Members of these professional groups in the community, not officially connected with the hospital, are also invited to use the library facilities, with loans made at the discretion of the librarian.

3. The library annex is for the convenience of the nursing staff. It is unsupervised and is open at all times.

4. The main library is open and the librarian is on duty on Monday through Friday from 8 a. m. to 4:30 p. m. and on Saturday from 8 a. m. to 12 noon. It is closed on Sundays and on all legal holidays officially observed by the hospital. During the hours when the main library is officially open, it may be used both as a reading room and for the issuance of loan material.

5. During the hours when the library is officially closed, the key may be obtained from the admitting office if signed for and returned promptly after the user leaves the library.

6. All books and periodicals taken from the main library must be charged. When the librarian is off duty, a self-charging system is permitted. The borrower fills out a loan slip and drops it into the depository provided.

7. With the exceptions noted below, books and bound periodicals may be borrowed for two weeks and may be renewed for another two weeks, provided they have not been requested by another reader. If, during the period of renewal, they are requested by another reader, they are subject to recall at once. If a volume that is on loan is needed for a reserved shelf, it may be recalled before the limit of the loan period. The latest bound volume of a journal may be borrowed for one week only.

8. Reference books, such as systems of medicine and surgery, bibliographies, indexes, dictionaries, encyclopedias, current periodicals (latest issue received), and books on the "new book table" may not be taken from the library under any circumstances.

9. Unbound periodicals, books reserved for special courses, and reference books (except those restricted under paragraph 8) may be borrowed for overnight use only, i. e., after 4 p. m. Monday through Friday and after 11 a. m. Saturday. They must be returned to the library by 10 a. m. the following morning. Such material taken out Saturday must be returned by 10 a. m. Monday. When the library is officially closed, one may return books and periodicals by using the drop boxes provided in the staff room and at the main library entrance.

10. If a book or journal is lost, the borrower will be taxed the cost of replacement.

PERIODICAL SUBSCRIPTION LIST

Much thought was originally given to the selection of journals. Any newly published journal is reviewed by the library committee, which decides whether or not to add it to our subscription list.

THE HOSPITAL MEDICAL LIBRARY

A library is a living organism. To survive it must be generously nurtured, carefully guided, and constantly tended. A library consisting of randomly selected books and journals, housed on a few dusty shelves, in inaccessible, cheerless quarters, an undeveloped, unused and uninspiring library, is a dead library. A quiet and respectful interment, as befits the honored dead, should be its generous reward. A library proudly supervised, judiciously enlarged through carefully chosen acquisitions, groomed, culled and cultivated, is a living library. Such a library provides knowledge and sustenance to those who seek its haven.

How can the average hospital acquire such a library? Money is necessary, yes. Money provided by the staff, by nurses, and by the administration. But above and beyond the need for money, there must be the deep conviction by medical staff and by administration, that no other single use of funds will so measurably improve the medical standards of the institution. Such a conviction does not arise *de novo.* One or more dedicated souls, members of the staff, must, by discussion, persuasion, exhortation, and conciliation, strive to engender this concept in both staff and administration. Once this has been accomplished, the library is secure.

92 Grand St.

MEDICAL MOTION PICTURES

Journey to Reality: (PMF 5116C) 16 mm., black and white, sound, showing time 40 minutes. Produced in 1951 by the United States Army. Procurable on loan from Area Surgeon of the Army Area in which the request originates.

This film points out the value of prescribed occupational therapy and shows the need for cooperative efforts between psychiatrists and occupational therapists as well as other ward personnel. A number of condensed case histories are given and the various therapeutic approaches illustrated by excerpts from the patient's daily activity. For example, the overactive manic patient is put at a loom, which allows her to expend considerable energy along useful channels; the hostile patient is encouraged to "make his own choice" in the metal shop, where he can work off feelings by pounding; the retarded and depressed patients are given simpler, less taxing activities.

A more descriptive title for this film might possibly have been "The Beginning of the Journey to Reality," since the span of the patients' total program is presented briefly, both in terms of time and of things to be done. Some of the case history portrayal is a bit strained and unrealistic. The medical aspects of the program are well presented. The photography and narration are well done. Its greatest usefulness would be for the orientation of hospital personnel generally and in particular occupational and physical therapists and psychiatric social workers. It could also be used in beginning courses for occupational therapists. Physicians interested in psychiatric problems would also be interested in this film.

BUREAU OF LEGAL MEDICINE AND LEGISLATION

MEDICOLEGAL ABSTRACTS

Charitable Hospitals: Liability for Burns to Child of Pay Patient.—The plaintiff, an infant, filed suit for damages for burns alleged to have been caused by the negligence of the servants of the defendant hospital. The plaintiff was placed in a bassinet immediately after her birth, and a hot water bottle was also placed therein to keep her warm. About two hours later, after removal to the nursery, she was found to have burns on her head and body. The defendant hospital made a motion for a summary judgment in its behalf on the ground that it was a charitable hospital and therefore free from liability in this case. The motion was heard by the superior court, Newcastle, Del.

The question of whether a nonstock, nonprofit, charitable corporation, which conducts and maintains a hospital by public and private contributions and funds received from those who are able to pay, is liable for injury to a patient caused by the negligence of its agents and employees, such as nurses, has never been decided in this state, said the court. It is an important question, and many decisions have been rendered on it by the various courts throughout the country. These decisions are not only conflicting but many of them are also accompanied by dissenting opinions. After discussing the history of the liability and nonliability of charitable institutions from 1846 to the present time and after citing numerous cases considering this question, the court quoted from section 294 of Harper on "The Law of Torts" as follows: "The immunity of charitable corporations in tort is based upon very dubious grounds. It would seem that a sound social policy ought, in fact, to require such organizations to make just compensation for harm legally caused by their activities under the same circumstances as individuals before they carry on their charitable activities. The policy of the law requiring individuals to be just before generous seems equally applicable to charitable corporations. To require an injured individual to forego compensation for harm, when he is otherwise entitled thereto, because the injury was committed by the servants of a charity is to require him to make an unreasonable contribution to the charity, against his will, and a rule of law imposing such burdens cannot be regarded as socially desirable nor consistent with sound policy."

It is contended, said the court, that the overwhelming weight of authority in the United States is in favor of the exemption of charitable corporations from liability for the torts of their agents and employees. After reading many cases decided in the various states throughout the country, the court was convinced that the contention is, at least, doubtful. The numerous distinctions made in the decisions certainly weaken their effect. There is quite a difference, the court continued, between charitable institutions today and those that existed when they were first held to be exempt from tort liability. Those that existed at that time were small and the funds that they had were not large when compared with many that we find today, the funds of which are provided for by foundations and in some instances are unlimited. The argument made in some of the earlier cases, that the funds of the institution would soon become exhausted or that persons would not make donations for charitable purposes if they were used to pay damages does not apply with the same force as it did at that time. There can be no doubt that the trend of decisions today and the views expressed in many of the law journals is in favor of imposing full liability on charitable corporations for the torts of their agents and employees.

Continuing, the court said that it was convinced that the general principle that makes every person responsible for his own legally careless action should apply with equal force to charitable corporations. The doctrine of *respondeat superior* also applies to charitable corporations and makes them responsible for the negligent acts of their agents and employees, when such acts are clearly incidental to the business of the corporation, to the same extent that individuals are. This is true whether or not the beneficiary pays for the service that he receives. It is unreasonable to hold that persons who do not pay for the service that they receive from a charitable corporation cannot recover for injury resulting from the negligent acts of those for whose acts the corporation is responsible when such acts are incidental to the business of the corporation. The court, therefore, held that whether the defendant was negligent by failing to provide proper equipment in the delivery room or nursery of his hospital or whether the agents or employees of the defendant were negligent by failing to properly care for the plaintiff were questions of fact for the jury, but it denied the defendant's motion for summary judgment. *Durney v. St. Francis Hospital, 83 A. (2d) 753 (Delaware, 1951).*

Ethics: Sale of Dental Practice by Widow for Share in Future Earnings.—The plaintiff sued to recover sums paid under a conditional sales contract covering the sale of a dental practice. From a judgment in favor of the defendant, the plaintiff appealed to the Supreme Court of Washington.

The defendant was the widow and executrix of the estate of a deceased dentist and as such became owner of the equipment, accounts receivable, lease, and going business of what was known as "Dr. Conway's Dental Clinic." She entered into a contract for the sale of this asset to the plaintiff dentist. The purchase price was to be $300 a month for ten years, a total of $36,000. In addition, and specifically as payment for the "going business," the defendant was to receive a share of the net profits for the first five years, her percentages to be 70% for the first year, 60% for the second year, and 55% for the following three years. Before any profits were to be divided, however, $300 was to be paid on the purchase price, and the plaintiff was to draw $750 each month, in addition to the payment of all other operating expenses. The plaintiff was taking over an established dental practice with no down payment. The contract with the plaintiff further provided that the defendant would ". . . of necessity dedicate a considerable portion of her time to the protection of her security and advancement of the interest of the business in order that it may return to her the amount contemplated by the parties as purchase price under all of the circumstances now existing." Implementing the above was paragraph 7 providing: "It is further agreed that the doctor shall spend his whole time to the professional care of the office and shall have exclusive control over professional conduct and practice in said office and that the seller shall have the right to be the manager of said business and shall have the right to superintend the business management of the office." Title to fixtures and equipment was reserved to the defendant to secure the payments provided for by the contract; the lease was to be renewed in her name until payment under the contract was complete, at which time it was to become the property of the plaintiff. All new accounts receivable were to be assigned to the defendant during the first five years "as security only."

After the contract had been in force about six months, the plaintiff notified the defendant that the contract was illegal as far as it provided for her participation in the business in any way, and any sums that it was contemplated that the defendant should receive therefor would be illegal. The notice also stated that the books of the clinic would no longer be open to the defendant's inspection or approval and that the defendant was to remain away from the clinic and the employees except for reasonable inspection. In conclusion, the notice said that in the plaintiff's opinion the contract was principally a legal one and that the illegal portions, which contemplated paying the defendant a percentage for management, could be deleted and the management cancelled, but the balance of the contract continued in force.

The Supreme Court said that it could find nothing in the contract to the effect that the percentage payments to the defendant were to be compensation for her management. The contract definitely stated that the percentage of profits she was to receive was "payment for the going business herein contracted to be sold." There was nothing unlawful in the defendant's ownership of the property and interests that she sold to the plaintiff, although, under state law and under decisions of the Washington Supreme Court, the defendant could not have employed anyone to take over her husband's practice and operate it for her. It can not be gainsaid that she had the right to sell that which she owned, either for cash or on terms, and, if on terms, she had the right to protect her interests in any way consistent with the public policy of the state. We have carefully examined the contract, the court continued, and as far as we can determine, it is a bona fide contract of sale calculated to protect and to benefit both parties, and none of the provisions of that contract brand it illegal or contrary to public policy. The fact that the defendant was to have a relatively large percentage of the net profits during the first five years does not establish that she owns, maintains, or operates an office for the practice of dentistry, but rather that she and the plaintiff had agreed on a formula for arriving at a value of the "going business," which we conceive to be the good will of the established dental practice.

The two provisions of the contract that gave the plaintiff great concern are the ones granting the defendant (a) a percentage of the profits and (b) the right to be the business manager and to superintend the business management of the office. It is common knowledge, said the Supreme Court, that many physicians and dentists, and certainly most clinics, leave the details of business records, the issuing of statements, the collection of accounts, and the details of disbursements for supplies and equipment to personnel employed for that purpose, and we do not regard the handling of those details as constituting the practice of medicine or dentistry, although their capable performance may be essential to make that practice profitable. The fact that the same details are supervised by one who, instead of being an employee, is selling the practice under a conditional sales contract does not of necessity constitute such ownership, maintenance, or operation of an office for the practice of medicine or dentistry as is contemplated by the statute, although it could well be a factor requiring a closer scrutiny of the entire transaction to determine whether it is actually what it purports to be or, instead, is a scheme to subterfuge to violate the law. If the plaintiff is entitled to any relief, it is not because the contract is illegal, but because the defendant has exercised a control over the business that constitutes, in fact, the management and operation of a dental office. Some of the conduct complained of by the plaintiff might well be within her rights under the provisions of the contract that were designed for her protection, but, concluded the Supreme Court, the allegations that she "effectuated her control and management of said business by maintaining control of the finances, control of the books, control of the patients, and control over appointments of patients . . .," if true, clearly entitles the plaintiff to relief of some sort.

The plaintiff also contended that the defendant could receive no percentage of the profits, that so long as he paid her $300 a month he was complying with all the legal provisions of their contract, and that he could deny her even an examination of the books. It must be borne in mind, said the court, that the plaintiff did not allege or even suggest that this was not a bona fide sale or that the sale was being used as a blind or pretext to enable the defendant to practice dentistry in contravention of the statute. We are in entire accord with the trial court that the plaintiff cannot recover on the theory that a portion of the contract is illegal. Plaintiff's recovery, if any, must be on the theory that the defendant's conduct so breached the contract as to warrant his abandonment of it, to his damage.

The Supreme Court, therefore, felt that the defendant's demurrer should have been overruled and the plaintiff afforded an opportunity to prove his allegations. The judgment of the trial court in favor of the defendant was accordingly reversed and the cause remanded to the trial court for further hearing. *Prichard v. Conway, 234 P. (2d) 872 (Washington 1951).*

MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A.M.A. Arch. Internal Medicine, Chicago

89:523-688 (April) 1952

*Control of Hypertension by Hexamethonium and *l*-Hydrazinophthalazine: Preliminary Observations. H. A. Schroeder.—p. 523.
*Histoplasmosis in Epidemic Form: Follow-Up Study. B. Schwartz and L. J. Spitz.—p. 541.
Mental Changes Associated with Hyperparathyroidism: Report of Two Cases. T. E. Fitz and B. L. Hallman.—p. 547.
Physical Medicine and Rehabilitation in Acute Myocardial Infarction. L. B. Newman, M. F. Andrews, M. O. Koblish and L. A. Baker. —p. 552.
Heatstroke in Parkinsonism. R. E. Litman.—p. 562.
Benign Bundle Branch Block. J. P. Vazifdar and S. A. Levine.—p. 568.
*Leptospiral Meningitis. P. B. Beeson and D. D. Hankey.—p. 575.
Effect of Therapy on Blood Volume, Blood Pressure, and Spleen Size in Polycythemia Vera. T. C. Prentice, N. I. Berlin and J. H. Lawrence. —p. 584.
l-Arterenol (Levophed®) in Treatment of Shock Due to Acute Myocardial Infarction. A. J. Miller and L. A. Baker.—p. 591.
Whipple's Disease: Review of Literature and Report of Two Cases. F. R. Russo.—p. 600.
Rationale and Clinical Use of Steroid Hormones in Cancer. S. I. Griboff. —p. 635.

Hexamethonium and *l*-Hydrazinophthalazine in Hypertension.—An antihypertensive drug of only moderate potency, *l*-hydrazinophthalazine, was found to be considerably more effective in patients who had undergone sympathectomy. Malignant hypertension was also found to be susceptible to its action. This suggested that "medical sympathectomy," induced by sympatholytic or ganglionic blocking agents, might enhance the antihypertensive action of *l*-hydrazinophthalazine. It was decided to combine its use with that of a soluble, apparently innocuous ganglionic blocking agent, hexamethonium chloride. Forty patients with chronic arterial hypertension severe enough to require hospitalization, and resistant to dietary measures, thiocyanate administration, surgical measures, and psychotherapeutic procedures were treated. Hexamethonium chloride was usually given in initial doses of 125 to 250 mg. every eight hours, which were increased daily until a maximum of 500 mg. every four hours was reached, or blood pressure showed wide fluctuations, reaching normal levels. At this point *l*-hydrazinophthalazine was added in doses of 25 to 50 mg. every eight hours, in amounts increased by daily increments until a dose of 100 mg. every four hours was reached or the blood pressure had become normal. After normal blood pressure had been achieved in the hospital, a member of the patient's family was instructed in measuring blood pressure; a schedule was arranged for use of the drugs at home, and the patient was discharged. Patients with "benign" arterial hypertension achieved normal or nearly normal blood pressure in every case. Continuous administration was essential for control. In some cases "malignant" hypertension was well controlled with oral doses, while in others it was only partially controlled. The malignant stage was altered favorably in all but two. The method was practical for use at home in intelligent patients. It represents a way of controlling hypertension, but it is not a "cure." Long-term results cannot be evaluated until more time has elapsed. Subsequent experience with 60 patients who have been taking these drugs for from 10 to 28 weeks has shown that 30 have maintained normal blood pressure levels. Of 22 in the malignant stage, 8 have normal blood pressure and 12 slight hypertension; the malignant stage has regressed in all instances. Tolerance to the drugs has not appeared.

Epidemic Histoplasmosis: Follow-up Study.—Histoplasmosis in its acute form occurs either as a benign or as a fulminating fatal disease. It usually appears sporadically, but this paper reports follow-up observations in 1 of 26 cases of diffuse pneumonitis that occurred as an epidemic at an Army camp in 1944 and were reported as cases of "an unusual pulmonary disease." The acute stage of the disease lasted one week. At this time roentgenographic inspection revealed a diffuse mottling throughout both lung fields. No tubercle bacilli were found, and the Mantoux test gave a negative reaction. The coccidioidin skin test likewise gave a negative reaction. Agglutination tests for brucellosis, tularemia, and typhoid and serologic tests for psittacosis all gave negative results. The reactions to serologic tests for histoplasmosis were also negative. A physical examination of one patient six years later showed no abnormality, but roentgenograms of the chest revealed shadows suggestive of the end-stage of pulmonary histoplasmosis. A skin test with histoplasmin gave a strongly positive reaction (4+), while reactions to coccidioidin and Mantoux tests were negative. A complement-fixation test for histoplasmosis, using the yeast phase antigen of Histoplasma capsulatum, was positive to a titer of 1:140 and negative for other fungi. Observations on this patient suggest that the other members of the group thought to have an unusual pulmonary disease also had a benign form of pulmonary histoplasmosis. The authors mention four similar epidemics, the cause of which has never been established. It is hoped that this report will stimulate further follow-up study in these cases.

Leptospiral Meningitis.—Only three strains of Leptospira (Lept. icterohemorrhagiae, Lept. canicola, and Lept. pomona) have been identified here. Although cases of Weil's disease (spirochetal jaundice) have been recognized in various parts of the country, there is reason to believe that the incidence of leptospiral infection is probably greater than is appreciated. The authors searched for evidence of leptospirosis in patients with "benign aseptic meningitis." Positive results were obtained in 6 of 35 patients admitted to one Atlanta hospital during a two year period. A seventh patient was observed in another Atlanta hospital during the same period. Serologic tests for evidence of leptospiral infection were made on approximately 500 pairs of serums obtained from other localities in the United States. These had been submitted to other laboratories for serologic tests for neurotropic viral infection. It was known, however, that many of them had been collected during epidemics of mumps, poliomyelitis, and encephalitis. Leptospiral agglutinins in titers of probable significance were found in 17 instances. Data on these 24 patients permit the following statements: 1. Leptospiral meningitis occurs in many parts of the United States. 2. The clinical features resemble those of meningitides of viral origin, such as mumps, lymphocytic choriomeningitis, and nonparalytic poliomyelitis. 3. Ten of the 24 cases occurred in the month of August. 4. The disease is commoner in males below the age of 30. Laboratory procedures for the diagnosis of leptospiral infection are described.

American Journal of Public Health, New York

42:353-498 (April) 1952. **Partial Index**

Emergency Sanitation Lessons from the 1951 Flood in Kansas. D. F. Metzler.—p. 364.
Principles of Emergency Feeding for a Large Metropolitan Area in Catastrophe. R. S. Goodhart and N. Jolliffe.—p. 373.
Membrane Filter in Water Quality Tests. H. F. Clark and P. W. Kabler. —p. 385.
Use of Differential Media with Membrane Filter. P. W. Kabler and H. F. Clark.—p. 390.
Immunization of Adults Against Diphtheria and Tetanus. G. Edsall. —p. 393.
Lead Poisoning: Case Finding Program. J. E. Smith, B. W. Lewis and H. S. Wilson.—p. 417.
Evaluation of "Breast Self-Examination" Film. E. M. Cohart and J. K. Hill.—p. 430.

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Titles marked with an asterisk (*) are abstracted.

American Review of Tuberculosis, New York

65:357-504 (April) 1952

Chemotherapy of Experimental Tuberculosis: V. Isonicotinic Acid Hydrazide (Nydrazid) and Related Compounds. J. Bernstein, W. A. Lott, B. A. Steinberg and H. L. Yale.—p. 357.
*Antituberculous Properties of Hydrazines of Isonicotinic Acid (Rimifon, Marsilid). W. Steenken Jr. and E. Wolinsky.—p. 365.
Pharmacologic and Toxicologic Observations on Hydrazine Derivatives of Isonicotinic Acid (Rimifon, Marsilid). W. M. Benson, P. L. Stefko and M. D. Roe.—p. 376.
Pharmacology of Isonicotinic Acid Hydrazide (Nydrazid). B. Rubin, G. L. Hassert Jr., B. G. H. Thomas and J. C. Burke.—p. 392.
Hydrazine Derivatives of Isonicotinic Acid (Rimifon, Marsilid) in Treatment of Active Progressive Caseous-Pneumonic Tuberculosis: Preliminary Report. E. H. Robitzek and I. J. Selikoff.—p. 402.
Absorption, Distribution, Excretion, and Short-Term Toxicity of Isonicotinic Acid Hydrazide (Nydrazid) in Man. D. F. Elmendorf Jr., W. U. Cawthon, C. Muschenheim and W. McDermott.—p. 429.
Observations on Electrocardiogram in Surgically Treated Pulmonary Tuberculosis. V. O. Hertzman and A. K. Mathisen.—p. 443.
Prevalence of Previously Unknown Tuberculosis in Persons Not Participating in Mass Chest Roentgenographic Surveys. W. T. Palchanis. —p. 451.
Traction Diverticula of Esophagus in Middle Lobe Syndrome. H. L. Katz.—p. 455.
Spirometric Studies in Pneumoperitoneum. G. C. Leiner and S. Abramowitz.—p. 465.
Correlation of Biologic Properties of Strains of Mycobacterium with Their Infrared Spectrums: II. Differentiation of Two Strains, H37Rv and H37Ra, of M. Tuberculosis by Means of Their Infrared Spectrums. H. M. Randall, D. W. Smith and W. J. Nungester.—p. 477.

Antituberculous Properties of Hydrazines of Isonicotinic Acid.—Preliminary observations on two hydrazines of isonicotinic acid ("rimifon" and its isopropyl derivative, "marsilid") indicate that they have tuberculostatic and tuberculocidal action in vitro and a beneficial effect on animals infected experimentally. Parenteral administration of 10 mg. of "marsilid" twice daily (approximately 35 mg. per kilogram of body weight daily) was tolerated well by guinea pigs for 60 days; up to 35 mg. per kilogram daily of "rimifon" was tolerated well for six weeks. Tolerance tests for oral administration to guinea pigs showed that doses up to 40 mg. per kilogram daily of both "marsilid" and "rimifon" were tolerated well for two weeks. Intramuscular tests with rabbits showed good tolerance for 70 days with doses of 30 mg. twice daily (20 mg. per kilogram daily) of "marsilid," and for 21 days with doses of 15 mg. twice daily (9 mg. per kilogram daily) of "rimifon." Guinea pigs infected subcutaneously with virulent human tubercle bacilli and treated with "marsilid" 21 days later revealed very little evidence of tuberculosis grossly when killed 73 days after infection. Thirty days of treatment produced marked changes in the tuberculin skin reactions, which were positive at the beginning of therapy. Five guinea pigs infected by the intracerebral method, which is rapidly and uniformly fatal in untreated animals, were treated with "marsilid" and remained well during 60 days of treatment; since treatment was stopped, 4 have died and the fifth is showing progressive paralysis of the hindquarters. Rabbits infected intravenously with highly virulent bovine tubercle bacilli and treated with "marsilid" for 71 days remained well and failed to develop miliary pulmonary lesions demonstrable by roentgenography. Discontinuance of treatment, however, was followed by relapse of tuberculosis within 10 weeks.

American Surgeon, Atlanta, Ga.

18:341-436 (April) 1952

The Training of a Surgeon. L. J. Starry.—p. 343.
Unusual Bony Reaction to Faulty Intramedullary Pin. E. B. Carpenter and L. S. Miller.—p. 352.
Colostomy Stoma Tumors. E. L. Miloslavich.—p. 358.
Papillary Thyroid Cancer Originating in Thyroglossal Cyst. B. L. Aronoff.—p. 362.
Dermal Grafts in Repair of Massive Abdominal Hernias: Use of New Split-Split Flap Technic. J. O. Shaffer.—p. 372.
Multiple Cancers of Oral Cavity. L. T. Byars and R. Anderson.—p. 386.
*Inflammatory and Neoplastic Lesions of Small Intestine. K. C. Sawyer and A. E. Lubchenco.—p. 392.
Plastic Reconstruction of Trachea and Bronchi. D. L. Paulson.—p. 403.
Waterproof Plaster. M. C. Cobey.—p. 413.
Necrotizing Fasciitis. B. Wilson.—p. 416.

Lesions of the Small Intestine.—Sawyer and Lubchenco feel that too few lesions of the small intestine are diagnosed before operation or autopsy. They summarize their experience in treating 26 inflammatory and neoplastic lesions involving the jejunum and ileum. Lesions proximal to the ligament of Treitz and congenital anomalies were not included. The 13 patients with inflammatory lesions included 8 with regional ileitis, which is a nonspecific chronic recurrent granulomatous disease characterized by a necrotizing, ulcerating, inflammatory process. Abdominal pain, diarrhea, and secondary anemia are the cardinal features. Further confirmation of the diagnosis can be obtained from the roentgenographic recognition of the string sign. The treatment of choice is resection of the involved ileum and the right side of the colon with end-to-end ileotransverse colostomy. There were two patients with tuberculosis of the small intestine, two with diverticulitis, and one with periarteritis nodosa. The seven patients with benign neoplasms included two with carcinoid, two with endometrioma, and one each with lymphangioma, leiomyoma, and lipoma. Five of the six malignant lesions were carcinomas and one was a lymphoma. Carcinoma is the commonest malignant tumor of the small intestine. The symptoms are usually related to perforation, bleeding, or obstruction. The authors stress that any patient with unexplained diarrhea, intestinal bleeding, or abdominal pain should be subjected to careful clinical and roentgenologic study of the small intestine. Examination of the entire small intestine during routine laparotomy is worth while, and frequently reveals the pathological condition responsible for the patient's complaints.

Angiology, Baltimore

3:69-204 (April) 1952

*Intraarterial Priscoline Therapy for Peripheral Vascular Disturbances. H. I. Lippmann.—p. 69.
Cardiac Catheterization and Other Physiological Studies in 50 Cases of Congenital Heart Disease. P. R. Lurie, F. D. Gray Jr. and R. Whittemore.—p. 98.
Budd-Chiari's Syndrome: High Inferior Vena Caval Obstruction Demonstrated by Venography. B. Bronte-Stewart and R. H. Goetz.—p. 167.
Experimental Studies of Gastric Blood Flow: Preliminary Report. J. R. Vane and J. E. Thompson.—p. 179.
*Dicumarol: Dangerous and Rather Ineffective Drug. F. Martorell. —p. 185.
Surgical Treatment of Congenital Abnormalities of Heart. A. R. Albanese.—p. 189.
Thrombophlebitis Migrans; With Particular Reference to Thrombosis of Renal Veins. N. Vinther-Paulsen.—p. 194.

Priscoline® Intra-Arterially in Vascular Disturbances.—If priscoline® (2-benzyl-4,5-imidazoline) hydrochloride is injected rapidly into the regional artery of the human limb, an increase in blood flow can be observed through the main arterial channels, in the blood vessels of the skin, and throughout the deep tissues of the extremity. This effect lasts only a few hours, but after a series of injections it becomes more sustained. At a Bronx, N. Y., hospital 76 patients with peripheral vascular disease were treated by intra-arterial administration of priscoline.® In 11 patients who had gangrene of the heel, the lesions of 8 healed and 3 had to undergo amputation. This compares favorably with a control group of 11 cases in which 9 patients had to undergo amputation. Of 49 ischemic lesions in other parts of the extremities, 23 healed, and in 26 cases intra-arterial therapy was ineffective. In two of seven cases of acute arterial occlusion, embolism was the cause, and both of these patients recovered without trophic lesions; of the five who had thrombotic occlusions, two required an amputation. Four cases of iliofemoral thrombophlebitis with spastic arterial occlusion rapidly improved with one or several intra-arterial priscoline® injections. In four patients with intractable Raynaud's disease attacks disappeared during and for some time after intra-arterial priscoline® therapy, the noninjected arm serving as a control. In five cases of functional vascular disturbances, intra-arterial administration of priscoline® had a temporary and localized vasodilator effect. Priscoline® is injected rapidly into the artery in amounts just short of systemic reactions. The most effective injection schedule must be determined individually. The goal of intra-arterial priscoline® therapy in peripheral vascular disease is a sustained (not always maximal) vasodilatation in the extremity. This is maintained by regular injections over a period of days or weeks. Interruptions of the schedule or a clumsy injection technique that produces pain and vasoconstriction may prevent sustained vasodilatation. Contraindications to intra-arterial priscoline®

therapy are systemic reactions after intra-arterial injections of 10 mg. of priscoline® or less, a temporary decrease in local blood flow or edema, and skin maceration in an ischemic area after intra-arterial injection. Intra-arterial injection of this drug is a useful diagnostic test similar to a posterior tibial or lumbar sympathetic nerve block, and, carefully used, may be a valuable adjunct to treatment of peripheral vascular disorders.

Dangers of Bishydroxycoumarin.—From clinical observations and from experiments on rabbits Martorell deduced that the effective antithrombotic dose of bishydroxycoumarin (dicumarol®) and the hemorrhage-producing dose are too nearly the same. Although this drug has the advantage of being administered orally, its therapeutic efficacy is doubtful and the danger attendant on its administration reaches such proportions that it should be asked whether the risk is less for the patient from pulmonary embolism, which is not necessarily a complication of venous thrombosis, or from death by hemorrhage caused by the drug even under the most favorable conditions and with the most rigorous control of the prothrombin index.

Blood, New York

7:387-482 (April) 1952

Mechanism of Hemolysis in Paroxysmal Cold Hemoglobinuria: III. Erythrophagocytosis and Leukopenia. W. S. Jordan Jr., R. L. Prouty, R. W. Heinle and J. H. Dingle.—p. 387.

*Pathogenesis of Postirradiation Anemia. J. B. Kahn Jr. and J. Furth.—p. 404.

Changes in Cellular Composition of Lymph Caused by Ionizing Radiations. M. H. Ross, J. Furth and R. R. Bigelow.—p. 417.

Genetic Aspects of Sickle Cell Anemia and Microdrepanocytic Disease. E. Silvestroni and I. Bianco.—p. 429.

Blood Picture of Pyridoxine Deficiency in Monkey. K. J. Poppen, L. D. Greenberg and J. F. Rinehart.—p. 436.

Effect of Methylxanthines on Hypocoagulability Induced by Chloroform in Dog. J. B. Field, L. Graf and K. P. Link.—p. 445.

Pathogenesis of Postirradiation Anemia.—The hemolytic effect of large doses of irradiation is well established; the objective of this study was to determine whether irradiation within the lethal range injures erythrocytes in vivo. One group of rabbits was injected with radioiron-tagged erythrocytes shortly before exposure to 800 to 1,000 r and another group immediately after irradiation. A control group received the tagged erythrocytes but no irradiation. There was a drop in red cell mass in both irradiated groups as compared with the controls, but there was no difference between the two irradiated groups, i. e., between the behavior of irradiated and nonirradiated erythrocytes in irradiated hosts. These findings indicate that there is no direct effect of irradiation in the median lethal range on erythrocytes in vivo. It requires nearly lethal doses of x-rays to cause conspicuous anemia. In the fatally irradiated animals the drop in red cell mass and total circulating radioiron is precipitous; in those nonfatally irradiated the drop is gradual and relatively slight, even though the animals may have received the same dose. Increase of specific organ activity of the liver and spleen of irradiated animals can be correlated with hemosiderin deposits and congestion. Deposition of hemosiderin is a consequence of irradiation, its degree increasing with the effectiveness of the irradiation. In rabbits the erythrocyte masses obtained by two direct isotopic techniques (P^{32} and Fe^{59}) and by one indirect technique (T-1824 dye) differ by constant ratios. The Fe^{59}:P^{32} ratio is 0.89; the Fe^{59} values are regarded as correct. With the aid of conversion factors either technique can be used. If an indirect technique is employed and the erythrocyte mass is estimated on the basis of plasma volume measurements, it is necessary to calculate first the average body hematocrit value. This is done by multiplying the large vessel hematocrit by a conversion factor, which is 0.83 in normal rabbits. The authors conclude that irradiation at or below the median lethal dose does not hemolyze erythrocytes directly, but some change caused by massive irradiation results in increased destruction of erythrocytes. It is postulated that this change is damage to the capillary wall causing diversion of erythrocytes into tissue spaces and lymphatics, resulting in injury of some erythrocytes. Thus, the anemia of irradiation is in part relative, in part absolute.

Gastroenterology, Baltimore

20:375-534 (March) 1952

Protection of Liver by Dietary Factors. C. H. Best, W. S. Hartroft and E. A. Sellers.—p. 375.

Use of Lipotropic Factors in Treatment of Liver Disease. D. Cayer and W. E. Cornatzer.—p. 385.

*Effects of Diet and Choline on Fatty Infiltration of Human Liver. J. Post, J. G. Benton, R. Breakstone and J. Hoffman.—p. 403.

Plasma Antithrombin Titer in Incipient and Advanced Liver Failure. I. Innerfield, A. Angrist and L. J. Boyd.—p. 417.

Studies on Non-Parietal Component of Gastric Secretion in Humans: Part I. Variations in Electrolytes and Dissolved Mucin of Gastric Juice Following Insulin Stimulation. G. B. J. Glass, L. J. Boyd, I. J. Drekter and A. Heisler.—p. 430.

Id.: Part II. Present Concept of "Alkaline Constituent" of Gastric Juice. G. B. J. Glass and L. J. Boyd.—p. 442.

Studies on Pepsin Secretion: II. Effect of Vagal Resection for Duodenal Ulcer. D. T. Book, A. B. Chinn and A. J. Beams.—p. 458.

Banthine for Peptic Ulcer: Clinical and Physiologic Considerations. Preliminary Report. A. Winkelstein.—p. 464.

Gastric Mucosa in Acute Infectious Hepatitis. J. R. Loughead and F. C. Golding.—p. 471.

Treatment of Gastroduodenal Ulcerative Disease and Ulcerative Colitis with Detergent Complexes. O. E. Lobstein, B. J. Hull and S. J. Fogelson.—p. 474.

Effects of Diet and Choline on Fatty Infiltration of Liver.—This report is based on studies of 17 alcoholic patients with fatty infiltration of the liver. They were hospitalized, and clinical observation, liver function tests, and needle biopsies of the liver were made. They were placed on dietary schedules, and at intervals of 3 to 15 weeks the liver biopsies were repeated. The histological specimens were graded 0 to 4 plus for fat, periportal lymphocytic, and fibrous infiltration. Liver function tests were checked biweekly. These patients were not critically ill. Some were malnourished, others had jaundice, and all were in the state usually observed after cessation of excessive alcohol intake. One group of patients received the regular hospital diet of 250 gm. of carbohydrate, 75 gm. of protein, and 75 gm. of fat, supplemented with vitamins of the B complex group in a few cases, because of peripheral neuropathy and glossitis. A second group of patients was offered this diet, but the patients ate poorly and continued to drink heavily, through clandestine measures. A third group received the aforementioned diet to which choline was added. The basal diet had significant lipotropic effects in 13 patients. The ingestion by four patients of 4 to 17 gm. of choline bicarbonate with the basal diet did not increase the lipotropin effects observed with the diet alone. Continued ingestion of alcohol with poor dietary intake prevented the disappearance of fat from the liver. Thus the studies demonstrated that with intake of approximately 1 gm. of protein per kilogram of body weight effective lipotropism occurs within four weeks. Addition of two to six times as much choline as had been used previously had no added demonstrable effect, either on the clinical appearance of the patient, the liver function tests, or the cell structure of the liver.

Geriatrics, Minneapolis

7:87-164 (March-April) 1952

Practical Diagnosis in Preventive Geriatrics. B. V. White.—p. 87.

Therapeutic Nerve Block in Pain Syndromes of the Aged. D. L. Burdick.—p. 93.

*ACTH, Cortisone and Pregnenolone in Arthritis and Allied Diseases. T. H. McGavack.—p. 99.

Protein Requirements of Old Age. A. A. Albanese, R. A. Higgons, B. Vestal and others.—p. 109.

The Psychiatrist's Role in Care of the Aging. E. B. Allen and H. E. Clow.—p. 117.

Effects of Stress upon Cardiovascular Functions in Old Age. J. T. Flynn.—p. 123.

Transurethral Resection in Benign Hypertrophy of Prostate. C. D. Creevy.—p. 130.

Morbid Anatomy of Old Age. T. H. Howell and A. P. Piggot.—p. 137.

Corticotropin (ACTH), Cortisone, and Pregnenolone in Collagen Diseases.—Pregnenolone, cortisone, and corticotropin (ACTH) were given to 250 patients with a collagen or allied disease. Effective initial doses ranged from 400 to 1,000 mg., 75 to 150 mg., and 80 to 120 mg., and maintenance doses from 200 to 400 mg., 25 to 50 mg., and 20 to 40 mg., respectively. In patients with rheumatoid arthritis complete remission was obtained in 10 of 40, 4 of 12, and 4 of 5 patients treated with pregnenolone, cortisone, and corticotropin, respectively. The effect of cortisone and corticotropin on the joint disturbances was much greater than that of pregnenolone, which affected only the periarticular and psychic manifestations of the disease. In patients with gout both corticotropin and cortisone were superior to pregnenolone, bringing about a quicker and more complete action. In patients with acute and chronic lupus erythematosus pregnenolone seems useless; cortisone, though valuable, may fail; and corticotropin will bring amelioration and remission. Cortisone and corticotropin were equally useful in psoriasis, scleroderma, uveitis, and asthma, whereas pregnenolone had some effect in scleroderma and psoriasis. Of 31 patients with osteoarthritis, 16 obtained considerable improvement with pregnenolone in the periarticular structures. There was a decrease in muscular spasm, and an increase in mobility but no alteration in the joint lesion itself. None of the drugs appeared to influence the articular disturbance. From the clinical point of view, the temperature becomes normal rather rapidly and abruptly with cortisone and corticotropin, whereas with pregnenolone it is not influenced in lupus erythematosus, and in the other diseases it becomes normal more or less by lysis. Cortisone and corticotropin produce eosinopenia in patients in whom they are useful, while pregnenolone does not. With cortisone and corticotropin euphoria is pronounced, and, if the drugs are administered too long, the patient may show irascibility, despondency, and true psychotic manifestations. These untoward effects on personality are avoided with prolonged use of pregnenolone. Undesirable side-effects appeared in 3 of 211 patients treated with pregnenolone, 13 of 41 treated with cortisone, and 6 of 26 treated with corticotropin.

Illinois Medical Journal, Chicago

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Management and Outlook of Coronary Disease. R. S. Berghoff.—p. 208.
Disseminated Lupus Erythematosus with Severe Thrombocytopenia in a Negro Male. I. A. Friedman, W. H. Kleinschmidt and S. O. Schwartz. —p. 212.

Indiana State Medical Assn. Journal, Indianapolis

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Malignant Tumors in Bone. C. F. Gregory.—p. 301.

Journal of Bacteriology, Baltimore

63:301-428 (March) 1952. Partial Index

Sulfur Metabolism in Escherichia Coli: III. Metabolic Fate of Sulfate Sulfur. E. T. Bolton, D. B. Cowie and M. K. Sands.—p. 309.
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Synthesis of Glutamic Acid and Glutamyl Polypeptide by Bacillus Anthracis: II. Effect of Carbon Dioxide on Peptide Production on Solid Media. C. B. Thorne, C. G. Gómez and R. D. Housewright.—p. 363.
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Journal of Immunology, Baltimore

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Metabolic Studies of Rickettsiae: II. Studies on Pathway of Glutamate Oxidation by Purified Suspensions of Rickettsia Mooseri. C. L. Wisseman Jr., F. E. Hahn, E. B. Jackson and others.—p. 251.
Studies on Acute Disseminated Encephalomyelitis Produced Experimentally in Rhesus Monkeys: VII. Effect of Cortisone. E. A. Kabat, A. Wolf and A. E. Bezer.—p. 265.
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Complement Fixation Tests with Homologous and Heterologous Types of Coxsackie Virus in Man. L. M. Kraft and J. L. Melnick.—p. 297.
Intranuclear Inclusion Bodies in Cultures of Human Tissue Inoculated with Varicella Vesicle Fluid. T. H. Weller and M. B. Stoddard.—p. 311.
Studies on Mode of Action of Aromatic Diamidines on Influenza and Mumps Virus in Tissue Culture. M. D. Eaton, M. E. Perry, C. G. Levenson and I. M. Gocke.—p. 321.

Journal of Investigative Dermatology, Baltimore

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Bacterial Flora of Skin: Factors Influencing Growth of Resident and Transient Organisms. D. M. Pillsbury and G. Rebell.—p. 173.
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Tissue Culture Studies on Human Skin: II. Comparative Effects of Certain Specific Contact Allergens on Sensitized and Non-Sensitized Human Skin. E. T. Everett, C. S. Livingood, C. M. Pomerat and F. Hu.—p. 193.
Relationship Between Hypersensitivity and Immunity to Vaccinia. G. D. Baldridge and A. M. Kligman.—p. 205.
Abnormal Cytology of Epithelial Cells in Pemphigus Vulgaris: Diagnostic Aid. H. Blank and C. F. Burgoon.—p. 213.
Evidence for Occurrence of True Mitosis in Bacteria and Certain Applications. E. D. DeLamater.—p. 225.
*Pathogenesis of Tinea Capitis Due to Microsporum Audouini and Microsporum Canis: I. Gross Observations Following Inoculation of Humans. A. M. Kligman.—p. 231.
Antibiotics and Fungous Infections. M. J. Lipnik, A. M. Kligman and R. Strauss.—p. 247.
Modification of Bennett's Method for Histochemical Demonstration of Free Sulfhydryl Groups in Skin. H. Mescon and P. Flesch.—p. 261.
Local Depilatory Action of Unsaturated Compounds: Effect of Human Sebum on Hair Growth. P. Flesch and S. B. Goldstone.—p. 267.
Histochemical Demonstration of Secretory Activity in Human Eccrine Sweat Glands. W. B. Shelley and H. Mescon.—p. 289.

Pathogenesis of Tinea Capitis.—The data reported in this paper were derived from work carried out at a state institution for congenital mental defectives where tinea capitis was endemic. A large number of persons living under confined circumstances could be inoculated, and the course of the disease minutely studied from its onset. In inoculations of the human scalp with Microsporum audouini and M. canis it was found that some form of minor trauma was a prerequisite for infection. About 60% of children were found to be susceptible to experimental tinea capitis due to M. audouini and M. canis. Although a somewhat smaller percentage of adults could be infected with M. audouini, the disease was trivial in this group. Tinea capitis has four phases: (1) an incubation period, (2) a period of enlargement and spread, (3) a refractory period, and (4) a period of involution. Tinea capitis consists of two distinct types of infection: (1) scalp infection, and (2) hair infection. Either may exist alone, and usually both are present in the early phase of tinea capitis. There are probably many persons with an innate resistance to the disease. In a susceptible person infection does not follow simple contact with the spores, which must be implanted into the stratum corneum. Minor trauma is hence a precondition for infection. The lower incidence of infection among girls is probably due to the fact that their long hair helps prevent the spores reaching the scalp surface. Transmission in the barber shop is possible, considering the minor trauma probably incident to combing and clipping. Boys who rub the backs of their heads against infected theater seats also are subjected to ideal circumstances for acquiring the infection. It might be assumed that the exclusion of infected children from barber shops and theaters would contribute substantially to control of this disease.

Journal Lab. and Clinical Medicine, St. Louis

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Metabolism of Iron: I. Intravenous Iron Tolerance Tests in Normal Subjects and Patients with Hemochromatosis. S. E. Gitlow and M. R. Beyers.—p. 337.
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Studies on Separation of Cell Types in Serosanguinous Fluids, Blood, and Vaginal Fluids by Flotation on Bovine Plasma Albumin. D. W. Fawcett and B. L. Vallee.—p. 354.
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Studies of Cortisone Effects on Inflammatory Response: I. Alterations of Histopathology of Chemically Induced Inflammation. D. M. Spain, N. Molomut and A. Haber.—p. 383.
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Electrolyte Changes Produced by ACTH, Cortisone and DOCA in Cirrhosis of Liver. W. H. Blahd, W. S. Adams, A. Leslie and others.—p. 393.
Estimation of Insulin Sensitivity by Modified Glucose Insulin Tolerance Test. S S. Lazarus and B. W. Volk.—p. 404.
Disappearance of Intravenously Injected Emulsified Fat from Circulation of Patients and Animals. W. A. Johnson, S. Freeman and K. A. Meyer.—p. 414.
Effect of Sweating on Normo-and Hypertensive Subjects. T. B. Gibbons and C. B. Chapman.—p. 420.
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Studies on Technique of Transporting Throat Swabs to Laboratory for Isolation of Pneumococci. D. A. Stevens.—p. 437.
Cold Hemagglutinins: Effect of Storage at —20° C A. E. Feller.—p. 440.
Hemolysis of Red Blood Cells Treated by Bacterial Filtrates in Presence of Serum and Complement. L. A. Rantz, A. Zuckerman and E. Randall.—p. 443.
Further Studies on Nature of Hemorrhagic State in Radiation Injury. D. P. Jackson, E. P. Cronkite, G. V. Leroy and B. Halpern.—p. 449.
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Journal of Thoracic Surgery, St. Louis

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*Simple Excision in Treatment of Pulmonary Tuberculosis. B. J. Ryan, E. M. Medlar and E. S. Welles.—p. 327.
Study to Determine Effect of Prevention of Overdistention of Remaining Lung Tissue on Elevated Right Ventricular Pressures, Following Resection of Lung Tissue in Dogs. B. C. Charbon and W. E. Adams.—p. 341.
Techniques of Endocardial Anastomosis for Circumventing Pulmonic Valve. T. J. Donovan, C. A. Hufnagel and H. H. G. Eastcott.—p. 348.
Fibrosarcoma of Diaphragm: Report of Case. P. D. Crimm and F. L. Kiechle.—p. 360.
Repair of Experimental Tracheal Defects with Fresh and Preserved Homologous Tracheal Grafts. O. G. Davies, J. M. Edmiston and H. J. McCorkle.—p. 367.
Hodgkin's Disease of Lung with Cavitation: Report of Three Cases. L. Efskind and P. Wexels.—p. 377.
Bronchial Adenoma: Case Report. H. H. Bradshaw, L. DeS. Shaffner and W. R. Deaton Jr.—p. 388.
Chondromatous Hamartoma of Lung. M. Rubin and J. Berkman.—p. 393.
Pulmonary Resection and Undecylenic Acid in Systemic Blastomycosis. J. E. T. Hopkins and J. D. Murphy.—p. 409.
Open Closure of Bronchus and Resection of Carina and of Tracheal Wall. V. O. Björk, E. Carlens and C. Crafoord.—p. 419.

Simple Excision for Pulmonary Tuberculosis.—Wedge or subsegmental resection of diseased lung areas was done in 30 patients with pulmonary tuberculosis for removal of tuberculous foci that remained after continuous daily treatment with streptomycin and *p*-aminosalicylic acid for four months or longer. Bilateral resections were done in three of these patients. Of the 30 patients, 4 had minimal pulmonary tuberculosis and none of these had shown tubercle bacilli in repeated cultures of sputum or gastric washings during hospitalization. They were operated on because minimal pulmonary tuberculosis always contains one or more necrotic lesions generally localized in the superior or posterior portions of lobes of lung, and it should be possible to remove these lesions completely by a small local excision. Of the remaining 26 patients, 16 had moderately and 10 had far-advanced pulmonary tuberculosis. None had had positive cultures of sputum or gastric contents for many weeks or months prior to surgical intervention. The amount of lung tissue to be excised, usually with disregard for the segmental anatomy of the lungs, was determined after a complete palpation of the lung had been done considering preservation of a maximum amount of functional lung. The wedge to be resected was outlined, the pleura incised a short distance beyond the disease area, and the area excised en bloc. Sutures were applied to bleeding points and bronchial leaks. Each resected specimen showed necrotic pneumonic lesions, and the cavity-bearing areas removed showed filled-in cavities with patent bronchial communications. Otherwise there were areas of scarred tissue, with extremely little evidence of unhealed non-necrotic pneumonic lesions. Postoperative cultures of sputum or fasting gastric contents were negative for tubercle bacilli. There was little change in the cardiorespiratory reserves postoperatively, as compared to preoperative findings. No postoperative tuberculous complications arose. No complications of any sort occurred in the four patients with minimal disease. As a rule, the postoperative course was benign. One year after the intervention all patients are in excellent physical condition. It is recognized that a lapse of five years may be necessary for final evaluation. The authors do not claim that their approach to the surgical problem of the tuberculous lung will eradicate all of the pulmonary disease, but it is anticipated that the small lesions left behind will cause no further trouble. The immediate results have been encouraging enough to warrant continuation of the program.

Journal of Urology, Baltimore

67:405-564 (April) 1952. **Partial Index**

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Operative Procedures for Stenosis Following Ureterosigmoidostomy. D. St. C. L. Henderson.—p. 479.
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New Concept Concerning Treatment of Extrophy of Bladder. W. H. Boyce and S. A. Vest.—p. 503.
Mucinous Carcinoma of Urachus Invading Bladder. M. S. Wessell, A. B. Kuritz, R. A. Burger and G. W. Reagan.—p. 523.
Postoperative Treatment Following Prostatic Surgery. J. B. Glanton, L. M. Orr and J. C. Hayward.—p. 533.
Clinical Effect of Estrogen Therapy on Metastatic Lesions from Carcinoma of Testis. C. H. deT. Shivers and H. D. Axilrod.—p. 537.
Solusponge as Hemostatic Agent in Urology. J. S. Ritter and H. Bloomberg.—p. 543.
Interesting Bone Changes in Plain Urograms. W. L. Fitzgerald and M. D. Thomas Jr.—p. 547.
Lipid Constituents of Human Male Reproductive Cells as Determined by Sudan Black B. R. L. Brown.—p. 557.

Bilateral Total Adrenalectomy.—Of 9 selected patients with advanced malignant hypertension and 5 with chronic nephritis, 13 were subjected to bilateral total adrenalectomy by the posterior approach, and one patient with nephritis had one adrenal removed as a first stage procedure. The patients operated on had failed to respond to medical treatment and at the same time their disease was considered too far advanced to be benefited by sympathectomy. A salt-free diet, diminution of edema by diuresis, digitalization, and sedation were fundamental preoperative measures. Additional measures for correction of renal insufficiency and electrolyte imbalance were necessary in four patients, external dialysis being used for one. Cortisone in doses of 100 mg. was given intramuscularly the night before operation and repeated the next morning before surgical intervention. During operation 100 to 200 cc. of aqueous adrenal cortical extract and 20 to 50 mg. of phenylephrine (neo-synephrine®) hydrochloride by intravenous drip was given as required for circulatory stability. Cortisone, 50 mg. every six hours, was given intramuscularly during the first 48 postoperative hours and then was gradually reduced to 25 to 37 mg. per day as a maintenance dose. In the late postoperative care salt depletion must be avoided by adding 3 gm. or more of sodium chloride to the diet and/or 1 mg. of desoxycorticosterone acetate per day three to six times a week as necessary to avoid weakness and hypotension. Two patients died of renal failure and two of cardiac complications of their vascular disease. Nine patients are living after complete adrenalectomy and seven of them are definitely improved both subjectively and objectively. One patient previously incapacitated for three years has been working for six months as a hospital orderly and another has resumed his work as an insurance agent. In their preliminary report the authors attribute the improvement to elimination of natural desoxycorticosterone-like factors and their administration thereafter in such a manner as to avoid excessive retention of sodium and water on the one hand and relative hypotension on the other. It has been shown that absence of the adrenal glands today may be compatible with an active existence, owing to available substitution therapy.

Kentucky State Medical Assn. Journal, Bowling Green

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Maine Medical Association Journal, Portland

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Lymphosarcoma of Urinary Bladder. D. F. Marshall and F. M. Dooley. —p. 114.
*Cysts and Degenerative Changes in Menisci of Knee Joint. L. J. McDermott.—p. 116.

Pulmonary Embolism by Amniotic Fluid.—A total of only about 30 pathologically verified cases of pulmonary embolism and obstetric shock caused by amniotic fluid have been reported. The case described here was that of a woman in whom there had been some bleeding coincident with membrane rupture. Less than two hours later, she complained of difficulty in breathing, and then had generalized convulsions, and became cyanotic, hyperpneic and pulseless. Simultaneously there appeared red blood from the mouth, numerous rapidly spreading ecchymoses on the abdomen, chest, back, arms, and legs, and massive submucous and subcutaneous extravasation of blood involving the vaginal walls, perineum, and inner right thigh. All needle punctures oozed constantly. The placenta was delivered intact following normal separation. Despite administration of whole blood and supportive measures, massive pulmonary edema and coma occurred, and the patient died. This case of pulmonary amniotic fluid embolism has some factors in common with other cases, but differs in respect to the postmortem uterine lesions and the local genital symptoms. No evidence of abruptio placentae, placenta previa or accreta, or deep cervical or placental tear was present, but a large tear was found in the uterine endometrium. The shock was definitely anaphylactoid, and the skin was warm and dry. Unlike the patients in most cases, the patient rallied to a considerable extent from the initial shock. In the presence of afibrinogenemia, even massive transfusion (short of exsanguination and replacement) will be of no appreciable benefit unless fibrinogen is available. Since no distinction can be made in vivo between patients with intravascular fibrin clotting mechanisms and patients with amniotic fluid embolism, treatment of these conditions is essentially the same, and consists of measures to overcome shock, to control hemorrhagic manifestations, and to combat pulmonary edema. The availability of blood and fibrinogen is important.

Cysts and Degenerative Changes in Knee Menisci.—McDermott prefers the term menisci to semilunar cartilages, chiefly because it has been observed that these structures do not assume the characteristics of a cartilage until the age of 15 years. He had available for study a group of 50 menisci. Some of these had been removed because of clinically diagnosed cysts and others because of rupture. The remainder of the menisci were removed post mortem from patients with no evidence of disease of the knee joint, or from human embryos. Theories concerning the etiology, nature, and pathogenesis of the cysts as well as their relationship to rupture of the menisci are discussed. The following conclusions are formulated: 1. Cysts of the menisci result from several types of degeneration of the connective tissue. 2. The cysts have no specialized lining. 3. Synovial membrane is not endothelial in nature. 4. Cysts of the meniscus present histologically resemble ganglion cysts and myxomatous intracutaneous cysts. 5. There is no histological resemblance between meniscal cysts and the synovial membrane to suggest relationship between them. 6. Some of the so-called ruptures of the menisci may well be spontaneous in nature due to a degenerative lesion of these structures. 7. Cysts of the menisci may recur if the entire meniscus is not removed. 8. The relatively greater exposure of the internal meniscus to trauma may explain the greater frequency of rupture of this structure and the greater incidence of cysts in the external meniscus. 9. Trauma has little or no etiological importance in production of degenerative changes and meniscal cysts. 10. The etiology of the degenerative process responsible for the production of cysts of the menisci remains obscure.

Medical Annals of District of Columbia, Washington

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Congenital Obstruction of Urinary Tract. M. Campbell.—p. 123.
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Management of Abnormal and Excessive Uterine Bleeding. R. Bieren. —p. 187.
Pathologic Anatomy of Degenerative Shoulder Lesions. J. S. Neviaser. —p. 194.
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Michigan State Medical Society Journal, Lansing

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Missouri State Medical Assn. Journal, St. Louis

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Induced Hypotension in Surgery of Intracranial Vascular Anomalies: Report of 2 Cases. R. D. Woolsey and H. J. Freiheit.—p. 316.
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New England Journal of Medicine, Boston

246:391-434 (March 13) 1952

Carcinoma of Right Colon. B. P. Colcock.—p. 391.
Acute Effects of Intravenous Administration of Preparation of Veratrum Viride in Patients with Severe Forms of Hypertensive Disease. N. S. Stearns and L. B. Ellis.—p. 397.
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246:435-476 (March 20) 1952

Multiple-Balloon-Kymograph Recording of Effect of Banthine, Belladonna and Placebos on Upper-Intestinal Motility. W. P. Chapman, A. B. French, P. S. Hoffman and C. M. Jones.—p. 435.
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Effect of Volatile 1-Cyclohexyl-2-Methylaminopropane ("Benzedrex" Inhaler) on Patients with Coronary Arteriosclerosis. J. B. Burnett and S. M. Gundersen.—p. 449.
Diseases of Thyroid Gland. O. Cope.—p. 451.

246:477-522 (March 27) 1952

*Splenectomy in Leukemia and Leukosarcoma. J. H. Fisher, C. S. Welch and W. Dameshek.—p. 477.
Concomitant Brucellosis and Tuberculosis: Report of Case. J. T. Sharp, H. J. Wheelwright and P. O. Nice.—p. 485.
Nonvisualized Gall Bladder. F. Martin and A. G. Massimiano.—p. 488.
Influence of Carbonated Water on Gastric Emptying. G. Lolli, L. A. Greenberg and D. Lester.—p. 490.
Some Practical Aspects of Cancer Prevention. E. L. Wynder.—p. 492.
Van Riebeeck and Cape Town. H. R. Viets.—p. 503.

Splenectomy in Leukemia and Leukosarcoma.—Eleven of the 18 patients in this series had splenectomy for some type of leukemia, and 7 were found to have leukosarcoma. The decision to remove the spleen was made only after long observation. Nearly all patients had had roentgen therapy over the spleen to the limit of toleration. In leukemia, nitrogen mustard, urethane, triethylenemelamine, adrenocorticotropic hormone, or cortisone had been used. Hypersplenism, as manifested by cytopenic disturbances alone, was present in seven patients; five had severe hemolytic anemia; four had hemolytic anemia and cytopenic hypersplenism; and in two the spleen was removed because of its large size and painful symptoms. Size alone is not a sufficient indication for splenectomy. For the best results of splenectomy in hypersplenism the bone marrow should be hyperplastic. If the patient has required frequent transfusions to control a hemolytic anemia or severe cytopenia, splenectomy may be indicated, but a markedly diminished number of megakaryocytes or nucleated red blood cells in the bone marrow contraindicates splenectomy. Hemolytic anemia or the cytopenic disturbance was controlled for a reasonable period of time in 11 of the 18 patients, 5 patients were not benefited, and 2 died in hospital after operation. In one man, who had been observed for 18 months before operation, severe hemolytic anemia was the indication for splenectomy. Follow-up studies of the peripheral blood revealed that there was complete remission of the hemolytic anemia until death resulted from coronary occlusion. In a patient with pancytopenia, hemolytic anemia, and lymphosarcoma complete remission of the pancytopenia and hemolytic anemia persisted for two years. Another patient who had hemolytic anemia and lymphocytic leukemia responded temporarily to splenectomy. This patient was given a course of corticotropin prior to splenectomy. Other investigators recently reported striking improvement of the hemolytic process following corticotropin therapy in cases of acquired hemolytic anemia. Such treatment should probably be used in every case prior to splenectomy, with operative intervention reserved for patients who fail to respond to medical therapy. While it has been suggested that splenectomy should be performed early in the course of leukemia to obtain the best palliation, these authors do not believe that splenectomy is indicated unless the spleen is extremely bulky and is causing pain. They do not advocate the indiscriminate use of splenectomy, but advise that it should be confined to a small number of specially selected cases.

Northwest Medicine, Seattle

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Proc. Staff Meet., Mayo Clinic, Rochester, Minn.

27:137-168 (April 9) 1952

*Mortality and Survival in Cancer of Stomach: Statistical Summary of Experience of Mayo Clinic. J. Berkson, W. Walters, H. K. Gray and J. T. Priestley —p. 137.
Problem of Gastric Cancer from Viewpoint of Internist. M. W. Comfort. —p. 151.
Advances in Surgical Treatment of Cancer of Stomach. W. Walters, H. K. Gray and J. T. Priestley.—p. 156.
Ileostomy Receptacle for Bed Patients. J J. Berens and J. deJ. Pemberton.—p. 161.
Hypersensitivity to Heparin: Report of 3 Cases. E. W. Hauch, H. M. Carryer and D. R. Mathieson.—p. 163.

Mortality and Survival in Cancer of Stomach.—During the years from 1907 to 1949, 9,620 patients underwent operation for malignant neoplasm of the stomach at the Mayo Clinic, and it is estimated that there were in addition about 3,500 seen but not operated on. Of the total of 9,620 operations, 4,597 (47.8%) were resections, 1,327 (13.8%) were palliative operations, and 3,696 (38.4%) were exploratory laparotomies, with respective average hospital mortality rates of 13.3, 12.3, and 3.8%. The survival rates following operation are analyzed with relation to metastases, to grade of malignancy, to gastric acidity, and to sex. In order to ascertain whether the outlook for the patient with gastric cancer has improved, the records of the period 1907-1916 are compared with those of the period 1940-1949. The five year survival rate for the recent period is 35%, whereas for the earlier period it is 29%. The 10 year survival rate for the recent period is 27%, while for the earlier period it is 22%. There has also been improvement in the operability rate, resectability rate, and hospital mortality rate. In the early period, only 5 of 100 patients with gastric malignant lesions could be expected to be living five years later; in the later period the expected number living five years after diagnosis has increased to 14. However, this figure conveys a false idea of the efficacy of surgical therapy, because in the later group 20 of the 86 patients who died within 5 years were found inoperable when they were first examined, and in 36 more laparotomy revealed a lesion that was not resectable. Thus 56 of 86 of the deaths are accounted for by the patient's being seen too late for effective surgical intervention. The other 30 patients can perhaps be considered the true surgical loss, since in spite of surgical effort they do not survive more than five years. If the aforementioned 56 patients should be seen by the surgeon when resection was possible, then without any further improvement in the surgical results, 18 survivors would be added to the present 14, more than doubling the number salvaged out of the original 100. Whether education or better diagnostic methods will accomplish this remains a problem for the future.

Rhode Island Medical Journal, Providence

35:121-176 (March) 1952

Acne Conglobata Treated with Aureomycin: Case Report. B. L. Schiff and A. B. Kern.—p. 137.
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Rocky Mountain Medical Journal, Denver

49:241-314 (March) 1952

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Municipal Water Supplies of Utah. M. E. Murphy.—p. 268.
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49:315-396 (April) 1952

SYMPOSIUM ON LUPUS ERYTHEMATOSUS

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Clinical Evaluation of Banthine. F. B. McGlone.—p. 350.
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Q Fever in Colorado. D. R. Barglow.—p. 357.

Southern Medical Journal, Birmingham, Ala.

45:287-380 (April) 1952

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Significance of Prepyloric Lesion. C. M. Caravati, J. M. MacMillan and T. R. Hood.—p. 319.
Gallstone Obstruction of Intestine. R. L. Sanders and R. M. Pool. —p. 328.
Recent Advances in Surgery of Infancy. J. W. Duckett.—p. 333.
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*Appraisal of Injection Treatment of Internal Hemorrhoids. J. H. Dodson and M. H. Dodson.—p. 347.
Antifungal Antibiotics of Clinical Significance. W. E. Wooldridge. —p. 351.
Results of Use of Chloramphenicol and Other Antibiotics in Proctologic Practice. E. Boling and H. Finch.—p. 354.
Conservative Treatment of Degenerative Arthritis of Knee. O. O. Selke Jr.—p. 358.
Observations on Diabetic Therapy with Special Reference to Insulin Allergy. A. A. Herold Sr.—p. 360.
Reflections of a Professor Emeritus. G. P. Lingenfelter.—p. 364.
Organization and Objectives of the Civil Defense Blood Program. J. B. Alsever.—p. 366.
What the Physician Should Know About Rabies. L. E. Starr.—p. 370.

Injection Treatment of Internal Hemorrhoids.—The Dodsons evaluate the results they obtained by injection treatment in 697 cases of hemorrhoids. Of the 697 patients, 443 (63.3%) had recurrences within five years, and the authors suspect that the actual recurrence rate was even higher. Of the 201 patients classified as having first degree internal hemorrhoids (bleeding only), 76 (38%) required re-treatment, 62 being subjected to hemorrhoidectomy and 14 to reinjection. Of the 176 patients with second degree hemorrhoids (bleeding and protrusion that is spontaneously reduced), exactly half required re-treatment. Of the 289 with third degree internal hemorrhoids, 140 have had hemorrhoidectomies, 63 have had re-treatment by injection, and 35 with recurrent symptoms refused further treatment but were told that surgical intervention would be necessary for cure. All 31 of the patients with so-called fourth degree lesions have had recurrences. Thus curability by sclerotherapy varies in inverse proportion to the degree of prolapse of the hemorrhoids. Best results of injection treatment are obtained in uncomplicated first degree internal hemorrhoids. Second degree hemorrhoids are sometimes adequately treated by injections, but in third degree and fourth degree cases only temporary and usually incomplete results can be expected. At best, the recurrence rate with injections far exceeds that of careful surgical treatment. For cases in which surgical treatment is contraindicated or refused, sclerotherapy is a useful palliative measure.

U. S. Armed Forces Med. J., Washington, D. C.

3:503-656 (April) 1952. **Partial Index**

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*Observations on Essentially Normal Young Men Treated with ACTH and Cortisone: III. Side Effects During Therapy. S. B. Appel, L. A. Fulton and K. D. Orr.—p. 531.
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Role of Physical Medicine in Management of Upper Extremity Amputee. R. C. Psaki and A. F. Mastellone.—p. 627.
Role of Psychiatry in Naval Retraining. J. M. Murphy and J. D. Grant. —p. 631.
Approach to Venereal Disease Control. E. F. Diamond.—p. 647.
Congenital Unilateral Superior Dislocation of Patella. S. L. Cohen and E. B. Reilly.—p. 655.

Normal Young Men Treated with ACTH and Cortisone.—Side-effects of corticotropin (ACTH) and cortisone were studied in young combat veterans with frostbite injuries that healed with medical care in all but one instance. Corticotropin (20 mg. in 1 liter of 5% dextrose in water or sodium chloride solution or in isotonic sodium chloride solution alone) was given to 13 patients daily over a six hour period for 31 consecutive days. Cortisone was given to 22 patients by mouth as follows: 400 mg. (100 mg. every 6 hours) for 1 day; 300 mg. (75 mg. every 6 hours) on the second day; 200 mg. (50 mg. every 6 hours) until the 12th day at which time 200 mg. (100 mg. at 8 a. m. and 8 p. m.) was given daily through the 31st day. There was a control group of 26 patients. Four patients (two in each hormone therapy group) were placed on moderate fluid and candy intake; otherwise, diet was unrestricted and contained excessive quantities of candy bars and soft drinks. There was no significant difference between the groups in regard to personal or familial illnesses, length of time in combat, or previous combat wounds. The side-effects noted in order of frequency were moon face, glucose intolerance, hypertension, acne, hirsutism, edema, and psychic changes. No untoward reactions were observed during intravenous administration of corticotropin, whether it was given slowly or rapidly. The effects of cortisone given orally did not differ from those obtained with intramuscular administration; no untoward gastrointestinal manifestations were noted. At least one side-effect was found in 83% of the group receiving hormone therapy; only in one case was the side-effect severe enough to force discontinuance of the treatment. Side-effects occurred in 43 patients who were given cortisone and in 17 who were given corticotropin. All the side-effects disappeared after the hormone treatment was discontinued; their common occurrence in essentially normal young men indicates pharmacologic effects, with moderate individual variation depending on dosage and duration of therapy rather than significant variation in end-organ response.

Wisconsin Medical Journal, Madison

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Diagnostic and Therapeutic Significance of Nasopharynx. W. H. Theobald.—p. 262.
Coarctation of Aorta. J. W. Gale.—p. 265.
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*Preliminary Report on Intravenous Use of Corticotropin (ACTH) in Treatment of Shock. T. Armstrong and R. Pozo Jiménez.—p. 168.
Anticoagulant Treatment in Thrombophlebitis. W. G. Anylan, F. H. Campbell and W. C. Shingleton.—p. 173.

ACTH Intravenously in Treatment of Shock.—Corticotropin (ACTH) administered intravenously in three cases of apparently irreversible shock produced such rapid responses that they are made the subject of a preliminary report. The adrenocortical hormones are essential factors in the normal balance of organic functions, and enable the body to meet and overcome stress. Sudden exhaustion of the adrenocortical steroids and loss of their action on the vasomotor tone may be important in the onset of shock. Intravenous administration is apparently a safe, effective, and economical method of giving corticotropin, and has been used in various conditions. Corticotropin and cortisone have heretofore been used for shock following burns and in two cases of the Waterhouse-Friedrichsen syndrome. Intravenous use of corticotropin for serious shock is a new method, and the rapidity of the response obtained indicates that corticotropin has an immediate and selective action on vasomotor tone. The patients treated, all of whom were undergoing serious surgical procedures, had received the usual remedies for shock without showing any favorable response; all three were moribund. Administration of 40 mg. of corticotropin in 500 cc. of isotonic sodium chloride solution at a rapid rate (120 drops a minute) was quickly followed by return of the pulse, which had been imperceptible, restoration of arterial pressure, and resumption of normal breathing. All three patients recovered and were discharged from the hospital. No permanent neurological sequelae were noted; but in one case an excessive dose of corticotropin, made necessary by recurring signs of shock, led to temporary hypercorticism that was rapidly dissipated when administration of the drug was discontinued.

British Heart Journal, London

14:153-308 (April) 1952

Circulatory Changes in Severe Pulmonary Emphysema. J. P. D. Mounsey, L. W. Ritzmann, N. J. Selverstone and others.—p. 153.
Electrocardiogram in Normal Children and in Children with Right Ventricular Hypertrophy. J. F. Goodwin.—p. 173.
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Pulmonary and Systemic Circulations in Congenital Heart Disease. D. C. Deuchar and R. Knebel.—p. 225.
*Acute Cardiac Infarction Without Pain. C. Papp.—p. 250.
Graphic Configuration of Apical Diastolic Murmurs. B. G. Wells.—p. 261.
Electronic Phonocardiograph Employing Double-Beam Cathode-Ray Oscillograph as Recording Device. F. W. Campbell, A. W. Sloan and A. M. Andrew.—p. 271.

Acute Cardiac Infarction Without Pain.—Acute cardiac infarction without pain is rare. In 150 cases in which coronary thrombosis with electrocardiographic signs of acute cardiac ischemia was diagnosed, Papp found seven patients in whom pain was completely absent, and in an eighth patient slight pain was present for a short time only. Three of the eight patients had congestive heart failure with cardiac infarction. Two died and at necropsy were found to have extensive arteriosclerotic heart disease with occlusion of more than one main coronary artery. The third, who survived, had a slight cardiac infarction during an attack of cardiac asthma. Acute paroxysmal dyspnea was the main symptom in all; this may overshadow coronary pain. Five patients had complete heart block during the attack. Four of them had no pain; in the fifth, who had intermittent heart block, pain appeared when normal rhythm was restored. Complete heart block was recorded in four, and lasted from one and a half hours to one week or more. The electrocardiogram indicated posterior infarction in three, anteroseptal infarction with right bundle branch block and later left bundle branch block in one, and anteroposterior infarction in one. All patients recovered; one had a small anterolateral infarction accompanied by pain, but not by heart block, 18 years later, from which he also recovered. Faintness with a pulse rate of 30 to 40 was the main clinical symptom, but complete loss of consciousness occurred only in one patient. These attacks differ from classical Stokes-Adams attacks in that ventricular standstill, ventricular tachycardia, and therefore complete loss of consciousness may be absent. Sudden fall of pulse rate accompanied by fall of blood pressure, as it occurs in complete heart block during acute cardiac infarction, may induce incomplete cerebral anoxemia; this causes faintness without loss of consciousness, but with loss of sense of pain. Prognosis is serious in cardiac infarction without pain and complicating heart failure; with complete heart block it may be less serious than is generally accepted, since transient block may be a sign of only temporary ischemia of the conducting system.

British Journal of Ophthalmology, London

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British Medical Journal, London

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Diabetes Mellitus in Surgical Patients. C. F. Rolland.—p. 737.
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Infantile Cortical Hyperostosis. W. J. Matheson and M. Markham.—p. 742.
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Anaemia of Hypopituitarism. V. K. Summers.—p. 787.
*Survey of Pulmonary Embolism in a General Hospital. D. S. Short.—p. 790.
*Deafness with Use of Dihydrostreptomycin in Tuberculous Meningitis. J. T. Naismith.—p. 796.
Growth of Biparietal Diameter of Foetal Head in Last Weeks of Pregnancy. I. Macdonald.—p. 798.
Poliomyelitis in Mother and Newborn Infant. G. A. Wright and T. K. Owen.—p. 800.

Incidence of Pulmonary Embolism.—During the past decade it has been realized that most pulmonary emboli are not massive and fatal, and that they concern internists at least as much as surgeons and obstetricians. Indeed, the present study suggests that thromboembolic disease of the lung may have become the commonest of serious acute pulmonary lesions seen in general hospital practice. Pulmonary thromboembolism was diagnosed in 120 patients in 30 months, of whom 113 were in the wards of a general hospital. The incidence of pulmonary thromboembolism in this hospital was higher than that of lobar pneumonia. Pulmonary embolism was directly responsible for 30 deaths, and pulmonary infarction was an important contributory factor in 13 more. Seventy of the 120 cases were classed as medical, and four-fifths of the patients had heart disease. There were three instances (one fatal) of pulmonary embolism appearing without warning in apparently healthy subjects. In 4 of the 30 surgical cases (8 fatal) pulmonary embolism occurred when the patient first got out of bed. Among the 20 obstetrical cases (2 fatal) pulmonary embolism was nearly three times as common in those over the age of 30 as in those under 30. Pulmonary embolism was seen only once in the absence of obvious femoral thrombosis or some complication of pregnancy or delivery. Pleuritic pain and hemoptysis are the two most striking symptoms of pulmonary infarction. Pleuritic pain was noted in 65% of all cases, and was present in 90% of the surgical and obstetrical cases. Hemoptysis was noted in less than 40% of all cases. The physical signs in the chest of pulmonary infarction depend on three factors, namely, consolidation, pleurisy, and elevation of the diaphragm. Signs of pleural effusion were present in one-fifth of cases. The fluid was blood-stained in 8 of 17 cases in which it was inspected. Jaundice was observed in almost one-third of the cases of heart disease complicated by pulmonary infarction. A paroxysm of auricular fibrillation was precipitated by pulmonary infarction in five cases. In several patients with congestive heart failure the symptoms were slight, and the presence of pulmonary infarction was overlooked. In postoperative and puerperal patients the condition was often misdiagnosed as pleurisy, pulmonary collapse, or pneumonia.

Deafness from Dihydrostreptomycin in Tuberculous Meningitis.—The results obtained in 26 cases in which streptomycin was administered were compared with those in 51 cases in which dihydrostreptomycin was given. Two-thirds of the patients treated with dihydrostreptomycin recovered, and one-half of those treated with streptomycin. While half of the survivors treated with dihydrostreptomycin were deaf, only 1 of 13 survivors in the streptomycin series had impaired hearing.

Deutsche medizinische Wochenschrift, Stuttgart

77:477-516 (April 18) 1952. Partial Index

Surgery of the Heart. E. K. Frey.—p. 477.
So-Called Arteriomesenterial Duodenal Closure. R. Geissendörfer.—p. 483.
Functional Problems in Cardiac and Pulmonary Disorders and Remarks on Surgery of the Heart and of Lung. H. W. Knipping.—p. 487.
*Surgical and Cardiologic Problems and Surgical Treatment of Stenosed Mitral Valves. A. Lezius and E. Gadermann.—p. 491.
Exclusion Anastomosis in Inoperable Carcinoma of Esophagus and Cardia. A. Gütgemann.—p. 497.
*Splenectomy in Therapy of Subacute Bacterial Endocarditis. K. Fellinger and W. Weissel.—p. 499.

Surgical Treatment of Mitral Stenosis.—The aim of the surgical treatment of mitral stenosis is not only to overcome the stenosis but also to preserve the closure function of the mitral valves. In the mild form of endocarditis, the stenosed orifice is usually a narrow oval, the classical button-hole stenosis, whereas in the severe form of endocarditis, the orifice is not oval, but round, irregular or triangular. Surgical correction of mitral stenosis is effected by splitting the margins of the coalescent cusps in the line of the former axis, that is, in the commissures. The authors mention Bailey, Brock, Harken, and O'Neill as having described techniques of commissurotomies or valvulotomies, and say that splitting is done most effectively with the finger equipped with a special knife, the finger being the basic instrument. Splitting of the commissures not only counteracts the stenosis but also produces functioning valves capable of closure. Injury to the valves outside of the commissures must be carefully avoided, because surgically produced defects in the valves may impair their closure. After commenting on several other problems, particularly the danger of postoperative acute mitral insufficiency, which may be brought on by the reduced tolerance of the left ventricle, the authors describe observations on seven patients in whom they performed operations for mitral stenosis. The patients ranged in age from 18 to 45 years. All patients experienced objective improvement, including those who had had decompensation for a long time and who had the corresponding anatomic and clinical changes. Severe myocardial lesions, even those in the region of the overloaded, hypertrophied and dilated right ventricle, and absolute arrhythmia after embolism, are not contraindications to the operation. It is essential, however, that no florid endocarditic process exists.

Splenectomy in Subacute Bacterial Endocarditis.—Although splenectomy was used in treatment of subacute bacterial endocarditis long before effective antibacterial therapy by means of antibiotics had become possible, now it is resorted to occasionally as an additional measure and in cases in which the antibiotics prove ineffective. In the 11 cases analyzed in this paper, splenectomy was done after treatment with antibiotics had failed. Clinical cure followed splenectomy in three of these cases, and in two others cure followed later, the fever subsiding only after a course of streptomycin, which had not been tried before splenectomy. The six other patients died despite splenectomy, but in one of these cure of the endocarditis could be proved by autopsy. There was no primary surgical mortality in these cases. Thromboembolic complications were not increased by splenectomy; at any rate, in the cases described here, the increase in thrombocytes that followed splenectomy did not result in thromboembolism. Even renal insufficiency is not an absolute contraindication to splenectomy. The time at which splenectomy is done seems important for the success of this measure, operation having been done in the successful cases during or after the first attack or relapse. However, the patients in whom splenectomy had proved successful had also received more penicillin and streptomycin. The authors make some suggestions on the possible mode of action of splenectomy, but complete clarification will require further observation and investigation.

77:517-572 (April 25) 1952. Partial Index

Kidney and Hypertension. E. Randerath and A. Bohle.—p. 517.
Prognosis of Acute Diffuse Glomerulonephritis. H. Sarre and H. Mahr.—p. 522.
Humoral Reactions of Blood in Tuberculous Patients. P. Klee, H. Hörlein and K. Jahnke.—p. 525.
*Recovery from Tuberculous Meningitis with Combined Treatment with Streptomycin and *p*-Aminosalicylic Acid in 90% of 36 Cases. W. Löffler, S. Moeschlin and H. Zollikofer.—p. 542.
*Behavior of Intestine Following Treatment with Chloromycetin. F. Sandmann.—p. 557.

Recovery from Tuberculous Meningitis with Chemotherapy.—Combined treatment with streptomycin and *p*-aminosalicylic acid was employed in 23 male and 13 female patients between the ages of 6 and 54 with tuberculous meningitis. Streptomycin was given intramuscularly in doses of 0.5 gm. twice a day for two months and dihydrostreptomycin, 0.5 gm. daily, for an additional four months. In addition streptomycin was given intraspinally in doses of 100 mg. daily the first week, 50 mg. daily the second to the fourth week, and then 50 mg. three times a week for one month, two times a week for three months, and once a week for two additional months. *p*-Aminosalicylic acid was given by intravenous drip in doses of 40 gm. daily the first week, 30 gm. daily the second to the eighth week, and 12 to 14 gm. intravenously five days a week and orally two days a week for four months. Of the 36 patients who were followed for 3½ to 38½ months, 31 were free of symptoms, 2 were still under treatment, and 3 had died of meningitis. Of the three patients who died, treatment was not started until the 20th day in one, and was discontinued too early in the other two at their own request. Of the 36 patients, 9 had associated miliary tuberculosis and 7 of them recovered. Complete blocking of cerebrospinal fluid, which had been observed frequently in patients treated with streptomycin alone, did not occur in any of the patients, and partial blocking occurred in only one patient in whom combined treatment was started on the 15th day. Bilateral impairment of the vestibular nerve occurred in 12 patients, and severe impairment of the eighth cranial nerve in 3. Of the 34 patients followed for one to three years, 3 died and 31 recovered, an average recovery rate of 90%, the best recovery rate so far recorded for tuberculous meningitis in patients followed for long periods. If one considers only the 23 patients in whom the presence of tubercle bacilli in the cerebrospinal fluid was established by culture, animal experiment, or direct detection, there were only three fatal cases after a one to three years' follow-up. Among the patients who recovered from meningitis was one with bilateral tuberculosis of the kidneys who no longer had symptoms and whose working capacity was restored. Complete recovery also resulted from 14 months' treatment with a total dose of 3,724 gm. of *p*-aminosalicylic acid intravenously and 1,737 gm. orally in an apparently hopeless case with paralysis of bladder and rectum and paraplegia. At present *p*-aminosalicylic acid administered by intravenous drip and combined with streptomycin is to be considered the method of choice for the treatment of tuberculous meningitis, miliary tuberculosis, and other severe tuberculous conditions.

Intestinal Upset After Treatment with Chloramphenicol.—Of 21 nursing infants between the ages of two and six months with pertussis treated effectively with chloramphenicol (chloromycetin®), seven had intestinal upset within 14 days after the discontinuation of therapy. Reduction of the bacterial flora in the intestines caused by the drug may result in dyspeptic disturbances, with recurrent increase of the bacterial flora after discontinuation of therapy. To 37 nursing infants with pertussis treated with chloramphenicol, 1 tablet of living Lactobacillus acidophilus was given three to five times immediately after administration of chloramphenicol was discontinued, and in addition up to 7% beta lactose was added to the formula. The administration of living lactic acid bacteria and lactose is recommended for prevention of intestinal disturbances after administration of chloramphenicol.

East African Medical Journal, Nairobi

29:41-78 (Feb.) 1952

Treatment of Urinary Bilharzia. W. L. Barton.—p. 41.
Glaucoma. A. J. Boase.—p. 45.
Removal of Foreign Bodies by Oesophagotomy. H. T. Laycock.—p. 53.
Prosthesis. N. R. E. Fendall.—p. 55.
Fixation of Fracture of Mandible. D. Barker.—p. 57.
*Report on Case of Intermittent Claudication Treated with Hexamethonium Bromide. C. H. Gurd.—p. 58.

Hexamethonium Bromide in Intermittent Claudication.—The patient described had had several operations on the kidneys. Following the first, bilateral femoral thrombosis developed. On examination the feet were cold and the pulse of the posterior tibial, popliteal, and dorsalis pedis arteries could not be felt. Because of the risk and doubtful benefit of operation a chemical sympathectomy with hexamethonium bromide was done, starting with a small dose with gradual increments until the patient was taking 0.5 gm. three times a day. The feet became less waxy and took on a rosy pink color; no pulsation, however, could be felt. The blood pressure fell about 20 mm. in each arm. The fact that hexamethonium bromide causes vasodilatation in the lower extremity appears to favor it in the treatment of this disease.

Fortschr. Röntgenstrahlen & Röntgenpraxis, Stuttgart

76:431-566 (April) 1952. **Partial Index**

Pneumonoconiotic Changes in Graphite Workers. H. Müller.—p. 452.
*Roentgenologic Aspects of Primary Cancer of Pleura. W. Pfeifer and A. Weiss.—p. 460.
Roentgenologic Differential Diagnosis of Tumors of Duodenal Bulb. R. Bauer and H. Hartweg.—p. 468.
*Etiology of Prolapse of Gastric Mucosa in the Pylorus and Duodenal Bulb. H. Etter.—p. 485.
*Roentgenologic Symptoms of Digestive Tract Following Vagotomy. Z. Zsebök.—p. 489.
Allergy of Small Intestine. P. Buffard and L. Crozet.—p. 497.
Demonstration of Transverse Layers of the Heart. A. Vallebona.—p. 508.
Changes in Shape of Ventricle During Cardiac Function. K. Heckmann.—p. 513.
Pathologic Forms of Pulsation of Ventricles. K. Heckmann.—p. 518.
Changes in Choroid Plexus During and After Toxoplasma Encephalitis. J. Schoeps.—p. 528.

Roentgen Appearance of Primary Pleural Cancer.—Primary pleural cancer includes endothelioma, sarcoma, and carcinoma. Of the 12 primary pleural cancers observed in three years by Pfeifer and Weiss, 11 were histologically verified while the patient was alive and in the 12th the growth was morphologically identified at necropsy. The authors employ the following diagnostic procedures: (1) fluoroscopy of the thorax; (2) withdrawal of the pleural exudate by puncture; (3) induction of a diagnostic pneumothorax; (4) a "second" fluoroscopic examination in two planes of exposure; (5) thoracoscopy and biopsy through the thoracoscope from the costal and visceral pleura or, if thoracoscopy proves impossible, exploratory thoracotomy with biopsy from both pleuras. They were able to carry out these procedures without complications in all 11 patients. The roentgenogram is not pathognomonic for primary cancer of the pleura, but the following findings suggest pleural cancer: (1) thickening of visceral and costal pleuras with nodulated or granulated outline, (2) absence of displacement of heart or mediastinum toward the healthy side; in advanced cases the diseased side may show shrinkage; (3) rigidity of the thorax on the diseased side and absence of mediastinal shift during respiration, even after a pneumothorax has been made; (4) tumor nodules the size of a cherry or plum on the thickened pleura; and (5) erosions of the ribs at the level of the pleural thickening.

Etiology of Prolapse of Gastric Mucosa.—Etter discusses the roentgenologic aspects of prolapse of the gastric mucosa into the pylorus and the duodenal bulb and describes a case in which the prolapse was unusually large. It was necessary to perform gastric resection because of increasing pyloric stenosis. Examination of the surgical specimen disclosed lipomatosis of the submucous coat of the antrum, which had resulted in extensive shifting of the mucosa in relation to the muscular coat and thereby produced the mucosal prolapse.

Roentgenologic Signs in Digestive Tract After Vagotomy.—Of 110 patients who had been subjected to vagotomy, 44 were reexamined roentgenologically from 18 to 24 months later. Roentgenoscopy was done first when the stomach was empty, and then again after a contrast medium had been introduced. Severe morphologic and functional changes were demonstrable throughout the digestive tract. Severe hypertrophic gastritis, disturbances in tonus and motility of the small intestine, and dyskinesia of the colon were the most important disturbances observed in patients following vagotomy. The author comments on the nature and possible etiology of these signs on the basis of his own observations and those reported in the literature.

Lancet, London

1:729-774 (April 12) 1952

Pathology and Treatment of Portal Hypertension. R. M. Walker.—p. 729.
Incompatible Transfusions: Their Causes and Prevention. G. Discombe.—p. 734.
*Effect of Adrenocorticotropic Hormone on Survival of Homografts. W. J. H. Butterfield, A. M. Williams and E. I. Evans.—p. 737.
Mysoline: New Drug in Treatment of Epilepsy. R. Handley and A. S. R. Stewart.—p. 742.
*Hypoplasia and Aplasia of Vagina Treated with Oestrogenic Hormone Implants. H. Ripman.—p. 744.
Pulsus Paradoxus. A. C. Dornhorst, P. Howard and G. L. Leathart.—p. 746.
Bone-Marrow Cultures of Koch's Bacillus. C. Choremis and S. Pantazis.—p. 748.
Sternal Osteomyelitis. D. Vellacott.—p. 749.

ACTH and Survival of Homografts.—Homografts in man and animals do not survive permanently because the host forms antibodies to the foreign proteins they contain. Since it was found that the use of corticotropin (ACTH) and cortisone alters the antigen-antibody relationships, the authors studied the effect of corticotropin on the survival of homografts in one man, one woman, and one girl with third degree burns of the trunk and thighs. None had had corticotropin or adrenocortical hormone therapy previously, and all had good adrenocortical reserves. When autografts and homografts were implanted, 100 mg. and 50 mg. of corticotropin were given daily to the adult patients and the child, respectively. Autografts and homografts took well, but the former grew and survived, whereas the latter degenerated in the woman after 11 weeks, and in the child after 51 days. In the man, who died, some of the homografts had died before his death. It is believed that, had he lived, the other homografts would not have survived because sections taken from them after death showed a very thin epithelium and absence of well-formed rete pegs. The length of survival of the homografts seemed to be related to the intensity of adrenocortical response. Corticotropin did not prevent bacterial invasion and clinical signs of infection, nor did it increase the percentage take of delayed autografts. High granulations grew over the burned areas. Side-effects attributable to corticotropin were moon face, mental disorientation, glycosuria, and, in the two adults, a diabetic state. Corticotropin prolonged but did not perpetuate the survival of homografts on deep flame burns. Early skin closure is, therefore, as urgent now as it was before the discovery of this hormone.

Estrogenic Hormone Implants in Vaginal Hypoplasia and Aplasia.—Estrogenic hormones were implanted in a 21-year-old married woman with severe vaginal hypoplasia and in a 24-year-old married woman with congenital vaginal aplasia. The first patient was actually a female castrate, her right ovary having been removed when she was three years old because of a cyst, and there was evidence of ovarian deficiency. Eight 25 mg. pellets of estradiol implanted subcutaneously in the thigh brought about improvement that became more pronounced after three 100 mg. pellets of stilbestrol were also implanted subcutaneously in the thigh 11 months later. In a follow-up three years later, sexual intercourse was described as satisfactory, and the husband, who prior to the treatment had considered seeking an annulment of the marriage, regarded their relationship as normal. The second patient, who presumably had no ovarian tissue, was almost certainly a male pseudohermaphrodite. Three 100 mg. pellets of estradiol, and six months later six 50 mg. pellets of estradiol were implanted subcutaneously in the thigh of the patient. The improvement was pronounced, and during a follow-up nine months later she reported that sexual relations were being accomplished with ease. It appears that implants of estrogenic hormones may be valuable therapy for patients with congenital vaginal aplasia, since many of them are deficient in ovarian function. Moreover, with this treatment, hospitalization, surgical intervention, and application of instruments to the genitalia are avoided.

Lyon Chirurgical

47:257-384 (April) 1952. **Partial Index**

*Hypertension Associated with or Due to Tuberculosis of Adrenal Glands. R. Leriche.—p. 257.
Treatment of So-Called Central Pain. P. Wertheimer.—p. 260.
*Compression of Common Bile Duct Due to Chronic Pancreatitis; Diagnosis and Treatment. P. Mallet-Guy, L. Eicholz and R. Almasque Dedeu.—p. 279.
Surgical Aspects of Silicotuberculosis. A. Bonniot.—p. 305.

Hypertension and Tuberculosis of Adrenals.—Left adrenalectomy combined with bilateral subdiaphragmatic sympathectomy was performed on a 37-year-old woman with essential hypertension. The blood pressure was restored to normal. The patient resumed her work and was in good health eight months after the surgical intervention. Microscopic examination of the removed adrenal gland revealed pronounced hyperplasia of the adrenal medulla associated with disseminated caseous nodular tubercles. These findings suggested that the patients' hypertension was conditioned by the medullary hyperplasia, which resulted from the tuberculous infection. While tuberculosis commonly produces hypocorticalism, it is rather exceptional for it to result in hypertension, demonstrating that there may be other manifestations of tuberculosis of the adrenal gland than Addison's disease.

Common Bile Duct Compression Due to Chronic Pancreatitis.—In the course of 1,575 surgical interventions on the biliary tract performed under radiomanometric control during a 10 year period 36 cases of common bile duct compression due to chronic pancreatitis were observed. The latter condition was manifested by inflammatory swelling and induration of the head of the pancreas. In 2 of the 36 cases roentgenograms and manometric readings revealed complete stenosis of the common bile duct; recovery followed choledochoduodenostomy. In 18 cases a relative stenosis of the common bile duct was demonstrated by abnormally high manometric readings and by the roentgenographic appearance of the common bile duct, which revealed a disturbance of evacuation and deformity of the portion of the common bile duct close to the pancreatic tissue, with dilatation of the upper two-thirds of this portion. A syndrome consisting of pain, chills, elevation of temperature, and icterus was observed in 12 of the 18 cases, while pain alone occurred in 6 cases. Narrowing of the common bile duct was revealed roentgenographically in the remaining 16 cases, but the manometric readings were normal; symptoms were limited to the common bile duct in 6 of these cases, and the gallbladder was involved in 10 cases. The authors emphasize that diagnosis of these various conditions may be established only by radiomanometric control during surgical intervention. Deformity of the common bile duct due to chronic pancreatitis differs considerably from that due to compression by a pancreatic cancer or to stenosis due to a tumor of the ampulla of Vater. Recovery resulted from external drainage of the biliary tract in four of seven cases in which it was performed; the three other cases were therapeutic failures. Recovery resulted from internal drainage of the biliary tract in all the 21 cases in which it was carried out. Consequently systematic internal drainage of the biliary tract is recommended by the authors. Cholecystogastrostomy is feasible only in the absence of lesions due to cholecystitis. With this exception, preference should be given to choledochoduodenostomy following cholecystectomy in cases of complete or relative stenosis, while cysticoduodenostomy is to be considered the method of choice in cases with roentgenographic evidence of stenosis of the common bile duct but normal pressure readings. This intervention is simple and harmless, and excellent results were obtained in 16 cases in which it was performed.

Medical Journal of Australia, Sydney

1:349-384 (March 15) 1952

Antibody Titre in Maternal and Infant's Serum as Indication for Treatment in Haemolytic Disease of Newborn. G. A. Kelsall and G. H. Vos.—p. 349.
*Enuresis Dyad. J. Bostock and M. Shackleton.—p. 357.
"Occupational" Fevers, Queensland, 1950-1951. J. M. Kennedy, C. R. Lulham and D. Gordon.—p. 360.
Movable Eye Implant: Description of Implant Technique Using Cutler-Hamblin Prosthesis. J. L. Bignell.—p. 365.

Enuresis.—The incidence of enuresis is considerably higher among children who have had early and rigid toilet training than among those allowed to achieve control naturally (34 of 38 as compared with 2 of 35). Severity and coercion may succeed in establishing a habit of continence, but subsequent anxiety and frustration may cause regression. Restoration or creation of a favorable psychological environment free from anxiety and with opportunities for normal development may reverse the process and reestablish control. Enuresis was noted in only two of 35 children who were subjected to severe frustration but who had not had coercive toilet training. Frustration alone, therefore, does not produce enuresis; it merely initiates it.

Minerva Medica, Turin

43:267-290 (Feb. 13) 1952

Glycuronic Acid in Arthropody. M. Paltrinieri.—p. 267.
*Action of Vitamin E in Collagen Diseases. P. Grifa.—p. 270.
*Khellin in Treatment of Angina Pectoris. A. Martelli.—p. 274.
Technical Changes in Human Inoculation with Variola Virus. G. Turletti and G. C. Angela.—p. 278.

Vitamin E in Collagen Diseases.—The mode of action of vitamin E in correcting the metabolism of the connective tissue in patients with collagen diseases is not known. The hypothesis was advanced that this vitamin and its derivatives may inhibit hyaluronidase, which is considered to be responsible for the pathological picture of these diseases. The author studied the in vivo and in vitro influence of vitamin E and tocopherols on testicular hyaluronidase. A wheal was produced in the left forearm of 15 fasting patients at complete physical rest by injecting 0.1 cc. of hyaluronidase along with 0.1 cc. of a hemoglobin solution as a dye that does not affect the enzyme. After the spreading reactions were measured, 1 gm. of water-soluble vitamin E was given (500 mg. intravenously and 500 mg. intramuscularly), and a new wheal was produced in the same forearm with the same procedure. The new spreading reactions were definitely less extensive than the first. Simultaneously 0.1 cc. of an isotonic sodium chloride solution along with 0.1 cc. of hemoglobin solution were injected in the right forearm of the same patients. These spreading areas before and after administration of vitamin E were the same. In a second study 0.1 cc. of an aqueous solution of α-tocopherol and 0.1 cc. of hyaluronidase with 0.1 cc. of hemoglobin solution were injected in the left forearm of 15 other patients, while an equal dose of an aqueous solution of α-tocopherol was injected in the right forearm of the same patients along with 0.1 cc. of an isotonic sodium chloride solution and 0.1 cc. of hemoglobin solution. The spreading reactions of the left forearm were smaller than those of the right forearm. Additional in vitro studies demonstrated that this vitamin and its derivatives inhibit the mucinolytic enzyme even in very low concentrations, and increase the antihyaluronidase activity of human serum. Thus, one important function of vitamin E and its derivatives may be to serve as regulators of hyaluronidase activity in man. When the vitamin E levels drop below a critical concentration in the body, then the enzyme may attack its substrate. The author, in agreement with Steinberg, concludes that collagen diseases are caused by a deficiency of α-tocopherol. This may result from (*a*) insufficient intake of vitamin E, (*b*) abnormal intestinal absorption of this vitamin as seen in cases of cirrhosis of the liver, and (*c*) normal intake and absorption but abnormal use of tocopherols by the connective tissue. If this hypothesis is accepted, the good results obtained by some authors with vitamin E in patients with collagen diseases are easily understood.

Visammin in Angina Pectoris.—Visammin (khellin) was given orally to 20 patients with angina pectoris in daily doses of from 60 to 100 mg. for 3 to 12 days. All the patients were benefited, and in some the anginal attacks disappeared during the therapy. Mild nausea was the only side-effect. The electrocardiograms of some patients with mild coronary disturbances who had improved returned to normal and remained normal for six days after the therapy was interrupted, revealing a prolonged pharmacological effect of the drug. In other patients with coronary disturbances in whom anginal pain was alleviated by the drug the electrocardiograms changed only slightly. In patients with left ventricular preponderance in whom anginal attacks had become less frequent and surprising improvement had occurred, the electrocardiograms remained unchanged. The drug had a vasodilating action on the coronary system but did not influence the blood pressure of the patients.

Münchener medizinische Wochenschrift, Munich

94:681-760 (April 11) 1952. Partial Index

Progress and Problems in Abdominal Surgery. R. Zenker.—p. 681.
Disturbances After Surgical Intervention on Stomach. V. Hoffmann—p. 691.
Diagnosis of Isolated Mediastinal Struma. G. Börger.—p. 699
Surgical Treatment of Scar Strictures of Esophagus. P. Patronicolas.—p. 705.
*Reduction of Surgical Blood Loss by Hexamethonium and by Postural Control. T. Hoffmann.—p. 711.

Reduction of Blood Loss During Surgical Intervention.—The C_6 and C_5 derivatives of the methonium salts (hexamethonium and pentamethonium) block the autonomic ganglions. By increasing the stimulus threshold for acetylcholine, they exert the same effect on the nerve cells as curare does on the muscles. General vasodilatation of the arterioles results in lowering of the blood pressure of various degree, depending on the individual, his posture, and the functional condition of the ganglions. Lowering of the blood pressure combined with positioning of the patient on an inclined plane with the operative area at the highest point reduces the loss of blood during surgical intervention. With systolic values of 40 to 60 mm. Hg the loss of blood may be reduced to a minimum. Consequently the amount of blood required for replacement is insignificant. It is, however, imperative that the total circulating amount of blood be maintained by immediate replacement of even the smallest loss of blood, and hyperoxygenation of the blood must be assured. The angle of the plane determines to a considerable extent the level of the blood pressure, and this makes possible controlled lowering of blood pressure. It should be emphasized that only in the hands of an experienced anesthetist is this method without risk.

Nordisk Medicin, Stockholm

47:379-410 (March 21) 1952. Partial Index

Cardiac Irregularities and Cardiac Arrest During Anesthesia and Operation. B. S. Pedersen.—p. 379.
*Surgical Treatment of Diseases of Heart: I. Pediatric Viewpoints. E. Mannheimer.—p. 383.
*Id.: II. Coarctation of Aorta. G. Jönsson and J. Karnell.—p. 388.
*Id.: III. Mitral Defect. L. Werkö.—p. 391.
*Id.: IV. Summarizing Viewpoints. C. Crafoord.—p. 394.
Hyperactive Carotid Sinus. L. Lindén.—p. 400.

Surgical Treatment of Heart Diseases in Children.—Mannheimer reviews the clinical diagnosis and indications for operative treatment of congenital heart disease in children, and urges that all patients with congenital heart defects be admitted to special cardiological departments for examination. He considers briefly congenital heart defects that do not produce cyanosis, such as patent Botallo's duct, coarctation of aorta, anomalies of aorta and subclavian artery, and pulmonary stenosis without an overriding aorta and without septal defects. He discusses the defects that cause cyanosis, such as the tetralogy of Fallot, pulmonary atresia with overriding aorta and defect of the interventricular septum, tricuspid atresia, and Fallot's triad. Valvulotomy by Brock's method done by Crafoord in 10 cases of pulmonary stenosis with septal defect resulted in improvement in eight cases; two patients died following the intervention. Follow-up examination of 78 cyanotic children, 76 with the tetralogy of Fallot and two with tricuspid atresia, operated on in Stockholm from late 1947 to early 1950, mainly at the age of from 2 to 10, showed 10 deaths. The mortality, however, has been decreasing steadily, and the results of the anastomosis operation in suitable cases of congenital cyanosis are considered encouraging.

Surgical Treatment of Coarctation of Aorta.—Coarctation of the aorta is often discovered accidentally. Jönsson and Karnell find that the only rational treatment consists of resection of the stenosis and suture of the aorta. Of 120 patients with coarctation of the aorta examined, 94 were operated on. There were seven deaths, but one was not attributable to the intervention. All the survivors were improved subjectively. Improvement occurs more slowly in older patients. Of the objectively demonstrable improvements, normalization of the blood pressure in arms and legs is the best sign that the anastomosis is functioning satisfactorily. Not all cases are operable. If the distal end of the aortic arch is long and wide, the condition is favorable from both the operative and prognostic viewpoint; if it is long and narrow, the prognosis is less favorable; if it is short, operation is often more difficult; and if the distal end is absent, operation is impossible. The changes in the aorta that make suturing less successful increase so much between the ages of 25 and 30 that patients over 30 are not good risks. An ideal age for operation is thought to be about 10 years. The presence of another heart defect does not in principle constitute a contraindication, and it may at times increase the indication, as when there is a patent arterial duct. If the diagnosis of coarctation of aorta is established, which can be done by simple clinical methods, and the patient is of operable age, operation should not be delayed because of the absence of subjective symptoms. Careful clinical and functional analysis, however, is necessary before operation.

Surgical Treatment of Mitral Stenosis.—The indications and contraindications for mitral commissurotomy are discussed from the standpoint of heart catheterization studies. The patients who have survived operative treatment of mitral stenosis have usually shown subjective improvement. How long the improvement will continue is not yet known; Bailey's first patients, operated on more than two years ago, are still free of symptoms. Of 19 patients with mitral stenosis operated on by Crafoord, 3 died immediately following operation, all from embolism. Moderate mitral insufficiency developed in two patients. Werkö concludes that in the majority of cases isolated mitral stenosis can be treated operatively with good results. Operation is not indicated in patients with slight narrowing of the ostium reflected in relatively insignificant symptoms. In cases of marked mitral stenosis it is important to operate early. Operation should not be done when the disability depends mainly on pulmonary changes, changes after arterial embolism, or cardiac insufficiency. The immediate operative mortality is low in young patients with uncomplicated valves, but increases with the age and with increasing involvement of the heart and circulatory system.

Summary of Surgical Treatment of Diseases of Heart.—Crafoord stresses the importance of teamwork in diagnosis and treatment of cardiovascular defects and diseases. He stresses the significance of angiocardiography and heart catheterization. When correctly carried out almost no risk is associated with these examinations. In over 1,000 angiocardiographic examinations carried out in Sweden only two deaths occurred, both in cyanotic newborn infants. Angiocardiography provides such exact information preoperatively that the surgical plan can be formulated on this basis. Heart catheterization is indispensable in the evaluation of mitral stenosis, isolated pulmonary stenosis, and cardiac defects that cause cyanosis. A smaller number of atypical, prog-

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Cardiac Emergencies and Heart Failure: Prevention and Treatment. By Arthur M. Master, M.D., Cardiologist, Mount Sinai Hospital, New York, Marvin Moser, M.D., 1st Lt., USAF (MC), Walter Reed Army Hospital, Washington, D. C., and Harry L. Jaffe, M.D., Adjunct Physician, Cardiology, Mount Sinai Hospital, New York. Cloth. $3. Pp. 159, with 13 illustrations. Lea & Febiger, 600 S. Washington Sq., Philadelphia 6, 1952.

The sudden development of a condition that requires prompt and intelligent treatment is a common occurrence in cardiac disorders. Ready access to information on how to treat a patient at that time is very important to the practicing physician. He wants precise directions for handling the emergency and not a learned treatise on the mechanisms involved. In this small book, Dr. Master and his group, out of their long experience in the treatment of cardiac patients at Mount Sinai Hospital in New York and in private practice, have provided for the general practitioner just what he needs to know in the management of the cardiac emergencies. In spite of the small size of this book, a broad field of therapy is covered. In the nine chapters, the following subjects are considered: arrhythmias, acute heart failure, coronary artery insufficiency, coronary artery thrombosis, syncope, hypertensive crises, surgical emergencies, and traumatic heart diseases. A few well-chosen illustrations and tables supplement the text. Although this book is simply written and concise, it is still up-to-date, including a discussion of the important new remedies, such as procaine amide hydrochloride in the treatment of arrhythmias and mercaptomerin (thiomerin®) sodium in the treatment of congestive heart failure. The practicing physician will welcome this practical little reference book on the treatment of cardiac emergencies.

The Human Blood Groups Utilized in Disputed Paternity Cases and Criminal Proceedings. *By P. H. Andresen,* Chief, Municipal Blood Bank, Bispebjerg Hospital, Copenhagen. Cloth. $3.75. Pp. 114. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto, 2B, 1952.

The objective of this book dealing with the human blood groups is, according to Andresen, to discuss the practical application and medicolegal significance of the subject. This material as is presented does not require too much knowledge of biology, and thus the information should be understood quite readily by physicians, lawyers, and trained technical personnel. The reader is introduced to a review of the Landsteiner system of blood grouping, the various subgroups, the M-N system, and the Rh system.

The author discusses the medicolegal utilization of blood groups in disputed paternity cases. It is pointed out that blood group determinations must be performed by specialists possessing wide training in the scientific and practical bases of blood group determinations and who are also thoroughly familiar with forensic medicine and the ordinary problems connected with the use of biologic tests for judicial purposes. Of the three essential rules governing the interpretation of disputed paternity cases, Andresen points out that although the newborn infant's blood group is not fully developed, renewed examination somewhat later is required only in exceptional cases. As a rule, a baby should be 1 month of age before the blood group is determined. This note by Andresen is exceedingly important: "In Denmark, all medico-legal laboratory tests are performed at the University Institute of Legal Medicine in Copenhagen—with the exception of the actual toxicological analyses, which are done at the University Institute of Pharmacology. This means that all tests are performed in an institution, economically as well as administratively, independent of law courts and police." We in this country can profit tremendously in this regard.

This book is highly recommended to all who are interested in the subject of human blood groups.

Doctors in Blue: The Medical History of the Union Army in the Civil War. By George Worthington Adams. Cloth. $4. Pp. 253, with illustrations. Henry Schuman, Inc., Publishers, 20 E. 70th St., New York 21, 1952.

In the first few chapters of this book is a sad story of mismanagement at the start of the Civil War brought about by the lack of an effective system of finding the wounded on the battlefield and of a hospital system whereby they could be properly cared for. The military personnel for the north that gathered in the wounded at first were simply volunteers from various units of the army, almost wholly untrained in medical work. Many of the physicians at this time were volunteers from civil life who came and went more or less at their own convenience. Civilian teamsters, hired to drive ambulances, ran away or broke into the liquor supplies and behaved abominably, refusing to give water to the wounded, refusing to put the wounded in ambulances, and in some cases even going through the pockets of their helpless passengers. From this "frightful state of disorder" Surgeon General William A. Hammond concluded that the army had to have an ambulance corps controlled by the medical department rather than by the Quartermaster Corps. It was nearly four years, however, before this was brought about, largely through the strenuous effort of civilian reformers led by Dr. Henry I. Bowditch of Boston whose wounded son was brought off the battlefield on a horse; when at last an ambulance reached him, it was without water and its driver would procure none. When young Bowditch died of his injury, his father "went into battle" against the War Department's apathy, and he never ceased fighting until the Ambulance Corps Act of 1864 was on the statute books. Previously, however, Jonathan Letterman, a regular army medical officer, had tried to improve the situation by organizing the supply system on the divisional basis and by organizing an ambulance and field corps for the Army of the Potomac. He set up a system of divisional hospitals and provided for division of labor among the medical officers attached to each hospital. The ambulance details now were trained daily. Letterman set up tent hospitals organized by divisions but grouped together to make a corps hospital with one medical officer in command of all. The Letterman ambulance system reached its peak of development on Aug. 24, 1863, with the issuance of a general order that made the Army corps the normal ambulance unit and laid down details for management of the ambulances. Letterman's ambulance service for the Army of the Potomac became a model for other armies; in fact, it was the basis of the ambulance service of most of the armies of the world until World War I. J. Marion Sims, an American surgeon, was decorated by France for having introduced it into the French Army during the War of 1870. A typical Letterman division hospital train consisted of 14 army wagons and 4 medical wagons loaded with 22 hospital tents and medical supplies and equipment considered sufficient for the care of more than 7,000 men in a division. Thus, the mismanagement of the wounded in the early battles of the war evolved from confusion to an orderly system.

Having in mind our present advancements in surgical technique and new drugs effective against infection, the medical treatment of so many sick and wounded men during our Civil War now seems crude. Chloroform was the chief anesthetic. Throughout the four years some 80,000 general anesthetics are said to have been given with 37 deaths resulting from the chloroform, 4 from ether, and 2 from a mixture of the two anesthetics. It is interesting to learn that maggot therapy was rediscovered in the Civil War, only to be forgotten, then rediscovered in World War I, and now almost forgotten again. The one great orthopedic contribution to the Civil War was the splint, invented by Surgeon John T. Hodgen of St. Louis, a device that is still in use, providing extension and preventing contraction of the limb and giving comfort to the patient. Pyemia, tetanus, "surgical fevers," erysipelas, typhoid, dysentery, malaria, and gangrene were serious problems. Tetanus flared up alarmingly in the field hospital at the battle of Antietam, no doubt largely because the hospital was an old stable, the floor of which had

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The reviews here published have been prepared by competent authorities and do not represent the opinions of any official bodies unless specifically stated.

been 2 ft. deep in manure. Many of the wounded at the Battle of the Wilderness were bedded on the floors of Fredericksburg stables. Tetanus, however, was relatively rare, the number of cases being given as 505 with a mortality of 89%. With all their flaws, the Civil War hospitals, the author says, may be considered a credit to a nation, which in four years had created a vast system of 136,000 beds.

The medical record of the Civil War, on the whole, the author says, was good. This has not been generally realized, partly because the war took place before bacteriology and aseptic surgery made some of the war's medical accomplishments seem "piddling" or "irrelevant." The Civil War's morbidity and mortality rates are said to have been 2,435 and 53.4 per thousand respectively.

The author did much research before writing this book, and many of his sources of information comprise an appendix at the close of his book. It seems that he well deserved the degree for which this material was first presented as a doctoral dissertation in history at Harvard University.

Psychotherapy of Psychosis. By Gustav Bychowski, M.D., Assistant Clinical Professor of Psychiatry, New York University College of Medicine, New York. Cloth. $5.75. Pp. 328. Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1952.

The author devotes nearly all of his 34 chapters, ranging in topic from the personality of the psychiatrist to problems and prospectives, to a review of current psychoanalytic theories and techniques in the therapy of the so-called functional psychoses. He bows to other schools of thought only occasionally, e. g., in an obscure section in the phenomenology of Husserl, Minkowski, and Kronfield and in occasional passages in which Bleuler, Schneider, and Goldstein are quoted to the effect that schizophrenia may, after all, be a constitutional, metabolic, or neurophysiological disorder. Bychowski also admits *pari passu* that hypnotism "with strong suggestion," pharmaconarcosis, and even insulin therapy might be helpful as ancillary techniques of treatment, but gives no specific directions as to how to employ these in clinical practice. Instead, the author attempts to combine an outmoded Kraepelinian nosology with a welter of Freudian metapsychology expressed in the usual jargon of "id," "narcissistic identification," "weak ego core," "counter-cachexis," "primordial introjection," and similar terms vaguely meaningful to the tolerant lexicographer of psychoanalysis but for the most part mercifully beyond the ken of the general practitioners.

A few quotations from only 10 pages of the book will suffice to illustrate its style and content. Page 14: "A patient, seemingly possessed of complete indifference, could produce a strong emotional reaction either temporary or of long duration"; page 16 ff.: "The schizophrenic is affected in his vital dynamics and is thus deprived of the organization necessary to assimilate dynamics"; page 19: "The morbid emotion depersonalizes the ego and shifts the accent to the other person in whose place one would rather like to be"; page 20: "After all, no matter what theory one holds regarding the initial and the essential disturbances in schizophrenia, still nobody could deny that schizophrenics think, speak, perceive and write with their brains like anybody else"; page 23: "By way of analysis of the stream of thought I could show that the waves of energy originating in the brain of schizophrenics have special characteristics. They are incomplete and imperfect as regards their tempo as well as the degree of their delimitation. The waves, while not directed by some superior impulse, become easily independent and are unable to reach their goal. The inhibition of the stream of cerebral energy . . ."

And so on to page 245: "The impact of the sadistic superego which time and again hits the ego and punishes it with suffering and misery seems to form the main dynamic in cases of chronic depression"; page 315: "Undoubtedly many of the so-called psychopaths are latent schizophrenics where the peculiar acting out occurs as a manifestation of split-off parts of the ego."

To nearly all nonpsychiatrists (and most modern psychiatrists and psychoanalysts) such statements will sound like conceptual mystifications and semantic confusions. Conceivably, however, to some analysts still immersed in classical animistic metapsychology they may represent nuggets of insight and erudition. Bychowsky's book is recommended only to the latter.

Contemporary Correction. Editor: Paul W. Tappan, Professor of Sociology and Lecturer in Law, New York University, New York. Cloth. $5.50. Pp. 434, with 20 illustrations. McGraw-Hill Book Company, Inc., 330 W. 42d St., New York 18; Aldwych House, Aldwych, London, W.C.2, 1951.

This book presents a comprehensive symposium on the major developments and trends in the field of correction. In the foreword Sanford Bates, commissioner of the Department of Institutions and Agencies of the state of New Jersey, says, "Our penal system cannot yet be held as a success. It is the hope of Dr. Paul Tappan and the rest of us that this volume may do something toward encouraging and stimulating more widespread and persistent effort to make it so." With this purpose in mind the editor has obtained articles by qualified leaders in their respective fields and has grouped them under five main headings: part 1, Correction: Preliminary Consideration; part 2, Administrative Organization and Classification; part 3, Programs in the Correctional Institution; part 4, Types of Correctional Institutions; part 5, Extramural Treatment. To one unfamiliar with this field the book will provide an amazing experience in the revelation of the myriad problems involved in prison life. To others it will be a welcome source of information.

Physicians will be particularly interested in two chapters under part 3; in chapter 12, on medical services, correctional medical services are discussed from the viewpoint of what medical services must be rendered an inmate during his institutional residence and what the rehabilitative effects of such services will be after the inmate's release. Both the short range and long range aspects of this program are discussed in considerable detail by Dr. Justin K. Fuller, medical consultant of the Department of Corrections of the state of California and former director of the Federal Bureau of Prisons. He states, "The situations and problems that confront a correctional medical service not only are as numerous, varied, and difficult as are those found in any public or private hospital, but present some peculiar features not common to other types of medical service. . . . Acceptable modern standards of medical service include not only the diagnosis and treatment of physical illness but also psychobiology, in its broadest sense, and research. These functions must be recognized and provided for in the correctional field because of the influence that a proper medical service may have on reducing the incidence of conduct disorder, delinquency, and crime." Chapter 14, on group therapy, further elaborates on one phase of the medical program and discusses means and methods of approach. This chapter is written by Lloyd McCorkle, director of the Highfield Project in New Jersey, formerly a group psychotherapist at the Fort Knox Rehabilitation Center for military prisoners, and a prison administrator with the U. S. Army of Occupation, Japan. His discussion contains many practical suggestions together with examples of technique used.

Understanding Heredity: An Introduction to Genetics. By Richard B. Goldschmidt. Cloth. $3.75. Pp. 228, with 49 illustrations. John Wiley & Sons, Inc., 440 Fourth Ave., New York 16; Chapman & Hall, Ltd., 37-39 Essex St., Strand, London, W.C.2, 1952.

Heredity, or the science of genetics, has developed so rapidly and so precisely within the last 50 years that it has become the most central and respected of all the biologic sciences. Furthermore, it embraces topics that are of vital concern and interest to everyone. In this small book one of the world's foremost geneticists has opened the door for everyone to this most recently developed science. The author discusses in simple but exact terms such topics as the machinery of heredity, the mode of inheritance of plant and animal characters, including some human, and the origin of new variations by mutation. Helpful illustrations and charts appear. In the concluding chapter the author gives a glimpse of the more technical facts and problems of genetics. He introduces the reader to the topics of how mutations are studied, how genes act physiologically, and how genetics contributes to an understanding of evolution. The book is not a textbook and not a reference book for the specialist. It is what its title indicates, an introduction to the exciting principles and problems of genetics for the person with little or no previous knowledge of this most fascinating and important biologic subject. The book is authoritatively and well written.

Liver Injury: Transactions of the Tenth Conference, May 21-22, 1951, New York, N. Y. Edited by F. W. Hoffbauer, M.D. Cloth. $3.75. Pp. 320, with 42 illustrations. Josiah Macy, Jr. Foundation, 565 Park Ave., New York, 1951.

This book is a verbatim report of a two day conference attended by some 25 of the leading investigators in the field of liver disease. As in all Macy Foundation conferences the discussion is centered around five or six informal papers. In this volume the following subjects are presented: serum proteins and lipids by Roy H. Turner; mechanism of action of lipotropic factors in animals by Camillo Artom and action of lipotropic factors in man by David Cayer; experimental ischemia of the liver and hepatic coma by A. M. Rappaport; microanatomy of the hepatic vascular system by B. G. Maegraith and the effect of insulin on the liver in normal and diabetic men by Sheila Sherlock, A. G. Bearn, and B. Billing; and liver disease in Jamaican children by Kenneth R. Hill. Much of the material presented represents work in progress and is unpublished. The discussion is highly informal and because of the character of the participants highly informative. This volume is particularly well illustrated with tables, graphs, and plates. It has been well edited so that it reads easily. Pertinent references are included.

This is an extremely interesting and stimulating volume, which should be read by all those interested in the field of liver disease. Although much of the material is hypothetical and is only of theoretical interest at present, it should prove of interest to anyone with an inquiring mind.

Die Bluteiweisskörper des Menschen: Untersuchungsmethoden und deren klinisch-praktische Bedeutung. Von Prof. Dr. med. Ferdinand Wuhrmann, medizinischer Chefarzt, Kantonsspital Winterthur, Zürich, und Dr. Ing. Chem. Charlie Wunderly, wissenschaftlicher Oberassistent der medizinischen Universitätsklinik Zürich. Second edition. Cloth. 43.70 Swiss francs. Pp. 387, with 145 illustrations. Benno Schwabe & Co., Klosterberg 27, Basel 10, 1952.

The second edition of this excellent compilation gives, as does the first edition, detailed data on the chemistry of the plasma proteins, qualitative and quantitative protein reactions, physical, chemical, and clinical methods of study as well as the clinical significance of the plasma proteins in different disease conditions, and the production of blood proteins. Newer material, which is extensive, includes the methods and data obtained from paper chromatography, electrophoresis, spectrophotometry, ultracentrifugation, and the measurement of combination and dissociation phenomena. The material is presented in textbook fashion, and the techniques of different methods and tests are given in detail. The statements are well documented, and the extremely free use of bibliographic references, especially the European ones, is valuable for anyone wishing to make additional studies of the material. Of value to the clinician is the section (76 pages) on blood plasma data in different disease conditions and the chapter on fibrin, globulin, and albumin variations in myeloma, leukemia, anemia, kidney, liver, gastrointestinal and skin diseases, as well as in shock and other conditions. The book should be very helpful and time saving for anyone having problems or inquiries about blood proteins.

Rheumatic Diseases Based on the Proceedings of the Seventh International Congress on Rheumatic Diseases. Prepared by Committee on Publications of American Rheumatism Association, Charles H. Slocumb, M.D., Chairman. Cloth. $12. Pp. 449, with 126 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; 7 Grape St., Shaftesbury Ave., London, W.C.2, 1952.

This volume is prepared from the talks or abstracts of the talks that were presented at the Congress on Rheumatic Diseases held in May and June, 1949. The subject matter includes such topics as histology and biochemistry of connective tissue, hormone therapy, physical rehabilitation, orthopedic surgery, psychogenic rheumatism, experimentally induced lesions, and serologic studies. The papers have been grouped into chapters according to topic discussed.

The current thinking and practice of leaders from all over the world in clinical and laboratory research are presented. Although this material was originally presented in 1949, much of it is still pertinent and of current interest. This volume will be of value to those interested in rheumatology and will serve as a milepost along the difficult road of progress in the better understanding of rheumatic disorders.

When Doctors are Patients. Edited by Max Pinner, M.D., and Benjamin F. Miller, M.D. Cloth. $3.95. Pp. 364. W. W. Norton & Company, Inc., 101 Fifth Ave., New York 3, 1952.

This book was written by physicians who are or were patients suffering from serious chronic illnesses. Some of them write under their real names and others under pseudonyms. Some of their subjective descriptions of the diseases that attacked them are short, others are quite detailed. All of them are particularly interesting because the writers are both physicians and patients. Many of the accounts are beautifully written. They reflect a stoic attitude toward life, which persons in good health too infrequently attain. These seriously ill physicians are still carrying on the practice of medicine either from a desire to keep busy or from a sense of responsibility to their families. Some of the diseases from which they suffer are multiple sclerosis, epilepsy, hypertension, migraine, manic depressive psychosis, rheumatoid arthritis, pulmonary tuberculosis, cancer, morphine and alcohol addiction, coronary occlusion, sinusitis, an acute anxiety state, and chronic brucellosis. The reader will not only learn much from these unusually instructive case histories, but he will be inspired and made thankful that he is able to live without the burden of disabling chronic disease.

Clark's Applied Pharmacology. Revised by Andrew Wilson, M.D., Ph.D., F.R.F.P.S., and H. O. Schild, M.D., Ph.D., D.Sc., Eighth edition. Cloth. $7. Pp. 691, with 120 illustrations. Blakiston Company (division of Doubleday & Company, Inc.), 1012 Walnut St., Philadelphia 5, 1952.

In undertaking revision of this well-known text, Drs. Wilson and Schild have adhered to Professor Clark's original pattern of discussing scientific evidence for the therapeutic action of the more important drugs and emphasizing the importance of this knowledge in the clinical application of the drugs. The text has been considerably revised, so that the advances of the 12 years intervening between editions could be incorporated without appreciably lengthening the book. Each chapter concludes with a short bibliography and a selection of drugs both from official British and American publications and from "New and Nonofficial Remedies." Dosages throughout the text are expressed in the metric system. The book can be recommended to medical students and practitioners as a convenient-sized volume that discusses the essentials of pharmacology in a clear, understandable manner.

Advances in Enzymology and Related Subjects of Biochemistry. Volume XII. Edited by F. F. Nord. Cloth. $9.75. Pp. 570, with illustrations. Interscience Publishers, Inc., 250 Fifth Ave., New York 1; 2a Southampton Row, London, W.C.1, 1951.

This book follows the style of the previous volumes of the series. Various authorities discuss and evaluate the advances that have been made in special branches of enzymology or in some closely allied field in which the author has been active. The nine chapters in this volume include a status report on starch chemistry and the enzymes involved in starch degradation and synthesis, the hydrolysis and synthesis of acetylcholine, oxidation reduction in chloroplasts, carbon dioxide fixation by autotrophs and heterotrophes, enzyme substrate compounds, peptidase specificity, biological methylation by molds, and the reaction of borates with substances of biological interest. Each chapter contains appropriate references, and the volume contains an author and subject index. A cumulative index is also included, which lists the authors and subjects of this and the previous volumes.

Seudoartrosis. Por el Prof. M. Bastos Ansart. Cloth. Pp. 207, with 80 illustrations. Publicaciones médicas José Janés, editor, Muntaner 316, Barcelona, 1951.

This monograph, which is not a reference work, discusses the author's personal experiences with ununited fractures. There is a general discussion of etiology and pathology, followed by a consideration of clinical features. The remainder of the text is devoted to a consideration of therapy. This section covers in some detail the various operative procedures preferred by the author, and he demonstrates some good results. Other existing operative procedures of value described in texts and dissertations on the subject are mentioned only briefly or not at all. The illustrations are pertinent and of fairly good quality, and the index seems to be well detailed. There is no bibliography.

Klinische Elektrokardiographie. Von Professor Dr. Carl Korth, Direktor der medizinischen Poliklinik der Universität Erlangen. Mit einer Einführung in die Grundlagen der Elektrokardiographie und der elektrokardiographischen Technik, von Dr. Joachim Ritzmann. Fifth edition. Cloth. 25 marks. Pp. 302, with 181 illustrations. Urban & Schwarzenberg, Thierschstrasse 11, Munich 22; Meinekestr. 13, Berlin W. 15, 1952.

The fifth revised edition of this textbook of electrocardiography is a valuable addition to other recent German publications on the subject. As in previous editions a clear and concisely written text together with well-selected and well-reproduced illustrations reflects the author's great experience as a clinical electrocardiographer and teacher. Multiple chest leads and aV limb leads are widely applied, and the most important chapters are further clarified by excellent diagrams, some of which are modified or taken directly from various American texts. Only a few inconsistencies in labelling and legends need correction.

Abnormalities of contour are approached from an empirical point of view with great stress upon the type of development and of regression of changes. Vectorial analysis of the abnormal electrocardiogram, recently so much favored, is more or less omitted. Some subjects, like electrolyte imbalance, apart from changes of the calcium level, and acute cor pulmonale, would warrant more emphasis and some illustration. The chapter on cardiac irregularities is excellent, as can be expected from an author who is an "old hand" in the interpretation of complicated arrhythmias. Theoretical aspects, such as the genesis of the normal electrocardiogram, are dealt with relatively briefly, in contrast to the considerable space devoted to technical details and description of instruments. The author's view that "the string galvanometer is today only of historical interest" sounds surprising, especially since he does not hesitate to apply extensively the recent advances achieved in American centers with this type of machine.

As a whole, the book can be recommended primarily as an instructive introduction to clinical electrocardiography.

Physical Rehabilitation for Daily Living. By Edith Buchwald, M.A., A.R.P.T., Director of Rehabilitation Courses for Physical Therapists, Institute of Physical Medicine and Rehabilitation, New York University—Bellevue Medical Center. In collaboration with Howard A. Rusk, M.D., Professor and Chairman, Department of Physical Medicine and Rehabilitation, George G. Deaver, M.D., Professor of Clinical Physical Medicine and Rehabilitation, and Donald A. Covalt, M.D., Associate Professor, Physical Medicine and Rehabilitation. Cloth. $7.50. Pp. 183, with 71 illustrations. McGraw-Hill Book Company, Inc., 330 W. 42d St., New York 18; Aldwych House, Aldwych, London, W.C.2, 1952.

The purpose of this book is to present a program of basic exercise and activity for daily living that can be taught to patients with disabilities of the lower extremities. Its senior author, Edith Buchwald is a registered physical therapist and the director of rehabilitation courses for physical therapists at the Institute of Physical Medicine and Rehabilitation, New York University—Bellevue Medical Center. Her collaborators were Drs. Howard A. Rusk, George G. Deaver, and Donald A. Covalt of the same institute. The members of this team have jointly produced an illustrated manual that serves as a guide in the teaching of an "A. D. L. program" (Activities of Daily Living).

The manual contains seven chapters and is well illustrated. In chapter 1 it is pointed out that the goal of the program is functional training, which involves the physical rehabilitation of the patient in order to enable him to handle himself as sufficiently and as independently as possible. To this end, chapters are included on bed and mat exercises, wheel chair exercises, self-care, ambulation, elevation and travel on crutches, use of wheel chairs and braces, activity of daily living testing, and special equipment and furniture useful in the "A. D. L. program." This book should serve as a useful guide to any member of the rehabilitation team engaged in teaching these activities of daily living to patients.

Recreation Through Music. By Charles Leonhard, Ed.D., Associate Professor of Music, University of Illinois, Urbana. Cloth. $3. Pp. 160, with 29 illustrations. A. S. Barnes & Company, 232 Madison Ave., New York 16, 1952.

This book is of principal interest to educators and recreation directors. As its title indicates, it deals with recreation through music. The roles of making music and listening to music are discussed. The use of music by recreation leaders, the techniques for community singing and choral singing together with procedures for a sing, repertory for a song leader, instrumental classes, instrumental ensembles, and an extensive list of references and song selections are included. For the physician this book might be of special interest in relation to the use of music in occupational therapy. The book itself does not deal with this subject specifically, but a physician with occupational therapy in mind would find little difficulty in translating the materials in this volume into terms of his own needs.

Orthopaedic Appliances Atlas: A Consideration of Aids Employed in the Practice of Orthopaedic Surgery. Volume I: Braces, Splints, Shoe Alterations. Published under auspices of American Academy of Orthopaedic Surgeons, Inc., in cooperation with Office of Surgeon General, U. S. A., and Veterans Administration Department of Medicine and Surgery. Cloth. $10. Pp. 588, with 945 illustrations. J. W. Edwards, 1745 S. State St., Ann Arbor, Mich., 1952.

The purpose of this amazing atlas is to present the development of standards and technical material relative to the construction, prescription, and fitting of orthopedic appliances. It is divided into seven major parts. The first part presents a fascinating historical review. Part two, entitled "Materials and Their Use," contains a detailed discussion of the materials used in the manufacture of these appliances and presents valuable technical data concerning leather, steel and its use in orthopedics, aluminum, plastic materials, rubber, adhesives, plaster of paris, wood, industrial sewing machines in orthopedic work, tools and machines commonly used by orthopedic limb and brace makers, and engineering considerations in the design of orthopedic appliances.

Other major parts of the book consider anatomy and physiology in relation to the prescription, construction, and fitting of orthopedic appliances as well as various types of appliances that are used for the spine and trunk, upper extremity, and lower extremity. Other parts of the book are devoted to braces for the paralytic patient and to the measurements of joint motion and that of parts of the body to which a brace, splint, or prosthetic device is to be applied.

This book is probably the first such comprehensive text in the English language and is a reference book which should be in the possession of every orthopedic surgeon, physiatrist, and orthopedic appliance maker as well as any other physician concerned with the use of orthopedic appliances.

The Doctors Jacobi. By Rhoda Truax. Cloth. $3.50. Pp. 270. Little, Brown & Company, 34 Beacon St., Boston 6, 1952.

This is a warmly written, well-paced story of Dr. Abraham Jacobi, famous American pioneer pediatrician known widely in New York during the last decades of the nineteenth century as "the doctor who saves the babies," and of his wife, Dr. Mary Putnam Jacobi, who studied pharmacy in New York and medicine in Philadelphia and had the distinction of being the first woman to gain admission to the École de Médecine in Paris. The biography of these two unusual people, the husband a refugee from European politics and poverty, the wife a daughter of the publisher G. P. Putnam, comes off exceptionally well, even though some of the dialogue and detail is fictionalized. This book falls "on that neutral ground between science and popularity" and is well worth the reading time of busy medical men and women.

How to Help an Alcoholic. By Clifford J. Earle. Cloth. $1.50. Pp. 96. Westminster Press, Witherspoon Building, Philadelphia 7, 1952.

By being matter-of-fact and emphasizing the practical aspects of alcoholism and its correction this volume provides inspirational guidance. The approach is one of broad appreciation and understanding and guides alcoholics to recognize and accept their roles and thus follow courses that are most likely to end in success. How alcoholism begins, clues for its early detection, and various treatment courses that may be followed are given instructive consideration. The chapter on guidance from recovered alcoholics and the 12 steps to recovery provide "treatment" material that may well be the turning point for many alcoholics. This small volume has universal applicability.

QUERIES AND MINOR NOTES

CARDIAC MASSAGE TO OVERCOME CARDIAC ARREST

To the Editor:—*Please outline the technique for entering the chest cavity for cardiac massage to overcome cardiac arrest during a surgical procedure. What treatment is established for surgical pneumothorax that follows as a result of this procedure?*

Thomas W. Kredel, M.D., Indiana, Pa.

Answer.—The important features in dealing with such a complication are (*a*) speed in instituting the cardiac massage and (*b*) the prevention and control of ventricular fibrillation if it should occur. To accomplish these objectives it is necessary that good exposure of the heart be rapidly obtained. Some surgeons prefer to incise the left half of the diaphragm and to enter the thorax in that way if the complication occurs during the course of an abdominal operation. Such an approach, however, does not give very satisfactory visualization of the heart and makes very difficult the application of an electric shock, if needed, to control ventricular fibrillation. It seems better, therefore, in all cases to approach the heart through an intercostal incision in the fifth or fourth left interspace. A rib spreader should then be applied in order to make the exposure of the heart adequate. Care should be exercised to avoid cutting the left internal mammary artery, which ordinarily lies within about 1 cm. lateral to the border of the sternum. After the chest wall is closed the lung should be reexpanded. This can best be accomplished by inserting two tightly fitting catheters, one anterior and one posterior, into the chest cavity to provide underwater drainage. Such a procedure will remove not only the air but also the fluid that always forms after an intrathoracic procedure. It is important to carry out the intrathoracic operation with endotracheal anesthesia. If no facilities for such anesthesia are available the use of a tightly fitting face mask may be substituted, although it is not as satisfactory as endotracheal anesthesia.

LOW BLOOD SUGAR LEVEL

To the Editor:—*A patient has a persistently low blood sugar content (67.5 mg. per 100 cc.). What could be the reason for this condition, and what can be done to correct it? Except for a feeling of weakness, he has no symptoms.*

Heinrich F. Wolf, M.D., New York.

Answer.—A blood sugar level of 67.5 mg. per 100 cc., whether fasting or at any time during the day, is a mild degree of hypoglycemia. It may have no significance, but it could be due to actual or relative hyperinsulinism, the opposite condition to diabetes. Fatigue or worry in the presence of adrenal deficiency may cause spontaneous hypoglycemia. Perhaps the patient is overworked or worries too much; if so, more rest and less worry may correct the condition. The excessive use of sweets, such as desserts, candy, or soft drinks, may cause hypoglycemia by stimulating an excessive secretion of insulin by the pancreas. The patient should reduce the cane sugar products in his diet. A high protein diet, i. e., one providing meat two or three times a day and cheese or peanut butter with crackers taken three hours after meals, is helpful in such patients who are underweight or of average weight. If the patient is overweight, he should eat not more than one slice of bread at a meal, an egg for breakfast, lean meat for luncheon and dinner, with large servings of green vegetables, and medium servings of yellow vegetables, such as carrots and turnips. Fruit juice—orange, grapefruit, or apple juice—should be given on awakening, three hours after meals, and at any time for weakness. If the symptoms persist, a six hour glucose tolerance test is indicated to determine whether the patient has actual or relative hyperinsulinism.

SLIPPED FEMORAL EPIPHYSIS

To the Editor:—*A 15-year-old boy has a slipped femoral epiphysis that has rotated so that the head is almost entirely off the neck of the femur. His symptoms began 18 months ago. For six weeks he has been unable to walk. He has an atypical Frölich's syndrome. There is no involvement of the other hip. Do you think the position of the head could be improved by traction? Roentgenograms show the epiphyseal line to be still present, and fusion has not taken place. If the position has improved after 6 to 12 weeks of traction and the head has not fused, do you think the use of a Smith-Petersen nail would be indicated?*

M.D., Minnesota.

Answer.—After 18 months' duration of this condition, traction on the leg would not change the position of the capital epiphysis. Skilfully applied manipulation, with the patient under anesthesia, may effect a change of position. If so, and if the restoration were considered satisfactory, a Smith-Petersen nail or suitable screw should be inserted, without splitting the head, for fixation of the head of the femur to the neck. If manipulation fails, open operation employing a cuneiform osteotomy, reposition, and nailing is indicated. Any shortening of the leg caused by an early fusion of the epiphysis thus obtained may be compensated for in part by overgrowth of the distal epiphysis. No surgeon except one highly trained in this type of work should attempt such an operation.

SIGNIFICANCE OF ARCUS SENILIS

To the Editor:—*What is the significance of a marked arcus senilis in a man 43 years old? Is there any relationship between it and atherosclerosis? Is it a sign of the aging process?*

M.D., Toronto, Canada.

Answer.—Arcus senilis is a form of lipoid deposition in the peripheral cornea; it is usually considered to be associated with senescence; however, the condition may occur early in life, and its appearance in the fourth decade is not uncommon. The relationship between arcus senilis and atherosclerosis is debatable. Boas (Arcus Senilis and Arteriosclerosis, *J. Mt. Sinai Hosp.* **12:**79 [May-June] 1945) found an appreciably higher incidence of coronary disease among those with arcus senilis than those without it. Garn and Gertler (Arcus Senilis and Serum Cholesterol Levels in the Aleut, *New England J. Med.* **242:**283 [Feb. 23] (1950) have disputed the relationship between arcus senilis and hypercholesterolemia discussed by Boas. They conclude that hypercholesterolemia or degenerative disease need not be a concomitant of arcus senilis.

EPISTAXIS AND HEART DISEASE

To the Editor:—*Since having an attack of coronary occlusion, a patient has been using neocurtasal® (a sodium free salt substitute consisting of potassium chloride, ammonium chloride, potassium formate, calcium formate, magnesium citrate, and starch), but he has had nose bleeding. Do you think the blood vessels are more fragile after neocurtasal® has been used?*

M.D., Illinois.

Answer.—Epistaxis is a common occurrence in patients with heart disease, especially when there is associated failure, hypertension, or rheumatic fever. Since it is stated that the patient had an attack of coronary occlusion, and neocurtasal® was then prescribed, presumably there was an episode of congestive failure. Neocurtasal,® which is primarily a salt of potassium chloride, is used as a substitute for salt, so that the ingestion of sodium ion may be restricted. There has been no evidence to suggest that this compound will cause increased capillary fragility that might lead to bleeding of any type. If nosebleeds are severe and occur especially from a single site, a small ulcer or erosion should be sought and if found should be treated by application of a simple chemical (silver nitrate) or electric cautery.

The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

STIMULATION OF SPERMATOGENESIS

To the Editor:—*A man two years ago had a sperm count of 1 million per cubic centimeter, and a total semen volume of 3 cc. Results of a testicular biopsy were normal. He was given small doses of methyltestosterone, and almost complete suppression of spermatogenesis occurred. Would it be possible at present for him to have a sperm count high enough to produce a pregnancy? If pregnancy in such a case occurs, would the baby be normal?* M.D., *Ohio.*

Answer.—Testosterone may be used in the treatment of oligospermia with some hope of success if testicular biopsy shows that no irreversible changes have taken place in the germinal epithelium. Large doses are employed—50 mg. administered parenterally 3 times a week up to a total dose of 2,000 mg. or more. The initial effect is a depression in spermatogenesis leading to complete or almost complete azoospermia, but some time after therapy is discontinued a majority of the patients exhibit a "rebound" phenomenon, with sperm counts rising well above the pretreatment levels. A few of them succeed in impregnating their wives. On the other hand, in about 15% of cases the depression is not followed by a rebound, and the patients may be in a worse condition than they were before. Small doses of testosterone can depress spermatogenesis, as occurred in this case. When they are discontinued, the previous seminal values are usually restored, with no benefit accomplished and no permanent damage done.

While impregnation sometimes occurs with notably low sperm counts, in general, any reduction in numbers below the conventional figure of 60 million spermatozoa per cubic centimeter involves a corresponding reduction in relative fertility. For this reason oligospermia should be treated. In the present case it might be worth while to try the large dose testosterone method. Adjuvant measures include attention to the general health and hygiene, a high protein, high vitamin diet, and enough thyroid to keep the basal metabolic rate at or a little above the +10 level.

An alternative method for impregnation would be the preparation of a centrifuged sperm concentrate and application of this to the cervix either with a syringe or in a plastic cervical cap. The rate of miscarriage of first pregnancies among couples relieved of sterility is slightly higher than in the general population. If this hazard is escaped, the chances of having a normal baby at term will be the same as they are in other matings.

METHANTHELINE BROMIDE AND VISION

To the Editor:—*Can methantheline bromide (banthine®), 50 mg., given at bedtime, cause continued blurred vision, "circles" under the eyes, and twitching of the eyelids in a male patient 41 years of age who apparently has normal vision?* M.D., *Ohio.*

Answer.—Methantheline bromide (banthine®) causes a cholinergic blockade and will affect accommodation, if the dose is large enough, in a manner similar to atropine. When instilled into the conjunctival sac it causes pupillary dilation and cycloplegia and has an action similar to that of homatropine. It seems possible, therefore, that methantheline bromide may cause blurring of vision, severer for near objects than for distant objects, and that twitching of the eyelids may be associated with this.

FRECKLES

To the Editor:—*What are the etiology and pathogenesis of ephelides? Are there any studies on the treatment of ephelides with cortisone and corticotropin (ACTH)?*

Nehmet H. Nagle, M.D., Turkey.

Answer.—Ephelides, or freckles as they are commonly called in this country, are due to precipitation of pigment in localized patches caused by exposure to sunlight. This is often a family trait, seen mostly in blonde and redhaired persons, although it occurs also in the mulatto. Cortisone and corticotropin (ACTH) have been found to be of no value in the treatment of this condition. It is possible by the use of monobenzyl ether of hydroquinone to reduce the number of freckles temporarily.

TESTOSTERONE AND THE MALE CLIMACTERIC

To the Editor:—*In a question and answer column in a medical periodical the answer stated that giving testosterone to uncastrated elderly men seems most unwise, and that whether the "male menopause" actually exists is open to question. My reading on the subject does not coincide with this viewpoint. I would appreciate having your opinion on this subject.*

E. W. Goldstein, M.D., Pittsburgh, Pa.

This inquiry was referred to two consultants, whose respective replies follow.—Ed.

Answer.—There is a difference of opinion as to the actual existence of a condition in the elderly man comparable to the female menopause. There is not much doubt, however, that many elderly male patients develop definite psychogenic symptoms, which apparently result from changes in glandular physiology and sex hormones. Although many of these patients are definitely helped by administration of testosterone, there are others in whom androgenic therapy has no effect. Whether beneficial results from testosterone are brought about by its action as a metabolic hormone or as a sexual hormone has not been definitely determined. It should be stated, however, that long-continued treatment with testosterone in large doses might have injurious effects. Cases have been observed in which there is apparently carcinogenic action in prostatic cells.

Answer.—There is still some difference of opinion on this question; however, most of the data available indicate that giving testosterone to older men will not in itself cause carcinoma of the prostate. It may perhaps accelerate the growth of a carcinoma already present, but androgen alone will not cause normal tissue to become carcinomatous. There is enough evidence available to demonstrate that there is a male climacteric, which responds specifically to androgen therapy; however, it is not nearly as common as the menopause in the woman, and great caution must be used in arriving at the diagnosis.

STERILIZATION OF SYRINGES

To the Editor:—*Is the practice of rinsing syringes and needles in acetone or ether a satisfactory method of cold sterilization? If not, what is the preferred cold method for use by the general practitioner?*

Newton H. Reid, M.D., College Park, Md.

Answer.—The risk of causing homologous serum jaundice precludes the use of any chemical agent for disinfection of syringes and needles. There is no evidence that such agents are effective in preventing the transmission of the virus. Immersion in boiling water for 15 minutes or dry heat sterilization for an hour at 165 C (329 F) is recommended for the disinfection of previously cleaned lancets, needles, and syringes.

DISCREPANCIES IN BLOOD CELL COUNTS

To the Editor:—*I have noted discrepancies in blood cell counts performed by different laboratories. I find that one laboratory makes all red blood cell counts and hemoglobin determinations from venous blood. Another uses both venous and capillary blood, and another uses only capillary blood. Are variations in blood cell counts to be expected and, if so, to what degree?* *John T. Hecker, M.D., Cedar Rapids, Iowa.*

Answer.—From a practical standpoint, no great differences will occur between hemoglobin determinations and red blood cell counts made from venous blood and those made from capillary blood. In regard to white blood cell counts, blood from the capillaries may give somewhat higher values (1,000 to 1,500 cells per cubic millimeter) than venous blood. The discrepancies in blood cell counts performed by different laboratories are not due to differences in the location from which the blood is obtained but are the result of the great variation in accuracy and techniques existing in the different laboratories. The various factors contributing to these discrepancies are discussed in the Syllabus of Laboratory Examinations in Clinical Diagnosis (Ham, T. H., editor, Cambridge, Mass., Harvard University Press, 1950).

BRONCHIECTASIS

To the Editor:—*A 45-year-old white woman had a lobectomy for bronchiectasis two years ago. She now has bilateral involvement. Her condition remains about the same with bed rest, supportive treatment, and terramycin, 250 mg., every six hours. Without terramycin, she runs a low-grade fever. Would penicillin-sulfonamide therapy or penicillin inhalation be of any value?*

Robert L. Bestoso, M.D., Newport, R. I.

Answer.—If complete bronchograms have not been obtained since operation, these should be done to determine the distribution of the remaining bronchiectasis, since certain cases, even when the disease is bilateral, are considered for surgical resection on the second side. If the distribution of the bronchiectasis in this patient is such that surgery can no longer be considered, then the objectives of treatment should be (1) to improve drainage from the bronchial tree, and (2) to eliminate infection with antibiotics. The first objective necessitates postural drainage, performed at least twice daily; undoubtedly periodic bronchoscopic aspiration would likewise be helpful in preventing stagnation of secretions. Reasonable physical activity should be allowed, unless this results in temperature elevation or undue fatigue.

Antibiotic treatment is preferably based on laboratory determination of the susceptibility of the organisms found in the sputum or, better, in secretions obtained at the time of bronchoscopy. It appears likely that systemic administration of antibiotics is more effective than administration by inhalation alone; however, for maximum effect, combined administration (systemic and inhalation) is preferred, about one-third of the total daily quantity being given by the latter method in four divided doses. Empirical use of a sulfonamide, such as gantrisin® (3,4-dimethyl-5-sulfanilamido-isoxazole) would be justified if response to antibiotics is not satisfactory. Usefulness of maintenance doses of antibiotics entails the problem of induced resistance of the organisms present, so that intensive treatment for periods of 7 to 10 days, preferably combined with bronchoscopic aspiration, followed by an interval without specific antibacterial treatment, would seem more rational.

PLASTIC INDURATION OF PENIS

To the Editor:—*About eight months ago a plastic induration on the middle of the urethral surface of the penis developed in a patient. At that time the involved area was treated with filtered roentgen rays, and the induration was greatly reduced. Later, the patient was given ten ultraviolet ray treatments, which helped somewhat. Now the induration is as large as it was before treatment. This is the first such case seen during my long practice, and I will appreciate your opinion on treatment and prognosis.*

Angelo Marcoglous, M.D., Greece.

Answer.—This patient has Peyronie's disease, which is sometimes called plastic induration of the penis and, also, fibrosis of Buck's fascia. The treatment of this condition is very discouraging. Diathermy and other types of local heat application over a long period of time have in some instances produced a softening of the scar tissue. Treatment with continuous doses of vitamin E in the form of eprolin® (a concentrate of natural alpha, beta, and gamma tocopherols) or tocopherol acetates, 100 to 200 mg. given 2 to 3 times a day, has in a few instances resulted in regression of the induration. This has been especially true in the patient who has Peyronie's disease and Dupuytren's contracture of the fingers simultaneously. Ultraviolet or infrared ray treatments should not be used. The best improvement to date has occurred with treatment by local irradiation with combined roentgen rays and radium topically applied by an experienced radiologist. Recently, injections of solvent material, such as hyaluronidase, have been tried, without appreciable permanent improvement. Almost every type of treatment has been used, including futile surgical attempts at incision and extirpation. The disease often disappears or improves spontaneously with no treatment. The only logical treatment, used by those who see many of these patients, is a 6 to 12 month course of vitamin E therapy and local irradiation. The lesion never becomes malignant as far as is known. This disease was thought to be rare some years ago, but in the United States, at least, it occurs in about 1% of all men over the age of 55.

TUBERCULOUS LYMPHADENITIS

To the Editor:—*A woman, aged 45, with two children, had an enlarged cervical gland under the right sternocleidomastoid muscle in 1936. It was the size of an orange. Guinea pig inoculation of material obtained surgically revealed tuberculosis. There was no recurrence until 1951, at which time the family moved from southern to northern California. The gland is now the size of a walnut. Chest roentgenograms show no tuberculosis. Roentgen therapy of the gland is being considered as the patient refuses any kind of biopsy. Is a tuberculous cervical lymph gland influenced in any way by dampness?*

Willis J. Bogue, M.D., Chico, Calif.

Answer.—The question of the influence of "dampness" on cervical lymph nodes is not of great importance. Dampness may cause frequent "colds," chilling, and "lowering of resistance," but the existence of a previous infection is the chief concern. The fact that the patient had a tuberculous node removed many years ago leaves little doubt about the nature of the process, since such infections may "smolder" for many years. The treatment should be that for a tuberculous node, at least until such treatment fails to be effective. In that event, a biopsy may be mandatory. X-rays can be used successfully on early tuberculous nodes, but the best treatment is the combination of streptomycin (1 gm. twice a week) and *p*-aminosalicylic acid (PAS) (12 gm. a day in four doses). If the nodes are soft and large, surgery may also be necessary.

SENSITIVITY TO RABIES VACCINE

To the Editor:—*Is there a satisfactory way to test sensitivity to rabies vaccine?*

George A. Peck, M.D., Salt Lake City.

Answer.—There is no reliable method for testing sensitivity to rabies vaccine. It has been presumed that severe reactions, when they occur, are due to the presence of the rabbit brain tissue in the vaccine. The frequency with which antirabies vaccination accidents occur is commonly overemphasized. Their occurrence has been variously stated to be from 1 in 9,000 to 1 in 600 persons (Los Angeles, 1940 to 1946). The problem of rabies vaccine reaction is discussed in "Correction of Summary on Rabies in 1951—Report of the Academy (of pediatrics) Committee on Immunization and Therapeutic Procedures for Acute Infectious Diseases" (*Pediatrics* **9**:336 [March] 1952).

MUMPS VACCINE

To the Editor:—*Is material for skin testing for determining immunity to mumps available? If a skin test or complement-fixation test is negative in an adult male, would you recommend using Hyland's hyperimmune mumps serum?*

Etta Link, M.D., Berkley, Mich.

Answer.—Material for skin testing for susceptibility to mumps is now available (Lilly). Mumps vaccine is also used for active immunization that is presumed to endure for six months to a year. The skin test is believed to be more reliable for adults than the complement fixation test. The latter is preferred in the case of children. Hyperimmune mumps serum for passive immunization is considered to be of value by some investigators. When used in large doses for treatment it is thought to reduce the frequency of orchitis.

PROTECTIVE GOGGLES

To the Editor:—*Could headache or dizziness result from the use of work goggles with clear or colored lenses, which are not fitted by prescription, by men who do not ordinarily wear glasses?*

J. M. Hesser, M.D., Benson, Ariz.

Answer.—Protective lenses will not cause headache or dizziness unless a prism or cylinder is ground into the lens. If the lenses are without power they should have no action other than reducing the amount of light entering the eyes. Sometimes safety goggles, because of their weight, cause head pains if the frame has been improperly fitted.

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DIAGNOSIS AND TREATMENT OF EPILEPTIC AND EPILEPTOID DISORDERS

Nicholas A. Bercel, M.D., Beverly Hills, Calif.

The subject of epilepsy is important in general practice since a little more than one out of every three private epileptic patients is treated by the general practitioner. The disease is as common as tuberculosis or diabetes. Although accurate descriptions of this disease have been available since biblical times, progress in diagnosis, let alone in treatment, began in reality only in 1870, when Jackson published his study on convulsions. The elusiveness of the diagnosis already plagued the ancient Romans, who were prompted to use tests by which attacks could be provoked artificially in slaves who were on sale. The difficulties of treatment were already known in the 19th century, when Esquirol put three epileptics in the hospital and tried one treatment after another, each different, on the same patient; every treatment was effective for a short time, but none brought about a lasting cure.[1] Even though a tremendous amount of progress has been made during the past 80 years, it seems that every new diagnostic device and every new anticonvulsant drug has added as many problems as it had solved.

DEFINITION AND INCIDENCE

"Convulsion" implies a motor element, which may be absent in most of the attacks; only 25% of seizures are generalized tonic-clonic motor fits, commonly called grand mal, while 71% consist of short-lasting disturbed states of consciousness, called petit mal, and only 3% of the attacks are described as psychomotor.[2] Epilepsy is a condition characterized by repetitious attacks. A single seizure, therefore, is not—although it may turn out to be—epilepsy. A good physiological definition of epilepsy is a tendency to "periodic involuntary neuronal explosions, most often resulting in either disturbed state of consciousness or motor release phenomena," [2] or various combinations in various degrees of both. Seizure is a term covering all forms of the epileptic attack and does not carry the prejudicious connotation that epilepsy does. Roseman's [3] definition, "a state of continuing dread interrupted by recurring attacks of involuntary behavior," ought to be preferable, being both realistic and comprehensive.

CAUSATION

In three out of four cases, the underlying cause is either unknown or unreliable; this kind of epilepsy is called idiopathic or essential. When the cause is known, one out of four, the condition is termed secondary or symptomatic epilepsy. Petit mal is more prevalent in infancy and childhood. Psychomotor epilepsy is most prevalent between the ages of 15 and 35. Grand mal, on the other hand, can occur at any time, at any age. The commonest idiopathic history consists of petit mal in the first five years of life, continuing with variable frequency up to adolescence, when it is replaced by or mixed with grand mal. In the majority of cases the epilepsy is idiopathic up to the age of 20. Tumors, infections (encephalitis and abscess), and hematoma are important causes between 20 and 40 years, whereas, after the age of 40, cardiorenal disease and cerebral vascular accidents will become increasingly frequent causes. Febrile convulsions are not uncommon between the ages of 1 and 3 years, and one out of five children with febrile convulsions will later become epileptic.

The over-all incidence of hereditary epilepsy is less than it is feared—a fraction of 10%. Lennox, Gibbs, and Gibbs, however, demonstrated about 60% abnormality in the brain wave records of blood relatives of epileptics, as contrasted with the 10% of the control group, who had no near relative with epilepsy. It is commonly thought that, however abnormal the brain wave record of an epileptic parent's child may be, if he did not have recurring convulsive attacks up to the age of 21, it is wholly unlikely that he will ever be afflicted with idiopathic epilepsy. The inheritance of abnormal brain waves might (but need not) be interpreted as a predisposition to epilepsy. The chances of a child's having epilepsy, if one parent is epileptic, is 1 out of 40, with a rising probability ratio when both parents are so af-

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From the Department of Physiology, University of Southern California, Los Angeles, California.

Read at the Fifth Clinical Session of the American Medical Association, Los Angeles, Dec. 4, 1951.

[1] Temkin, O.: Falling Sickness: A History of Epilepsy from the Greeks to the Beginnings of Modern Neurology, Baltimore, Johns Hopkins Press, 1945.

[2] Fetterman, J. L., and Weil, A. A.: Practical Aspects of Epilepsy (with Special Consideration of Epilepsy in Children), M. Clin. North America **31:** 1273-1288 (Sept.) 1947.

[3] Roseman, E., and Aring, C. D.: Present Status of Epilepsy, California Med. **66:** 38-44 (Jan.) 1947.

flicted. These figures should be made available to those epileptics who so often want to put the decision of marriage and of having children up to the physician. While there may be a question about the advisability of marriage and family creation when both parties to the union are epileptic, counseling against it when only one partner is epileptic is more of a reflection on the insecurity of the adviser than a realistic appraisal of the facts as we know them today.

Birth injury, or birth anoxia, is probably a more important factor than is commonly realized. Nielsen[4] claimed that more than 50% of epileptics are first-born children, whose heads are subjected to the often traumatizing task of dilating the birth canal. Painstaking control of the anesthesia during delivery as well as better care of the newborn child and its respiratory problems could eliminate this unnecessary potential source of epilepsy.

Since the electroencephalogram can demonstrate central nervous system complications even in benign infections, such as infectious mononucleosis,[5] and since the same test done serially in a postacute period can point out focal potentially epileptogenic residuals, much of the guesswork surrounding the role of encephalitis can be done away with. Extension to the brain of the commonest childhood infections, especially scarlet fever, smallpox, influenza, measles, and typhoid, is fairly frequent. Sinus and middle ear infection used to supply a large percentage of abscesses, half of which predictably resulted in epilepsy. This is rapidly becoming a thing of the past, owing to the use of antibiotics.

Cerebral trauma, not counting subdural hematoma, is another important source. About 35% of dura-penetrating brain injuries—so common in war time—will result in post-traumatic epilepsy, while, in closed head injuries, the figure is much lower, none of the statistics going over 5%. Why so few patients with closed head injuries (which are very common) experience seizures might find an explanation in inherited cerebral dysrhythmia, which makes one more vulnerable to an accidental blow on the head. In brain trauma, serial electroencephalograms can monitor the clinically invisible state of events, pointing out the danger when post-traumatic seizure discharges make their appearance, which would even justify prophylactic anticonvulsant medication during the critical first two years following head injury, when the majority of post-traumatic epilepsies occur.

In any adult, when seizures supervene for the first time after the age of 20, cerebral neoplasm has to be looked for with bulldog tenacity. Two out of three tumors of the hemispheres result in seizures. Among the tumors most commonly causing seizures, seizure was the initial symptom in 68% of cases of astrocytoma, in 60% of meningioma, and in 51% of glioblastoma.[6]

DESCRIPTION

The contributions of electroencephalography and neurosurgery (experimental neurophysiological studies on the human) have changed our thinking with respect to the various seizure types and their origin. Grand mal is often preceded by an aura that is part of the seizure and usually indicates that the epileptogenic focus is in the cerebral cortex. These auras either are patterned (indicating successive detonation of neighboring cortical areas), e. g., the Jacksonian march, or are focal seizures, which in turn might involve motor functions (of the eyes, head, limbs, and mastication) or sensory functions (somatic, visual, olfactory, and auditory sensations). Dizziness and a variety of psychical seizures associated with disturbed consciousness and automatic behavior may appear as the forerunner of an attack. Such seizures, if they occur without the generalized motor follow-up, are classified as minor or focal seizures. Petit mal is characterized by momentary loss of consciousness, although there may also be a few minor flickers of the eyelids, usually three per second, or jerks of the arms. These attacks rarely last longer than 30 seconds. It is customary to include in the petit mal triade the myoclonic jerk and the akinetic seizure consisting of loss of postural control. Seizures initiated by loss of consciousness, or consisting of loss of consciousness only, are referred to as highest level seizures. Penfield and Jasper[7] stated that

> . . . the opinion has been held traditionally that the highest level in central nervous system hierachy is to be found in the more recently acquired areas of the cortex, those areas which serve to distinguish the brain of men from that of the ape. Experimental and EEG data suggest that the frontal lobe is not the highest level, but that in the subcortex (particularly in some of the thalamic nuclei near the midline) there is a collection of nerve cells with ready access to all parts of the cerebral cortex which, when a neural discharge originates in them, will lead to instantaneous suspension of the conscious processes.

Psychomotor seizures are characterized by transient lapses of consciousness of different degrees accompanied by automatic behavior. They are often preceded by smacking of the lips, masticatory movements, and temporary arrest of speech. It is now believed that these seizures are focal in origin and that the focus is usually in or near the tip of the temporal lobe. Automatic behavior is a dangerous aspect of epilepsy, since, in this state, without the censorship and control of consciousness, the patient can engage in actions that may be violent and even homicidal. Many of such patients have been placed in state hospitals with false diagnoses, for which mistake there can be little excuse nowadays, since the electroencephalogram, particularly done while the patient is asleep, will pinpoint the characteristic psychomotor patterns and will make them available for medical or surgical treatment.

As the clinical and experimental material with respect to cortical localization accumulates, the baffling variety of conditions under which an epileptic attack can masquerade becomes more and more evident. The following conditions were found to be in reality minor epileptic attacks: paroxysmal abdominal pain, coronary occlusion, periodic mental dulness, anxiety hysteria, renal or biliary colic, acute appendicitis, Ménière's syndrome, syncope, paroxysmal perspiration, aphasia, blindness, migraine, criminal psychopathic behavior, schizophrenia, manic-depressive psychosis, primary behavior disorder, hysterical fugue, enuresis, and nymphomania. One ought to be, therefore, sensitized to the fact that, whenever one deals with an attack that does not fit into the usual clinical picture, more particularly one that is refractory to proved accepted forms of treatment, its epileptic nature should be suspected and confirmed or ruled out by electroencephalographic study.

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[4] Nielsen, J. M., and Butler, F. O.: Birth Primacy and Idiopathic Epilepsy, Bull. Los Angeles Neurol. Soc. **13**: 176-178 (Sept.) 1948.

[5] Bercel, N. A.: Electro-Encephalogram in Cerebral Complications of Infectious Mononucleosis: Report of Case, J. Nerv. & Ment. Dis. **107**: 537-544 (June) 1948.

[6] Hoefer, F. A. P.; Schlesinger, E. B., and Pennes, H. H.: Seizures in Patients with Brain Tumors, in Association for Research in Nervous and Mental Disease: Epilepsy, Baltimore, Williams & Wilkins Company, 1947, pp. 50-58.

[7] Penfield, W., and Jasper, H.: Highest Level Seizures, in Association for Research in Nervous and Mental Disease: Epilepsy, Baltimore, Williams & Wilkins Company, 1947, pp. 252-271.

It is reliably estimated that 65% of children with behavior problems have abnormal records, as compared with a corresponding figure of 16% in a control group. The assumption is that there is similarity between the electroencephalographic pattern of the epileptics and of these children with behavior disorders, and that the symptoms of both groups are due to sudden periodic cerebral cortical changes.[8] The majority of these records show some form of paroxysmal dysrhythmia even though the true spike, or wave and spike, explosions of the classical epileptic are rarely encountered. Antiepileptic treatment, at times successful, has not reached a degree of reliability so that it could be used as a diagnostic counterproof. Since, however, periodic explosive behavior can be a form of epilepsy, the use of electroencephalograms in all cases of behavior disorders is justified, particularly when expensive long-term psychotherapeutic management or segregation in special classes is contemplated.

TREATMENT

Treatment of epilepsy—as of diabetes and tuberculosis—is a long-term program. Owing to the unpleasant and (to the uninitiated) frightening nature of an epileptic attack, the physician is often placed under a considerable amount of pressure on the part of the family or the patient to terminate the attack as soon as possible. This is not the primary aim of treatment, nor is it to use as few pills a day as possible. The general therapeutic principles can be expressed in the following way:

1. Finding the most suitable anticonvulsant and its right dose requires an average of six months. 2. One should start out with the best established drug that has proved its value and increase the dose slowly, in order to avoid the toxic effects that are common at the beginning of treatment with a new medicament or when the dose is increased suddenly. 3. One should change to newer medications only when the older ones used in adequate near-toxic doses have consistently failed. The more recent a drug, the more toxic it is, which means that precautionary laboratory examinations will also make them more expensive. 4. One should not follow fixed formulas; anticonvulsant medication has to be tailor-made. 5. Epileptics do not live on drugs alone. With these principles in mind, approximately 70% of all epileptics who have seizures can be successfully controlled or the seizure incidence decreased to the minimum.

Bromide.—This drug, introduced in 1853, was the first successful anticonvulsant. It is used chiefly in institutionalized epileptics. It appears that the side-effects of bromide medication have been exaggerated and the virtues neglected. The therapeutic dose is 5 to 15 grains (0.30 to 1 gm.) of the sodium or the triple salt, given two or three times a day.

Phenobarbital Sodium.—Phenobarbital sodium, introduced in 1912, still one of the best standbys, is quickly effective if taken on an empty stomach; on the other hand, the action lasts longer if it is taken after meals. The average dose is 1½ grains (0.10 gm.), taken three times a day, a single dose giving protection for approximately 6 to 12 hours. The commonest cause of status epilepticus (continuous seizures) is sudden cessation of phenobarbital medication. Bromides and phenobarbital sodium have a sedative effect, which varies from patient to patient.

Diphenylhydantoin Sodium.—A great landmark in modern treatment of epilepsy was the introduction of the use of diphenylhydantoin (dilantin®) sodium by Merritt and Putnam, in 1938. This drug (either the sodium salt or in oil), considering its low toxicity and high efficacy, is the best available anticonvulsant. It should be given after meals, with a single capsule of 1½ grains at the start and the dose increased by one capsule every week, until either signs of toxicity appear or seizures are controlled. By the time six capsules, in equally divided doses, are given a day, if seizures continue, one should consider combining this treatment with the administration of 1½ grains of phenobarbital sodium, particularly at nighttime, rather than increase the dose. Gradual increase until the effective dose is reached is the best safeguard against the toxic reactions, consisting of ataxia (some patients have been arrested for drunkenness), drowsiness, blurring of vision, morbilliform rash, gastric distress, and hirsutism. Hypertrophy of the gums occurs more often; massaging with rubber tooth brushes, strict dental care, and occasional surgical removal of the protruding excess gum is advisable. Because of the slow decomposition of the drug in the system, it can also be given in the morning, all at once.

Trimethadione.—The drugs so far mentioned have comparatively little effect on the petit mal triad. In fact, in some patients these minor seizures might be aggravated by the administration of diphenylhydantoin alone. The introduction of the use of trimethadione (tridione®), by Lennox, Richards, and Perlstein, constitutes an advance in treatment for petit mal no less outstanding than the discovery of diphenyldantoin for use in grand mal. The dose should be one capsule of 5 grains (0.30 gm.) for infants, two capsules between the ages of 2 and 4, and three capsules from 5 years and upward. These amounts may be increased to a maximum of three times the above amounts. Among the toxic by-effects are sensitivity to light, particularly at the beginning, and drowsiness. Trimethadione is a potentially dangerous drug, because it depresses the hematopoietic system, and some fatalities from resultant agranulocytosis and pancytopenia are on record. These occurred as often at the beginning of as after prolonged medication. Gradual administration is again the rule. A biweekly and, later (after two months), a monthly complete blood cell count, including a platelet count, is a necessity. Should the neutrophil count, which often indicates an insignificant drop, reach 2,500, decrease in dosage or even temporary discontinuance of medication is advisable. Unfortunately, even blood cell count monitoring may not give a sufficient warning, because of the insidious nature of neutropenia, for which reason this drug should not be given to patients who have a history of allergic hypersensitivity to other drugs. It seems that trimethadione given alone will aggravate grand mal seizures. In patients with mixed seizures, therefore, it is well to combine trimethadione with either phenobarbital sodium or diphenylhydantoin sodium.

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[8] Kennard, A. M., and Willner, M. D.: Significance of Paroxysmal Patterns in EEG'S of Children Without Clinical Epilepsy, in Association for Research in Nervous and Mental Disease: Epilepsy, Baltimore, Williams & Wilkins Company, 1947, pp. 308-328.

Paramethadione.—For those who are refractory to trimethadione medication, an allied preparation called paramethadione (paradione®), given in similar doses might be of value, it being clearly understood that the same precautionary measures must be observed. Paramethadione therapy is effective in cases of "highest level" seizures. Approximately 80% of patients with petit mal will show a good response to medication with the "diones."

Methylphenylethyl Hydantoin. — Methylphenylethyl hydantoin ("mesantoin") has been recently introduced and found to be efficacious in patients who fail to respond to diphenylhydantoin sodium therapy. The mode of administration and the dose are those identical with the latter drug, but, because of its relatively greater toxicity, special emphasis should be placed on slow, gradual increase to the effective dose, e. g., one-half tablet per week. Sleepiness is a common side-effect, while lymphadenopathy, morbilliform rash, exfoliative dermatitis, and neutropenia have occasionally occurred. A few cases of fatal blood dyscrasia have also been reported. As with diphenyldantoin sodium, it is best to add phenobarbital sodium if six tablets of methylphenylethyl hydantoin per day fail to produce the desired effect. Experience indicates its efficacy particularly in cases of grand mal and Jacksonian convulsions, with less effect in cases of other forms of seizures.

Phethenylate Sodium and Phenacemide.—Phethenylate ("thiantoin") sodium and phenacemide (phenurone®), two newer drugs, are also available. The latter is especially effective for psychomotor attacks, but, besides blood cell counts, liver function tests are a necessity for the detection of liver damage, which occasionally occurs.

Other Forms of Treatment.—In status epilepticus, slow intravenous injection of phenobarbital sodium, amobarbital sodium, vinbarbital ("delvinal") sodium, paraldehyde, and trimethadione is of definite value. Diphenylhydantoin sodium can also be given, through a stomach tube, along with fluids, electrolytes, and nutrients. In the refractory cases, sometimes heroic measures are needed; one has to diverge from the beaten track. Thus the use of amphetamine sulfate often helps in petit mal cases, as does the introduction of gas in the subarachnoid space by means of ordinary pneumoencephalography. Electric convulsive treatments—which raise the convulsive threshold after each seizure—were used not only with impunity but with success when everything else failed. Carbon dioxide inhalation has also been tried and has been of value. Putnam and co-workers[9] advocated "hyperintensive" treatment either with continued barbital narcosis or with toxic doses of the whole battery of anticonvulsants given on successive days; the physician should then choose the combination of drugs as a steady regimen that most improves the electroencephalogram.[9] Some drugs given in toxic doses, paradoxically, increase the electroencephalographic seizure pattern; their use should be abandoned altogether. The underlying philosophy of these special measures is the same: one's ingenuity should never concede defeat to epilepsy.

Because of technical difficulties, ketogenic diet and the administration of glutamic acid are not in great favor. Food substances producing alkalosis should be decreased and replaced by a diet that produces acidosis. The use of alcoholic beverages in any amount is absolutely contraindicated in successful treatment of epilepsy.

Surgical treatment (consisting of removal of posttraumatic scars and subpial resection of atrophied gyri) has been advocated in cases in which medical management, after a conscientious trial, has failed. In cases of refractory psychomotor epilepsy, good results may be obtained with the recently developed temporal lobectomy, which is not as yet universally adopted, although preliminary reports show encouraging results.[10] According to these, unilateral electroencephalographic spike focus, organic pathological changes, absence of rage, and good interseizure personality are related to the best postoperative results.[11]

Poor cerebral circulation due to inadequate blood flow and oxygenation is receiving increasing attention. Breathing exercises as well as any form of physical exercise are to be encouraged, even though some patients are sensitive to the results of overventilation. If they can be taught to control their breathing and therefore keep carbon dioxide from being dissipated, the nonviolent sports are permissible. Improvement of general circulation and accumulation of lactic acid with exercise are the advantages that far outweigh the small statistical probability of bringing about a seizure owing to overbreathing. Although the exact mechanism is not known, emotional upset and sudden discharges of intense affect at times will initiate an attack in the epileptic.

COMMENT

While statistics indicate that only 18% of colleges in the United States refuse admittance to epileptics,[12] only 48 per 1,000 epileptics attend college. If this is due to unofficial discrimination, this cannot find justification in the assumption that epileptics as a group have a lower I. Q., because, as a matter of fact, 65% of them have above average intelligence.

While epileptics should not be encouraged to engage in occupations where they are exposed to danger by machines, the blanket refusal of occupation in factories and stores is not justified. The personnel offices of most large firms submit a questionnaire in which the occurrence of epileptic seizures is prominently displayed. At present, many an epileptic has to perjure himself and run the risk of refusal of compensation in case of accident if he wants to make a living.

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[9] Putnam, T. J.; Bercel, N. A., and Rothenberg, S.: Hyperintensive Treatment of Recalcitrant Epileptics, to be published.

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[12] Potter, G.; McBride, M., and Lennox, W. G.: Distribution of Medical Care and Higher Education of Epileptics, in Association for Research in Nervous and Mental Disease: Epilepsy, Baltimore, Williams & Wilkins Company, 1947, pp. 539-560.

In certain states, such as California and New Jersey, it is mandatory that the physician notify the authorities whenever he handles an epileptic. There is no statistical evidence indicating that a larger percentage of epileptics are subject to traffic accidents than are normal persons. While it is good practice to prohibit driving, exceptions should be made in the cases of well-controlled, nondrinking, conscientious, and intelligent patients, and such factors as sufficient warning, strictly nocturnal attacks, and rare seizures should receive proper consideration. In some states, the personnel of the motor vehicle department judge every case on its own merit, with ample advice from and cooperation with the treating physician. A partial solution to this problem might be the issuance of temporary driver's licenses, renewable every three or six months, after recheck by the treating physician. One would welcome a comparable zeal on the part of authorities if they were to clear the highways of recidivant alcoholic drivers, a far greater potential source of accidents.

Friendly, trustworthy cooperation between physician and patient is of great importance in cases of epilepsy. The International League Against Epilepsy is an effective alliance among physicians, patients, and lay groups, which is exerting an unceasing effort to better the unenviable lot of the epileptic. Every physician should encourage his patient to join the league and use his influence to promote this remarkable enterprise.

It cannot be hoped that the lot of the epileptic can be successfully improved until every physician, particularly the general practitioner, undertakes an educational role in his community in order to change prevailing public attitudes. The following statistics are from a recent public opinion survey[13] on a national scale: 50% of persons interviewed had actually witnessed a seizure; school and play participation by epileptics was approved by over half and objected to by one-quarter; employment was advocated by 45% and objected to by 35%. While this reflects a significant progress in the understanding and acceptance of persons subjected to convulsive disorders, there still prevails enough ignorance about the epileptic, particularly in small communities, to amount to a dark stigma, which is just as undeserved as it is unjustified. The aim of treatment in epilepsy, therefore, is not just control of the seizures but also assistance to the epileptic in his adjustment on a social and economical as well as educational level.

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[13] Caveness, W.: Survey of Public Attitudes Toward Epilepsy, Epilepsia **1**: 19 (Dec.) 1949.

CLINICAL-ELECTROENCEPHALOGRAPHIC CORRELATIONS, WITH SPECIAL REFERENCE TO EPILEPSY

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Although correlations of a high degree of statistical significance have been demonstrated between changes in the electroencephalogram and certain clinical conditions, notably in epilepsy and focal organic brain lesions, every electroencephalographer is aware that dissociation of the electroencephalographic pattern and the clinical status is not uncommon. To the investigator, these facts are of great importance, since they suggest clues regarding the functional significance of rhythmical cortical electrical activity. From the clinician's standpoint, however, such discrepancies are disappointing; for, in spite of logical reasons to the contrary,[1] the hope is still widespread that a laboratory test will someday be found that will furnish clinical diagnoses. Experience with electroencephalography has shown that this is not the case, but the view is still prevalent that with the aid of the electroencephalogram one can distinguish between "functional" (usually meaning psychogenic) and "organic" (usually meaning nonpsychogenic) states.

It is the purpose of this paper to call attention to evidence that clinical and electroencephalographic changes may be widely dissociated and to discuss the significance of these findings with regard to the possible functions of spontaneous cortical electrical activity and the use of electroencephalography in clinical practice. Much of the clinical evidence is well known to electroencephalographers. However, additional illustrative material will be drawn from incidental observations on clinical-electroencephalographic studies that have been made in the course of investigations of other problems.[2] The term electroencephalogram, as used in this paper, refers to the spontaneous electrical activity of the brain as recorded clinically from scalp and, in some cases, basal leads in man, and from implanted dural leads in animals, under standard conditions.

PSYCHOSES

It is well known that the electroencephalograms of most patients with schizophrenia are within normal limits. Whether this syndrome is always associated with "cortical dysfunction" may be debatable, but there can be little doubt that impairment of cortical function is associated with both mental deficiency and Korsakoff's psychosis. Yet, in the former, entirely normal electroencephalograms are often encountered, and such dissociations have also been observed in the latter condition.[3]

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[2] Wikler, A.: Unpublished data.

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Normal electroencephalograms may also be recorded in patients with cortical atrophy demonstrated pneumoencephalographically.[4] Toxic deliriums of various sorts are usually associated with abnormalities in the electroencephalogram. However, Romano and Engel [4] found that such changes are correlated with disturbance of consciousness and not with "character and expression of anxiety, the content of thought, and the nature of sense deceptions," even though the changes may be presumed to be associated with cortical dysfunction. Furthermore, in deliriums of long standing, the electroencephalogram tends to return to a more normal pattern.[5]

Occasionally during chronic barbiturate intoxication, and very frequently during the barbiturate abstinence syndrome that follows abrupt withdrawal of the drug, psychoses may be observed. It is of interest that these "toxic" psychoses may assume the form of almost any psychiatric nosologic entity in different persons and in the same person at different times. Anxiety and panic states and hallucinatory-delusional, confusional, delirious, or amnesic-confabulatory syndromes have been observed. The concomitant electroencephalographic patterns are variable, and in some instances they may be within statistically normal limits, even when the patient is extremely disturbed. When the patient has apparently recovered completely by clinical standards, the electroencephalogram may be within normal limits and yet gross evidence of "organic" brain dysfunction may be revealed by the Rorschach technique. While impairment of consciousness is usually associated with electroencephalographic changes in the barbiturate addiction cycle, the relationship is not quantitative. This may be demonstrated most strikingly in nonpsychotic subjects during chronic barbiturate intoxication.[2] The relationship between states of consciousness and electroencephalographic changes will be further discussed below.

AFFECTIVE STATES

Many of the psychotic states referred to above are associated with disturbances of affect, and, as has already been noted, they do not appear to be related quantitatively to changes in the electroencephalogram. Such dissociation can also be demonstrated by the administration of certain drugs to nonpsychotic persons. In patients with previous histories of drug addiction ("post-addicts"), the intravenous injection of as much as 30 mg. of morphine almost invariably produces pronounced euphoria, accompanied by a transient "thrill" a second or two immediately after injection, which the subjects compare with orgasm. Concomitantly, in the majority of subjects, no significant changes can be detected by inspection in electroencephalographic tracings from multiple scalp and basal leads.[2] In the same subjects, dysphoria may be induced by large doses of mescaline or N-allylnormorphine.[6] When the affective experience is pronounced, there may be a reduction in the percentage time that alpha activity is present in the record ("alpha percentage") and also in the average voltage of alpha activity. The strong affective state may not be unpleasant. Some post-addicts whose electroencephalograms showed such changes described their experience as one of "drive"; others, as one of "anxiety." Apparently the electroencephalographic changes are related not to the quality of the affective experience but to the degree of "arousal" with which it is associated.

CHANGES IN CONSCIOUSNESS

While sleep and stupor are always associated with fairly characteristic electroencephalographic changes, the latter may be encountered occasionally in persons who are wide awake, as judged by their responsiveness to whispered instruction or by their continual execution of regular voluntary activity during recording (for example, rhythmical finger tapping).[2] Dissociated electroencephalograms of this sort have been recorded with epileptic patients during seizure-free intervals and include patterns characterized by alternating rhythmical "spindles" and slow waves, or continuous 2 to 4 cps delta activity. Continuous delta activity may also dominate in the records of patients who have been treated by repeated electroshock convulsions but who were certainly not stuporous at the time the electroencephalogram was made, several days after the last treatment. During chronic addiction to methadone, patients may have electroencephalograms that consist largely of continuous 5 cps rhythmic activity, even though a pronounced tolerance has clinically developed in the subjects and they are awake throughout the period of recording.[7] Likewise, during chronic barbiturate intoxication, there is no quantitative relationship between the severity of the electroencephalographic changes and the degree of impaired consciousness. Indeed, the electroenchephalograms of the subjects who are quite wide awake may be indistinguishable from those of nontolerant subjects who have been anesthetized by intravenous injection of barbiturates.[2]

Striking dissociations of electroencephalographic "sleep patterns" and states of consciousness have also been demonstrated in animals. In the rat, moderate doses of morphine (20 mg. per kilogram of body weight subcutaneously) produced alternating fast "bursts" and slow rhythms that resembled closely those produced by pentobarbital (30 mg. per kilogram intraperitoneally). Yet, after the doses of morphine, the rat was certainly not asleep, since it had to be restrained in a holder to permit recording, while after the injection of the doses of barbiturates, the animal was anesthetized.[8]

Similar findings have been observed in the dog. In this species, a subcutaneous injection of 5 mg. per kilogram of morphine produces regular, long-recurring "bursts" and slow wave patterns, chiefly bifrontal, which disappear abruptly when the dog is called or whistled to. In contrast, similar electroencephalographic changes that are produced by intravenous injection of 13 mg. per kilogram of pentobarbital are not altered at all by these or other more intense stimuli. After the subcutaneous injection of convulsive doses (40 to 60 mg. per kilogram) of methadone in dogs, three successive types of change may be noted in the electroencephalogram: (*a*) a "burst and slow wave" pattern, (*b*) a continuous low to moderate voltage fast activity superimposed on low voltage slower rhythms, with occasional high voltage random spikes, and, finally, (*c*) recurring, rhythmic, high voltage spike discharges. Early during stage (*b*) the dog may respond readily to stimuli, but, later, the animal may be entirely unresponsive. Very striking dissociations of electroencephalographic "sleep patterns" and behavior may also be demonstrated in dogs after the administration of N-allylnormorphine (2.5 mg. per kilogram) or of atropine (5 mg. per kilogram). Although the electroencephalogram is dominated by the "burst and slow wave" pattern, the dog may be restless and a restraint may be required to permit recording.

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[8] Cahen, R. L., and Wikler, A.: Effects of Morphine on Cortical Electrical Activity of the Rat, Yale J. Biol. & Med. 16: 239-243 (Jan.) 1944.

Converse dissociations have also been demonstrated. Magoun [9] reported that "In . . . animals with large hypothalamic and midbrain tegmental lesions, the excellence of electroencephalographic activation induced by afferent stimulation contrasted sharply with the poverty of associated behavorial arousal and the lesions appeared, in fact, to have dissociated the two features of the waking state."

SENSORIMOTOR ALTERATIONS

It is well known that sensory stimuli may be followed by an abrupt cessation of the alpha rhythm. In man, light stimuli are most effective. However, there is evidence that such stimuli produce their effects on the electroencephalogram not because of the specific anatomic arrangement of the visual system but because of the attention that they arouse.[10] Vivid visual hallucinations may be induced regularly in man by oral administration of about 5 mg. per kilogram of mescaline. Concomitantly, the alpha percentage may or may not be reduced. Usually, the subject's reports of visual hallucinations coincide with a transient reduction in alpha activity, but the sensory experiences may also occur in the absence of concomitant changes in the electroencephalogram. Conversely, marked changes in electroencephalographic tracings from the occipital regions may be noted in persons whose sensory visual system is unimpaired. Thus, in children with intact vision, subtentorial tumors may be responsible for "focal" delta activity in one or both occipital lobes, presumably because of upward pressure on the easily displaced tentorium.[2]

Jasper and Andrews [11] found that a close relationship could be demonstrated between the frequencies of the alpha rhythm recorded from the motor areas and the frequencies of finger tremors and that light stimuli produced parallel changes in both. However, these observers also noted exceptions in which electroencephalographic and finger tremor frequencies could be dissociated. One may observe clinically, in patients with various cerebral neurological disorders, the occurrence of maximal electroencephalographic changes in tracings recorded from locations on the scalp that are relatively remote from the presumed site of the organic lesion. Often, in these cases, no dysfunction can be demonstrated that can be related to the cortical area corresponding to the location of maximal electroencephalographic changes.[2] Further examples of the dissociation of electroencephalographic patterns and motor activity will be discussed in relation to epilepsy.

EPILEPSY

"Paroxysmal activity," or suddenly appearing high voltage fast or slow rhythms, is indeed highly correlated with epilepsy. The nature of the seizures exhibited by the epileptic patient is more closely related, however, to the locus of origin of the paroxysmal electroencephalographic discharge than to the patterns of the discharge.[12] Furthermore, there is a considerable body of evidence indicating that the "epilepsy," in the clinical sense, is determined not only by the presence of paroxysmal activity in the electroencephalogram but by the coexistence of other factors as yet unknown.

In patients with organic brain damage, as in severe post-traumatic states, or with brain tumors, focal paroxysmal abnormalities may exist in the absence of fits of any kind. In the nonepileptic post-addicts, hydration caused by vasopressin injection (pitressin®) may produce paroxysmal alterations in the electroencephalogram without seizures of any sort.[13] During the barbiturate abstinence syndrome, a severe paroxysmal mixed spike and slow wave activity may be observed in the electroencephalogram, with or without concomitant clinical seizures. Likewise, in some patients with epilepsy, severe paroxysmal disturbances may continue unabated, while overt seizures may be prevented entirely by administration of diphenylhydantoin (dilantin®) sodium. In patients with petit mal epilepsy, Jasper and Andrews [11] found that by overbreathing, " . . . one to ten of the spike and slow wave patterns would appear before clonic movements could be detected." Similar dissociations can be demonstrated in dogs after administration of convulsive doses of pentylenetetrazole (metrazol®). White and Giarman [14] have shown that in dogs anesthetized with cyclopropane, the intravenous injection of procaine may evoke long-sustained, continuous, diffuse high voltage "spike and dome" rhythms in the electroencephalogram, without any concomitant motor changes.

Dissociation of electroencephalographic patterns and clinical manifestations has also been observed in recent studies on the relation of the anterior portion of the temporal lobe to psychomotor epilepsy. While Gibbs, Gibbs, and Fuster [15] found that clinical evidence of psychomotor seizures was present in the great majority of epileptics in whom spike foci could be demonstrated in the anterior temporal lobes, this was true in only 46% of similar patients who were studied by Jasper, Pertuisset, and Flanigin.[16] In the latter series, a marked improvement after resection of the anterior temporal lobe was usually associated with the disappearances of the electroencephalographic foci, but exceptions were also noted. Conversely, subjective disturbances in patients with temporal lobe disease may be aggravated, while generalized seizures are controlled by anticonvulsant therapy.[17]

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COMMENT

That clinical-electroencephalographic dissociations may be demonstrated should not be surprising, for "correlations" between two sets of data imply only the possible existence of common mechanisms. "Clinical" entities are defined arbitrarily from a utilitarian standpoint, and classifications of disorders may be revised if, by so doing, prediction or treatment is furthered. "Laboratory" data are expressed in the framework of hypothetical constructs that are implicit in the use of particular techniques; and, therefore, the functions they measure may contribute to, but rarely, if ever, determine, the clinical phenomenon under investigation. Therefore, the cause, and even the definition, of clinical entities is nearly always "multifactorial." In the case of epilepsy, the demonstration of "paroxysmal activity" in the electroencephalogram does not alone define the clinical condition. Other factors, perhaps pathologically lowered thresholds of neurons that may be depolarized by electrical field changes induced by "paroxysmal" voltage disturbances arising from independent sources, may be required for the production of epilepsy in the clinical sense. It follows, therefore, that clinical terminology should not be used to describe laboratory data. Cogent reasons have been adduced for avoidance of the terms petit mal, grand mal, and psychomotor in describing electroencephalographic patterns.[18] One may go further and suggest that the word epileptic be avoided also in describing high voltage paroxysmal discharges.

Several alternative hypotheses may be advanced to explain the clinical-electroencephalographic dissociations that have been described. However, for clinical orientation and for use in further research, it may reasonably be inferred that the alpha and beta rhythms, and the diffuse changes that may occur in the electroencephalogram in the direction of synchronization or desynchronization, are determined by activity in a specific neuronal system that is, at least in part, independent of those subserving such cerebral functions as ideation, mood, consciousness, and possibly sensory and motor activity. Such a view is compatible with known facts concerning the neurophysiological basis of the electroencephalogram and the effects of drugs thereon.[19]

The functions of such a hypothesized specific neuronal system have yet to be elucidated. That it may subserve a "scanning" mechanism concerned with sensory perception and attention has been suggested by McCulloch[20] and Walter.[21] Evidence that it may subserve homeostatic functions has been adduced by Jasper[10] and Magoun.[22] However, alternative interpretations are possible concerning the specific conditions under which such homeostatic responses are likely to occur.[23]

Such hypotheses have opened new fields for investigation that may clarify greatly the proper use of the electroencephalogram in clinical practice. At present it appears timely to stress the importance of avoiding clinical terminology in describing electroencephalographic changes and to emphasize the "multifactorial" nature of clinical entities.

SUMMARY

Clinical, physiological, and pharmacologic evidence is presented indicating that wide dissociations may occur between electroencephalographic patterns and changes in sensorium, the state of consciousness, sensation, motor activity, and ideation. It is postulated that the alpha and beta rhythms, and the diffuse changes that may occu in the electroencephalogram in the direction of synchronization or desynchronization, are determined by specific neuronal system that is, at least in part, independent of neuronal systems that subserve behavio Hence, changes in the electroencephalogram, particularly those that are diffuse and bilaterally synchronou can represent only one factor that contributes to th genesis of any clinical entity, including epilepsy.

Brucellosis of the Urinary Tract.—Brucellosis of the urinary tract should be suspected in all patients complaining of urinary symptoms whose past history reveals previous systemic infection due to Brucella. Acute cystitis and roentgenographic evidences of pyelonephritis with deposits of calcium are suggestive signs. The presence of pyuria in the absence of bacteria when routine staining and cultural methods are employed lends further suggestive evidence. Positive blood agglutination reactions for Brucella at high titers, for example, 1:400, provide corroborative evidence. It must be remembered that brucellergin skin testing and a "therapeutic trial" of Brucella vaccine therapy may greatly increase the agglutinins; the agglutination reaction has uncertain diagnostic value if performed after either of these procedures. A definitive diagnosis is established by isolation of the Brucella organisms on suitable media.

Therapy with a combination of dihydrostreptomycin and sulfadiazine in our cases produced prompt and gratifying relief of the patients' symptoms. . . . A newer and more successful method of treating brucellosis has recently been advanced. This consists of the daily administration of 750 mg. of aureomycin every six hours together with 1 gm. of dihydrostreptomycin every 12 hours. Such therapy is continued for 12 to 14 days in cases of acute brucellosis and for 21 to 28 days in cases of chronic or complicated disease. This combination of drugs appears to be superior to the combination which we employed. —Laurence F. Greene, M.D.; Lyle A. Weed, M.D., and Donald D. Albers, M.D., Brucellosis of the Urinary Tract, *The Journal of Urology*, May, 1952.

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TREATMENT OF MALARIA WITH A SINGLE DOSE OF AMODIAQUINE OR CHLOROQUINE

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Since the development of 4-aminoquinoline compounds, particularly chloroquine (aralen® [Winthrop-Stearns]) and amodiaquine (camoquin® [Parke-Davis]), the treatment of malaria has become much simpler than it was a few years ago. Published reports stress the rapidity with which symptoms are alleviated and the speed with which parasites disappear from the peripheral blood when either of these drugs is administered.

Chloroquine and amodiaquine have the following pharmacologic and clinical properties in common: 1. They are rapidly and essentially completely absorbed from the gastrointestinal tract of experimental animals and presumably of man. 2. They are distributed unequally in the body tissues and are present in much higher concentrations in some tissues than in blood plasma. 3. They are slowly excreted from the body on cessation of drug therapy. 4. They are highly effective in terminating acute attacks of malaria due to either Plasmodium falciparum or P. vivax. 5. They usually result in complete eradication of P. falciparum infections. 6. They decrease the number and frequency of P. vivax relapses. 7. Neither has any marked gametocidal effect. 8. Neither has any marked effect against sporozoites, and therefore both are without prophylactic value. 9. Toxic effects with therapeutic doses are insignificant.

Chloroquine is 7-chloro-4-(4-diethylamino-1-methylbutyl-amino)-quinoline. In the World War II evaluation of potential antimalarials, it emerged as one of the more active and least toxic quinoline derivatives. Consequently it was employed widely, and has given satisfactory results in thousands of infections. It is commonly supplied in the form of the diphosphate as 250 mg. tablets, of which 150 mg. (or 60%) are chloroquine base. The usually recommended treatment of acute malaria in nonimmune persons consists of administration of 4 tablets (1 gm.) as an initial dose, followed by 2 tablets (500 mg.) after 6 to 8 hours and 2 tablets on each of two successive days.

Amodiaquine is 4(3'diethylaminomethyl-4'hydroxy-anilino)-7-chloroquinoline, supplied in the form of dihydrochloride dihydrate. It was synthesized in 1946, and subsequent trials in several parts of the world have demonstrated good results with doses frequently even smaller than those recommended for chloroquine. The 300 mg. tablets contain 200 mg. of the base. Three tablets equal 0.6 gm. of the base, and 2 tablets 0.4 gm.

From the time of its first clinical trials, amodiaquine has been frequently employed as a single dose treatment and as a one day treatment. This is true also for chloroquine, but most clinicians have preferred the three day regimen referred to above. To date, there seems not to have been any large scale study that directly compares the therapeutic effects of these two compounds, gram for gram.

In our hands [1] 0.6 gm. of amodiaquine base in a single oral dose has been completely satisfactory for treatment of acute attacks of both P. falciparum and P. vivax malaria in northwestern Honduras. We wondered whether chloroquine might be as effective when given in a comparable single dose in the same geographic area. One previous publication [2] provided a clue. Butts reported a series of 449 cases of malaria in Honduras, Costa Rica, and Panama, in which treatment with chloroquine was given on 16 schedules; 92 of this group received single doses of the drug varying from 0.08 gm. of base to 0.62 gm. of base. Butts noted that these amounts of chloroquine "appeared almost as satisfactory in this adult immune group as large doses extended over a three-day period." We thought it worth while to explore further the practicability of single dose treatment with chloroquine or amodiaquine on a comparative basis.

MATERIAL AND METHODS

This study is based on the treatment of 600 persons who resided in or near the Tela Railroad Company (United Fruit Company) headquarters at La Lima in northwestern Honduras. This is an area where Anopheles albimanus is the principal vector and where malaria is endemic all year around, but of slightly decreased incidence during the dry season from May to September. Because of the endemicity of the disease, the adult native population may be considered as a relatively immune group. All of these patients were hospitalized between February, 1950, and May, 1951, in the Tela Railroad Company Hospital located in La Lima.

The ages ranged from 7 years to 61 years. Eighty-six per cent were males and 14% were females, the difference being explained by the fact that the experiment was conducted largely on the male medical wards of the hospital. All were of mestizo stock, and represented various mixtures of Indian, Negro, and Spanish ancestry.

Amodiaquine was given to 380 patients, including 325 of the previously reported group.[1] They were placed in one of four groups to which either 0.4 gm., 0.6 gm., 0.8 gm., or 1.0 gm. of the base was given in a single dose. Chloroquine was given to 220 patients. As a result of experience already gained in the amodiaquine series, only two chloroquine schedules were employed: 0.45 gm. of base (0.75 gm. of aralen®) or 0.6 gm. of base (1.0 gm. of aralen®) given in a single dose.

Whichever of the two compounds was employed, the amount of drug and the time of administration were not related to the parasite density, to the stage of the parasite cycle observed in the blood film, or to the degree of fever. As soon as a diagnosis was confirmed by blood film, the patient was treated with whichever schedule was currently in use. Amodiaquine treatment was given from February, 1950, through December, 1950. Chloroquine treatment was given from January through May, 1951. The oral temperature was recorded every four hours on all patients. Throughout the hospitalization, thick blood films were examined twice daily, at 6 a. m. and at 3 p. m. The groups were comparable in respect to degree of parasitemia when treatment was started.

RESULTS

Clinical Response.—By reference to the table, it can be seen that only the 0.45 gm. dose of chloroquine did not prove to be quite satisfactory. There is indication that

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Dr. A. J. Walker of Tulane University of Louisiana assisted in preparation of this manuscript.

[1] Hoekenga, M. T.: The Treatment of Malaria with a Single Dose of Camoquin, Am. J. Trop. Med. 31: 139, 1951.

[2] Butts, D. C. A.: Results on 449 Cases of Naturally Acquired Malaria Treated with Chloroquine, J. Nat. Malaria Soc. 9: 44, 1950.

patients with P. falciparum malaria treated with 1.0 gm. of amodiaquine base achieved a normal temperature a little more quickly than persons given lesser amounts of this drug, but a similar difference is not noted in the temperature response of patients with P. vivax malaria. In all but nine patients headache, malaise, vomiting, joint pains, and other symptoms attributable to malaria disappeared within 36 to 48 hours, regardless of the treatment given. Convalescence was usually rapid; most patients were able to return to work by the fourth day after treatment.

Parasitological Response.—The results are given in detail in the table. In tabulating the number of hours before disappearance of parasitemia, the persistence of gametocytes was not included. Although precise records on gametocyte clearance were not kept, our impression does not differ from the accepted concept that the 4-aminoquinoline compounds are only weakly gametocidal. P. vivax gametocytes usually disappeared promptly, but P. falciparum gametocytes often persisted until after further treatment with primaquine (8-4 [amino-1-methylbutylamino]-6-methoxyquinoline) or pentaquine (8-[5-isopropylaminoamylamino]-6-methoxyquinoline).

Results of 600 Single Dose Treatments with Amodiaquine and Chloroquine*

		P. Falciparum Infection			P. Vivax Infection		
			Average Number of Hrs. Until			Average Number of Hrs. Until	
Doses, Gm. of Base	Total Number of Patients	Number of Patients	Temperature Remained Normal	Parasitemia Disappeared	Number of Patients	Temperature Remained Normal	Parasitemia Disappeared
Amodiaquine	380	185	....	....	195	....	....
1.0	75	33	13.2	26.3	42	15.3	27.5
0.8	80	40	18.5	34.6	40	14.6	25.6
0.6	125	62	17.7	31.1	63	17.8	25.0
0.4	100	50	19.8	30.7	50 (3)†	18.2	27.6
Chloroquine	220	97	....	....	123	....	....
0.6	100	41 (1)	20.0	30.2	58	17.4	22.1
0.45	120	52 (3)	31.4	41.9	63 (2)	19.2	28.5

* Later relapses are not indicated in this table.
† The figure in parentheses represents the number of cases in which the response to the single dose treatment was not satisfactory.

Treatment Failures.—Among the 220 persons who received chloroquine, there were 6 who did not respond to this treatment (one from the 0.6 gm. schedule, the others from the 0.45 gm. group). These six persons continued to have schizonts in the peripheral blood as well as elevation of temperature and other clinical symptoms that necessitated re-treatment. All responded satisfactorily to a three-day chloroquine regimen. Among the 380 individuals included in the amodiaquine groups, there were 3 who did not respond, all of whom had been treated by the 0.4 gm. schedule; they responded promptly to re-treatment with 1.0 gm. of amodiaquine. The distribution of these failures is included in the table.

Relapses.—The known number of relapses is small. Only 12 persons reported back to the hospital or outpatient dispensaries with return of symptoms and parasitemia. All had had malaria due to *P. vivax*. Four of these had been treated with 0.4 gm. of amodiaquine, five with 0.45 gm. of chloroquine, and three with 0.6 gm. of chloroquine. It is possible that some others may have taken antiplasmodial drugs on return of fever without coming to our attention in the hospital or at the dispensaries. The number of these is believed to be small, however, because most of these persons are too poor to privately purchase antimalarial medicines.

Toxicity.—In the dosages administered, no serious toxic effects were noted. Five of the amodiaquine-treated patients and four of the chloroquine-treated patients vomited after administration of the drug; in all of these, the tablets were well tolerated when administration was repeated, and so it is more logical to ascribe the vomiting to the disease than to the medicaments. Both compounds were administered to persons with malaria complicated by a number of other conditions, such as cardiovascular disease, marked malnutrition or cachexia, hepatic or renal disease, pulmonary diseases, and advanced pregnancy. In no instance was there clinical evidence of drug toxicity or intolerance.

COMMENT

It should be emphasized that this study was done in an area where malaria is endemic and the adult native population is presumably highly immune. Results of treatment among a nonimmune population or among an immune population of another location may be less favorable than our results, or might be better. Variation in parasite strain may also produce responses not completely in line with our observations.

In this locality it appears to be practical to treat most patients with malaria on an ambulant basis with very few tablets of either amodiaquine or chloroquine in a single oral dose. Probably this will prove to be true for many other endemic areas in the world, and in these areas such treatment will be economical of money, time, and hospital facilities. However, we do not advocate ambulant treatment for all cases; patients with P. falciparum infections, particularly, should be hospitalized for 36 to 48 hours whenever this is possible in order to better cope with potential complications.

The time for disappearance of parasites from the blood is stated conservatively in all instances. Since blood films were made only two times a day, at 6 a. m. and at 3 p. m., the disappearance of parasites would only occasionally be noted shortly after its actual occurrence; frequently a lapse of a few hours could be presumed between the actual disappearance of parasites and its demonstration at the next regular time for blood examination.

None of the patients received any medication parenterally. Amodiaquine has been used parenterally with success [3] elsewhere, and chloroquine has been employed intravenously, with success here in Honduras.[4] It is essential that reliable preparations such as these that can be given parenterally be always available for treatment of those severely ill patients who, because of coma or vomiting, are unable to take the medication orally.

SUMMARY

Six hundred Hondurans were treated for malaria with a single oral dose of either amodiaquine (camoquin®) or chloroquine (aralen®). Four amodiaquine doses were used among 380 patients: 0.4 gm., 0.6 gm., 0.8 gm., and 1.0 gm. of the base. Two chloroquine schedules were employed among 220 patients: 0.45 gm. and 0.6 gm. of the base. All of the amodiaquine groups as well as the 0.6 gm. chloroquine group showed satisfactory clinical and parasitological responses. The response in the 0.45 gm. chloroquine group did not prove entirely satisfactory.

Only 9 individual treatment failures were observed among the 600 patients. Six patients had received small amounts of chloroquine (0.45 or 0.6 gm. of base), and three had received 0.4 gm. of amodiaquine. These cases do not include those in which there was persistence of gametocytes in the peripheral blood, which occurred in a small but unknown number of additional persons. Twelve relapses of P. vivax malaria were observed. Four of these occurred after treatment with 0.4 gm. of amodiaquine and the others occurred after small doses of chloroquine (0.45 gm. or 0.6 gm.). The toxic side-effects of both compounds are insignificant with the dosages used. Obviously, it is of great advantage to have two antiplasmodial agents that are applicable to mass treatment with a single small oral dose.

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[3] Payne, E. H.; Sharp, E. A., and Nickel, K. C.: Parenteral Use of Camoquin Hydrochloride as an Antimalarial, Am. J. Trop. Med. **29**: 353, 1949. Payne, E. H., and others: Intravenous Amodiaquin (Camoquin) in Naturally Acquired and Induced Malaria, ibid. **31**: 698, 1951.

[4] Scott, V.: Single Intravenous Injections of Chloroquine in the Treatment of Falciparum Malaria: Toxic and Immediate Therapeutic Effects in 110 Cases, Am. J. Trop. Med. **30**: 503, 1950.

A STUDY OF RUBBER ADHESIVES IN SHOES AS THE CAUSE OF DERMATITIS OF THE FEET

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Dermatoses of the feet are troublesome and rather common. They range from mild fissuring between the toes to severe vesiculation, oozing, and edema of almost the entire foot. They may be of short or long duration. They may be sufficiently severe so that a physician is consulted early in the course of the disease, or they may be mild enough to have been self-medicated for many months before the aid of a physician is sought. The signs and symptoms of different dermatoses of the feet may be sufficiently similar so that it becomes difficult to diagnose and treat the dermatoses. It is the purpose of this paper to discuss the specific agent that causes allergic eczematous contact-type dermatitis of the feet, a condition that we believe is now being confused with epidermophytosis, bacterial infections, and inflammatory skin conditions from other causes. This condition clears readily when the allergen is eliminated. Usually we have been able to determine the specific allergen in these cases, and we can now state which allergens should be used for testing patients with such conditions.

In 1949, Gaul and Underwood [1] pointed out that many dermatoses of the feet, which are diagnosed and treated as fungous infections, are more correctly diagnosed as a dermatosis caused by an irritant or allergen in the shoes of the patient. They were able to support their opinion with positive results of patch tests to various portions of the patients' shoes. The patch test data led Gaul and Underwood to be suspicious of the adhesive materials in the shoes. They attempted to obtain more information about these materials but reported, "Letters to companies manufacturing adhesive materials brought two samples and a decided reluctance to divulge their ingredients."

Jordon [2] studied 43 cases of dermatitis of the feet and concluded that resinous shoe linings were the cause of the dermatitis, though he was unsuccessful in obtaining any positive reactions to any resin or other single ingredient in the resinous lining in any case that he studied. A study of rubber footwear as a cause of foot eczema led Bonnevie and Marcussen [3] to the opinion that the specific allergen was one or more of the accelerators of the mercaptobenzothiazole type, which were used in the manufacture of the rubber.

During the past year we have studied 24 cases of dermatitis of the feet that seemed to be caused by shoes. In each of the cases there was a history of a dermatitis that could be correlated to the wearing of one or more pairs of shoes. Positive patch test reactions to portions of the shoes were obtained to support the history in 20 of the cases. In three we were unable to test the suspected shoes. In only one were we unable to obtain a positive reaction from any of the shoe materials.

Twenty-one of the 24 patients were women, and in a large majority of them we found positive patch test reactions to the light-colored, combined, cloth linings in the

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[2] Jordon, J. W.: Contact Dermatitis Due to Synthetic Resins in Shoe Linings, Arch. Dermat. & Syph. **62**: 671-680 (Nov.) 1950.

[3] Bonnevie, P., and Marcussen, P. V.: Rubber Products as a Widespread Cause of Eczema: Report of 80 Cases, Acta dermat.-venereol. **25**: 163-178 (Sept.) 1944.

shoes. These combined cloth linings usually consisted of a faille and a coarser backing cloth, which were held together with a rubber adhesive. The close association of dermatitis due to shoes with dermatitis due to rubber has been discussed in each of the previously cited references. We felt that the rubber adhesive used in the combined linings might be the cause of the dermatitis, and we proceeded on this basis.

Some survey testing of the patients indicated that it was likely that they were sensitive to an antioxidant or accelerator used in the rubber adhesive, as in Bonnevie and Marcussen's [3] cases. Ten representative antioxidants and 17 accelerators [4] used by the rubber industry were made available to us, and as many of the patients as possible were tested with these materials. Numerous vesicular reactions were observed, but it became apparent that a good many of the patients reacted to none of the materials tested. Also, it was not known definitely that these particular antioxidants or accelerators were used in the fabrication of the adhesives used in the combined linings.

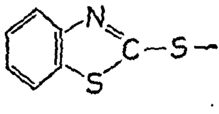
Subsequently a manufacturer of combined linings supplied us with the separate ingredients and combinations of ingredients that are present in the adhesives used in some combined linings. These ingredients included various kinds of rubber, an antioxidant, a tackifier, a plasticizer, a peptizing agent, and two accelerators. It soon became apparent that the patients reacted violently to the antioxidant and to any combination of ingredients containing the antioxidant. This antioxidant had not been among those previously used by us for patch tests. Some of the patients who had reacted to none of the other antioxidants or accelerators were recalled and were shown to react positively to this antioxidant. Unfortunately all patients could not be recalled for testing with the new material. This antioxidant is the monobenzyl ether of hydroquinone.

Table 1 shows the patch test data for 21 of these patients. (The three patients who showed no reactions to any of these chemicals are omitted.) Five of the 17 accelerators contain a compound possessing a mercaptobenzothiazole radical:

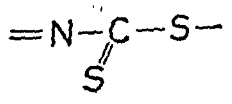
TABLE 1.—*Patch Test Reactions to Accelerators and Antioxidants**

	Group I										Group II			Group III				Group IV			
Case number	1†	2†	3	4	5	6	7	8	9	10	11	12	13	14†	15†	16	17	18	19	20	21
Accelerators																					
Mercaptobenzothiazole type																					
2-Mercaptobenzothiazole	−	−	−	−	−	−	−	−	−	−	+	+	−	−	+	−	−	+	+	+	−
2,2'-Benzothiazyl disulfide	−	−	−	−	−	−	−	−	−	−	+	+	+	−	+	−	−	+	+	−	−
Zinc benzothiazyl sulfide	0	−	−	−	0	0	−	0	0	0	+	+	+	−	0	0	−	0	0	0	0
Benzothiazyl sulfenamide	−	−	−	−	−	−	−	−	−	−	+	+	+	−	+	−	−	+	+	−	−
3-Anilinomethyl-2(3)-benzothiozolethione	−	−	−	−	−	−	−	−	−	−	+	+	−	+	+	−	−	+	+	−	+
Mixed																					
Zinc benzothiazyl sulfide and tetramethylthiuram monosulfide	−	−	−	−	−	−	−	−	−	−	+	+	+	−	+	+	+	+	+	−	−
A mercaptobenzothiazole and a dithiocarbamate	−	−	−	−	−	−	−	−	−	−	+	+	+	−	+	−	−	+	+	−	−
Dithiocarbamyl type																					
Tetramethylthiuram monosulfide	−	−	−	−	−	−	−	−	−	−	−	−	+	−	−	+	+	−	−	−	−
Tetramethylthiuram disulfide	−	−	−	−	−	−	−	−	−	−	−	−	+	−	−	−	+	−	−	−	−
Selenium dimethyl dithiocarbamate	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+	−	−	−	−
N-pentamethylene ammonium pentamethylene dithiocarbamate	−	−	−	−	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−
Miscellaneous																					
2-Mercaptothiazoline	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Diphenylguanidine	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Diphenylguanidine phthalate	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Butyraldehyde-aniline condensation *product*	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+
Triethyltrimethylenetriamine	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Hexamethylene tetramine	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Antioxidants																					
Monobenzyl ether of hydroquinone	+	+	+	+	+	+	0	0	0	0	−	−	−	+	+	+	+	0	0	0	0
S-di (β-naphthyl)-*p*-phenylenediamine	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Mono and dioctyl diphenylamines (mixture)	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Polymerized trimethyl dihydroquinoline	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+
Aldol alpha naphthylamine	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Phenyl beta *naphthylamine*	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+	−
Phenyl alpha naphthylamine	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+
Reaction product of diphenylamine *and* acetone	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
A diarylamine-ketone-aldehyde reaction product and N,N'-diphenyl-*p*-phenylenediamine	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Acetone and aniline condensation product	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Polyalkalated phenol sulfide	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−

* − = no reaction or mild erythema; + = vesiculation; 0 = not tested.
† These patients were tested with special shoe linings (see text).



Four contain a compound that has a dithiocarbamyl radical:



$$=N-\underset{\underset{S}{\|}}{C}-S-$$

Two are mixtures of both types of compounds. The other accelerators and antioxidants have various chemical

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4. Mr. C. C. Davis of the Boston Woven Hose and Rubber Company supplied these materials.

structures. All the materials were tested in 1% concentration in petrolatum. The tests were made on the upper arm, were kept on the skin for 48 hours, and were read one hour after removal. Only vesicular reactions are recorded as positive; no reaction and mildly erythematous reactions are recorded as negative; and "0" indicates that the patient was not tested. These data are summarized in table 2.

The numbers in parenthesis in table 2 show the number of individual accelerators of the type indicated to which the patient showed reactions. The patients have been divided into five groups. In group I are the patients who reacted only to monobenzyl ether of hydroquinone or those whom we suspect would have reacted to this material had we been able to recall them for testing. (These latter patients had positive reactions to typical combined linings and were not sensitive to any other antioxidant or accelerator.) In group II are the patients who were not sensitive to monobenzyl ether of hydroquinone but were sensitive to the mercaptobenzothiazole or dithiocarbamyl compounds. In group III are the patients who were sensitive to monobenzyl ether of hydroquinone and to either the mercaptobenzothiazole compounds or the dithiocarbamyl compounds. In group IV are patients who were sensitive to mercaptobenzothiazole compounds and occasionally to some other compound but who were not tested to monobenzyl ether of hydroquinone. In the last group are three patients who showed no positive reactions to any of the antioxidants or accelerators. One was sensitive to a typical combined lining, one to a box toe in the shoe, and one to brown dyed leather.

TABLE 2.—*Summary of Patch Test Data**

Case No.	Shoes	Monobenzyl Ether of Hydroquinone	Mercaptobenzothiazole Type	Dithiocarbamyl Type	Others
Group I					
1†	+	+	—	—	—
2†	+	+	—	—	—
3	+	+	—	—	—
4	+	+	—	—	—
5	0	+	—	—	—
6	0	+	—	—	—
7	+	0	—	—	—
8	+	0	—	—	—
9	+	0	—	—	—
10	+	0	—	—	—
Group II					
11	+	—	+ (7)	—	—
12	+	—	+ (7)	—	—
13	+	—	+ (5)	+ (5)	—
Group III					
14†	+	+	+ (1)	—	—
15†	+	+	+ (6)	—	—
16	0	+	—	+ (2)	—
17	+	+	—	+ (4)	—
Group IV					
18	—	0	+ (6)	—	—
19	+	0	+ (6)	—	—
20	+	0	+ (1)	—	+ (1)
21	+	0	+ (1)	—	+ (2)
Group V					
22	+	—	—	—	—
23	+	—	—	—	—
24	+	—	—	—	—

* — = no reaction or mild erythema; + = vesiculation; 0 = not tested. Numbers in parentheses indicate the number of accelerators of the type indicated to which the patient reacted.

† These patients were tested with special shoe linings (see text).

It is our opinion that monobenzyl ether of hydroquinone is frequently the allergen in dermatoses that arise from the combined linings of shoes. The patients who were sensitive to this antioxidant often gave a history of dermatitis from other rubber articles (garters, elastic, rubber tubing used for a tourniquet, and adhesive plaster). Patients who reacted to the antioxidant, monobenzyl ether of hydroquinone, obtained from a rubber company, also reacted to the pure chemical, monobenzyl ether of hydroquinone, as obtained from the Eastman Kodak Company. This would tend to rule out the possibility that the patients might be reacting to an impurity in the antioxidant. They often showed a severe vesicular reaction to a concentration of 1 part of the chemical in 10,000 parts of petrolatum. Those whom we have tested do not react to hydroquinone.

The combined lining manufacturer made two linings for us, which were the same in all respects except that one contained monobenzyl ether of hydroquinone and an accelerator of the mercaptobenzothiazole group and the other contained neither of these materials. All other ingredients were identical in the two linings.

These linings were fabricated into shoes,[5] one lining in the right shoe and the other lining in the left. Pairs of these shoes were given to four patients (cases marked with a dagger [†] in groups I and III). The patients were not told that the linings were different, nor could they tell any difference from looking at the linings. They were asked to wear the shoes for short periods of time and report to us. Each patient showed vesicles on the dorsum of the foot on which was worn the shoe made from the lining containing monobenzyl ether of hydroquinone and the mercaptobenzothiazole accelerator. This vesiculation occurred 12 to 24 hours after the shoes had been worn for approximately 30 minutes. The other foot remained normal.

It would not be possible from this experiment to determine whether the monobenzyl ether of hydroquinone or the accelerator was the allergen for two patients (cases 14 and 15), since they were sensitive to both substances. For two others (cases 1 and 2), however, monobenzyl ether of hydroquinone must be the allergen, since these two patients were not sensitive to the accelerator.

COMMENT

Monobenzyl ether of hydroquinone is the antioxidant that was reported by Oliver, Schwartz, and Warren [6] to have caused leukoderma among tannery workers who wore rubber gloves that contained this antioxidant. There have been other reports [7] of the depigmentation that this compound causes. The ability of this compound to cause allergic hypersensitivities has not been stressed. Schwartz [8] stated that, on trying to cause depigmentation of his own skin, he caused an eczematous-type reaction. In the discussion of the Oliver, Schwartz, and Warren paper,[8] Sulzberger stated that, in an attempt to cause depigmentation with monobenzyl ether of hydroquinone by experimental patch tests, he produced an eczematous, allergic specific sensitization in "approximately 50 per cent of the cases" by the application of full-strength monobenzyl ether of hydroquinone. Lockhart and Loewenthal [9] commented, "Although the reaction of contact-type, eczematous dermatitis is obtained by patch testing, several authors believe that depigmentation may occur without such initial reaction." Botvinick[10] reported a case of leukoderma and dermatitis from a pair of rubber gloves that were shown to contain monobenzyl ether of hydroquinone. We seldom saw evidence of depigmentation in any of our patients. It is true, however, that we did not follow many of these patients for very long after the acute phase of the dermatitis had subsided.

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[5] The United Shoe Machinery Corporation, Beverly, Mass.

[6] Oliver, E. A.; Schwartz, L., and Warren, L. H.: Occupational Leukoderma, Arch. Dermat. & Syph. 42: 993-1014 (Dec.) 1940.

[7] Spencer, G. A.: Leukoderma Produced by Antioxidants: Report of Cases, Arch. Dermat. & Syph. 58: 215-219 (Aug.) 1948. Bernstein, E. T., and Sachs, P. M.: Pigmentary Disturbance Following Exposure to Monobenzyl Ether of Hydroquinone, ibid. 59: 542-548 (May) 1949.

[8] Schwartz, L.; Oliver, E. A., and Warren, L. H.: Occupational Leukoderma, Pub. Health Rep. 55: 1111-1130 (June 21) 1940.

Monobenzyl ether of hydroquinone has been studied mostly with respect to its depigmenting action among rubber workers and wearers of rubber articles. Its eczematizing action has been observed during attempts to cause depigmentation experimentally. Allergic, eczematous, contact-type dermatitis among wearers of rubber articles containing monobenzyl ether of hydroquinone has been less frequently reported. The manufacturer of this material has reported to us that no dermatitis has occurred among the workmen who handle this antioxidant. Nevertheless, we suspect that its sensitizing index is relatively high when it gets into contact with the skin in shoes or adhesive plaster, though certainly not nearly as high as the 50% reported by Sulzberger in his small patch test series. It seems sufficiently high so that every effort should be made to prevent the use of monobenzyl ether of hydroquinone in rubber articles that come into contact with the skin.

Allergic, eczematous contact-type dermatitis to other antioxidants and accelerators that we tested is less frequent. It must not be concluded that those compounds that showed completely negative reactions in this group of patients are safe compounds to use. It may be that these patients had not previously been in contact with these compounds and that, therefore, they would not be expected to show a reaction to them. A good example of this situation is the compound hexamethylenetetramine, an accelerator that is seldom used by the rubber industry today. It has been discarded because it was found to have a high index of sensitization. Also, it must be remembered that substances that are not allergenic for this group of patients might sensitize even a higher percentage of persons from the population at large.

In a suspected case of dermatitis caused by shoes it is certainly not necessary to test to the large group of materials we used. Three types of compounds could be used: (1) monobenzyl ether of hydroquinone, (2) 2-mercaptobenzothiazole, and (3) tetramethylthiuram monosulfide. If these three compounds are used, it is probable that reactions will be found to one or more of them if the shoe dermatitis is caused by one of the rubber ingredients of the shoes.

We wish to emphasize the opinion previously expressed by Gaul and Underwood [1] that, when a patient is seen with a dermatitis of the feet, it is important to remember that the dermatitis may result from an allergic hypersensitivity to some ingredient in the shoe. In the cases of this type that we have studied, the most frequently observed allergen was an antioxidant or accelerator used in the rubber adhesives in the shoes.

SUMMARY

An acute dermatitis of the feet may be an allergic, eczematous, contact-type dermatitis. The allergen for those patients we have studied was most frequently the antioxidant monobenzyl ether of hydroquinone, which is used in the rubber adhesive in the combined linings of the shoe. Some of these patients were specifically sensitive to other rubber antioxidants and accelerators.

Fruit St. (14) (Dr. Blank).

9. Lockhart, R. J. J., and Loewenthal, L. J. A.: Occupational Leukoderma: A Report of 6 Cases Observed in a South African Factory, South African M. J. 23: 867-868 (Oct. 22) 1949.

10. Botvinick, I.: Dermatitis and Secondary Leukoderma Due to Fabric-Lined Rubber Gloves: Report of a Case, Arch. Dermat. & Syph. 63: 334-335 (March) 1951.

STERILITY OF MARKET COTTON

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The consumer's guarantee of sterility in products labeled "sterile" lies chiefly with the producer. In the case of "sterile" market cotton,[1] the control program of the Food and Drug Administration, in the Los Angeles area at least, consists of taking random samples at periodic intervals. These are sent to the Federal Drug Administration in Washington, D. C., for sterility testing. Results of these investigations are not available. In view of this, it was decided to make a survey of "sterile" market cotton to determine whether the present control program is adequate to insure sterility in all packages of cotton.

EXPERIMENTAL DATA

Procurement.—Samples of "sterile" market cotton were purchased from retail stores in various parts of the city of Los Angeles. Generally a single 4-oz. roll of a nationally prominent brand was purchased from each retailer. At the time of purchase, no mention was made of the survey being conducted. Only undamaged packages, labeled "sterile," were used.

Sampling.—For the purpose of sampling, a large transfer chamber containing a Westinghouse sterile lamp

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[1] Purified cotton (sterile absorbent) U. S. Pharmacopeia XIV.

was used. The chamber was completely enclosed except for hand inserts. Previous to use, the chamber was completely swabbed with a technical grade of benzalkonium chloride (roccal,® 1: 1,000 solution). All sampling procedures were carried out under ultraviolet rays.

The outside of each package of cotton was thoroughly wetted with benzalkonium chloride before being placed in the transfer chamber. The moist package was exposed to the ultraviolet rays for at least 10 minutes before the outer covering was broken. This allowed for complete drying in a sterile environment. The cotton roll was removed from the package, and the paper enclosing the roll was swabbed with benzalkonium chloride. The first sample was taken from the end of the roll, and subsequent samples were taken at intervals of approximately 12 in., moving toward the center of the roll. In total, five samples were obtained from each 4-oz. roll.

Sterile applicator sticks were handled with hemostats, which were previously allowed to stand in benzalkonium chloride for a period of three minutes and then flamed before use. An amount of cotton, approximating that used for a throat swab, was twisted on the end of the stick. The stick was then broken at a point above the sample, with sterile hemostats used for all manipulations, and the swab was dropped into an 8-oz., wide mouth, sterile bottle containing approximately 50 ml. of sterile fluid thioglycollate medium (as recommended in the U. S. Pharmacopeia for sterility tests). Previous to use the outer surfaces of the culture bottles were swabbed with benzalkonium chloride, and the bottles were then allowed to stand in the chamber for 10 minutes.

The bottles containing the medium were incubated for 48 hours at 37 C previous to inoculation as a check on the initial sterility for the medium. Along with each roll sampled, a control bottle was inoculated with a 3-cm. section of a sterile applicator stick that was handled in exactly the same manner used for all other samples taken. It must be emphasized that the control bottle for each roll of cotton tested was inoculated in the same atmosphere as the test samples and had equal opportunity for chance contamination.

The samples thus obtained were incubated at 37 C for three weeks and examined at weekly intervals. Those bottles showing turbidity were checked for the presence of micro-organisms by means of microscopic observation of gram-stained smears. The viability of the organisms was determined by subculture on nutrient agar.

Results.—Table 1 summarizes the results obtained from the examination of 85 different rolls of market cotton. It can be seen that in 14 out of 413 different samples viable organisms were detected. Twelve out of 85 rolls of cotton contained viable micro-organisms. In two instances two positive results were obtained from the same roll. Of the 14 samples containing viable organisms, one was obtained from the outermost portion of the roll; five were obtained 1 ft. from the outside; four were obtained 2 ft. from the outside; two were obtained 3 ft. from the outside; and two were obtained from the innermost center of the roll. As nearly as could be determined the positive samples each contained a single type of bacteria. Gram-positive cocci were found in seven samples, gram-negative, non-spore-forming rods in three, a gram-positive, anaerobic, spore-forming rod in one, a gram-negative spore-forming rod in another, and the gram-positive rods in two.

ISOLATION AND DETERMINATION OF HEAT RESISTANCE OF CONTAMINANTS FOUND IN MARKET COTTON

Four of the positive samples were selected for further study. Pure cultures were obtained from the thioglycollate broth with the use of the loop dilution and streak plate techniques. The cultures thus obtained were as follows: no. 49, short, gram-negative, non-spore-forming, aerobic rods; no. 52, gram-positive micrococci (albus type); no. 58, short, gram-positive rods showing extreme pleomorphism with a definite tendency to show coccoid forms; and no. 63, gram-negative, aerobic, spore-forming rods.

For the determination of the heat resistance of the above isolates, the organisms were grown at 28 C in bottle slants containing nutrient agar and harvested at the point of maximum growth. The cells were suspended in M/20 phosphate buffer with a pH of 7.0, washed twice by centrifugation, and resuspended in the buffer. Plate counts were made from the final buffer suspension.

TABLE 1.—*Results of Sterility Tests on Market Cotton* *

Brand	No. of Rolls Examined	No. of Samples	No. of Controls	No. of Positive Samples Showing Viable Organisms	No. of Positive Controls Showing Viable Organisms
A................	15	75	15	2	0
B................	15	75	15	5	0
C................	15	75	15	2	0
D................	15	75	15	2	0
E................	15	75	15	3	0
F................	3	15	3	0	0
G................	6 †	18	6	0	0
H................	1 ‡	5	1	0	0
Total........	85	413	85	14	0

* Unless otherwise indicated all rolls are 4 oz.
† Two-ounce roll.
‡ One-pound roll.

Thermal death time tubes were made by sealing off one end of an 8-in. section of 8-mm. soft glass tubing. After the open end of these tubes were plugged with cotton, the tubes were sterilized in the autoclave. Into each of these tubes 1 ml. of the cell suspension was pipetted aseptically, and the open end of the tube was then sealed. The tubes were held at room temperature for 15 minutes and then immersed in an oil bath at 80 C plus or minus 0.5 C. At 5, 10, 15, 20, 30, and 60 minutes tubes of the test organisms were removed from the bath. The tubes were opened aseptically and the cell suspension poured into a Petri dish and mixed with nutrient agar. Plates were incubated for 96 hours at 28 C and observed for growth.

The results are shown in table 2. It can be seen that no. 52 survived for 60 minutes at 80 C, while 63 survived for more than 60 minutes at 80 C. The other two cultures did not survive the shortest time interval tested. It thus appears that two of the four isolates tested can be considered heat resistant.

COMMENT

Of the 415 samples taken, 14 were shown to contain viable organisms. All samples were obtained from packages labeled "contents sterile until package is opened." In terms of rolls, 12 out of 85, or 14.2%, were contaminated. It should be emphasized again that all precautions were taken to prevent extraneous organisms from entering the bottles of sterile medium. None of the control bottles (85 in all), which were handled in the same manner as the bottles inoculated with samples, gave positive results. If the positive samples were the result of air contamination of the medium, one would expect at least a few of the controls to show the presence of viable organisms. The conclusion is forced that the cotton itself was the source of the bacteria.

TABLE 2.—*Heat Resistance of Bacterial Contaminants Isolated from Market Cotton*

Organism	Description	Plate Count	Survival Time
49	Gram-negative, short rod	630,000,000	Less than 5 min.
52	Gram-positive cocci	6,100,000	More than 30 min., less than 60 min.
58	Gram-positive rod	85,000,000	Less than 5 min.
63	Gram-negative, short rod; spore former	2,100,000	More than 60 min.

The processing temperatures of the different producers undoubtedly vary. However, we have no information available as to the times and temperatures used. The one producer who did volunteer the information was using times and temperatures that should kill all types or organisms. However, the cotton fibers are very poor conductors of heat and as such may give protection to an organism easily killed under ordinary conditions.

These findings indicate that there should be a more stringent control program on the part of both the producers and the Food and Drug Administration in the case of "sterile" market cotton. That viable organisms were found at all different levels in the roll of cotton suggests, also, the need for a rigorous investigation into the heat insulation protection afforded to bacterial life at the center of cotton, gauze, or even a hospital surgical pack. Such studies have been made on canned foodstuffs and have, in many ways, revolutionized the food-processing industry.

SUMMARY

Market cotton labeled "sterile" was found to contain viable micro-organisms. Thermal death time studies on certain of the contaminating organisms indicated that in some cases heat resistant forms were present, while other organisms were heat sensitive and must have survived the sterilization process as a result of the protective effect of the cotton fibers or because of understerilization of the product. In view of the results obtained, it is concluded that the present control program is not adequate to assure the consumer of the sterility of each package of market cotton.

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HORMONAL MANAGEMENT OF HEREDITARY HEMORRHAGIC TELANGIECTASIA

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The purpose of this paper is to report a new method [illegible] reducing the tendency to hemorrhage in patients with [illegible]editary hemorrhagic telangiectasia (Osler-Rendu dis[illegible]se). Although the periods of observation of the patients treated were of short duration, the longest having been just over one year, the results to date of writing were of sufficient interest to merit a preliminary report.

DEFINITION

The term hereditary hemorrhagic telangiectasia was first advocated by Hanes [1] in 1909. He defined this disease as a "hereditary affection manifesting itself in localized dilatations of the capillaries and venules forming distinct groups of telangiectases which occur upon the skin of the face, nasal and buccal mucous membranes, and give rise to profuse hemorrhage either spontaneously or as a result of trauma." The cardinal findings for the establishment of the diagnosis of this disease are contained in Hanes' definition: (1) the hereditary tendency toward the disease, (2) the presence of typically distributed telangiectatic areas, and (3) the tendency for the telangiectatic areas to bleed. Although the hereditary factor is usually elicited from the patient's history, Fitz-Hugh [2] was the first to call attention to certain patients with no characteristic family history. He explained this on the basis of atavism or "skipped generations" in the inheritance of this disease.

The telangiectases may occur on any of the mucous membranes or anywhere on the external surface of the body. The nasal mucous membrane is oftenest affected, with tongue, palate, lips, buccal area, gum, pharynx, and conjunctiva following in order of disease incidence. The larynx, colon, lung, stomach, liver, bladder, and brain are also affected, but owing to their relative inaccessibility the reported incidence may be less than that actually occurring. On the skin the lesions are found on the face, trunk, arms, finger tips, and nail beds. Aneurysm of the pulmonary artery has been reported by Rundles,[3] but the relation of vascular anomalies to this disease is uncertain. No evidence for a blood dyscrasia except that associated

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Drs. William MacComb, Oliver Moore, and William Money, and Miss Martha von Lackum assisted in the preparation of this paper.

The ethinyl estradiol (estinyl®) and "gynetone" (a tablet containing estradiol and methylestosterone in a 1:10 ratio) used in this study were supplied by Schering Corporation, Bloomfield, N. J.

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[1] Hanes, F. M.: Multiple Hereditary Hemorrhagic Telangiectasia Causing Hemorrhage (Hereditary Hemorrhagic Telangiectasia), Bull. Johns Hopkins Hosp. **20:** 63-73, 1909.

[2] Fitz-Hugh, T., Jr.: The Importance of Atavism in the Diagnosis of Hereditary Hemorrhagic Telangiectasia, Am. J. M. Sc. **166:** 884, 1923.

[3] Rundles, R. W.: Hemorrhagic Telangiectasia with Pulmonary Aneurysm, Am. J. M. Sc. **210:** 76-81, 1945.

with chronic blood loss has been noted. The telangiectases begin as dilatations of the capillaries and have a bright red color. As these areas increase in size, the venules participate in the ectasia, giving the cutaneous lesions a violaceous color. The telangiectases of the mucous membranes are always a brilliant red. According to Hanes,[1] the papillae of the corium are obliterated, with loss of the undulations of the stratum germinativum. Immediately subjacent to the attenuated epidermis are wide blood spaces lined by a single layer of endothelium and without either an elastic or a muscular coat.

CLINICAL SUMMARY

The commonest symptom of the disease is epistaxis, but other mucous membranes or the skin may be the exclusive sites of hemorrhage. Hematemeses, hemoptyses, melena, hematuria, and cerebrovascular accidents may occur. Anemia often results from the long-continued blood loss, and fatal hemorrhages have occurred. Trauma is not the only cause of bleeding, for it may occur spontaneously or may arise from irritation or congestion. The telangiectases usually do not appear before the age of puberty. In the third and fourth decades of life they become more numerous and more subject to hemorrhage.

Treatment of this condition has been well summarized by Peluse.[4] Medical management has largely been directed to the control of frequent severe epistaxes. The measures used include the application of caustic agents to the nasal mucosa, submucosal injections of sclerosing solutions such as quinine, ethyl carbamate, and sodium psylliate (sylnasol®), or the use of hemostatic yarn or gauze as nasal packing. Surgical amelioration has been directed to the ligation of the external carotid arteries or anterior ethmoidal arteries and to electrocauterization of the nasal septum and turbinates. X-ray therapy and radium applications to the nasal passages have been of no benefit and have often produced atrophic rhinitis and frequently have caused the sloughing of large areas of the nasal septum. General measures include the injection of moccasin snake venom to increase blood coagulability, and, more recently, favorable reports have followed the use of vitamin P (rutin).[4] Change of climate has been advised in an effort to decrease the incidence of acute coryza with associated nasal hemorrhages. Whole blood transfusions and iron replacement therapy have been required constantly to correct the secondary anemia that usually occurs. No single therapeutic agent or surgical approach, or combination of them, has heretofore consistently decreased the incidence or severity of recurrent hemorrhage. Below are presented the case reports of five patients with hereditary hemorrhagic telangiectasia whose tendency to bleed was reduced with estrogen therapy.

REPORT OF CASES

Case 1.—A 44-year-old woman had a familial history of hereditary hemorrhagic telangiectasia. Her epistaxes began just prior to her menarche at the age of 15. For many years thereafter epistaxes occurred during the five days prior to the onset of menstruation, then disappeared on the first day of her menstrual period. Many local therapeutic measures, including electrocautery, sodium morrhuate injections, submucous resection, and radium implantation, were unsuccessful in alleviating her condition. In 1948, when she was 42, roentgen castration was performed in an attempt to control the epistaxes. There was an immediate radiation reaction, characterized by nausea, vomiting, and cramping abdominal pain. No menstrual periods occurred thereafter. Immediately following this artificial menopause, the patient noted an increase in the severity and incidence of the epistaxes. They became completely acyclic in character and remained so until the time of her admission to Memorial Center on Dec. 13, 1950.

On admission, physical examination revealed the typical telangiectases of Osler-Rendu disease distributed over the tongue, mouth, and nasal mucous membranes. The nasal mucosa was edematous, crusted, and relatively avascular (except for the telangiectatic areas). An extensive perforation of the nasal septum from previous surgery and irradiation was noted.

Laboratory studies revealed a hemoglobin level of 8.0 gm. per 100 cc., a red blood cell count of 2,900,000, and a white blood cell count of 9,500 with a normal differential count. The hematocrit was 29%. Reticulocytes were 1.1%; platelets, 397,000. Bleeding time was 1½ minutes; clotting time, 12 minutes; prothrombin time, 17.9 minutes. Urinalysis was within normal limits. A urinary gonadotropic, follicle-stimulating hormone assay was positive for 104 mouse units and negative for 208 mouse units per 24 hours. (This is within the postmenopausal range by the method used.[5])

The patient received 1,000 cc. of whole blood on Dec. 15, 1950, and again on Dec. 18. The next day the hemoglobin level was 11.2 gm. per 100 cc. Because of the history of cyclic epistaxes and the increase in incidence and severity following roentgen castration, a course of orally administered ethinyl estradiol, 0.5 mg. twice daily, was started Dec. 22. A week later an examination of the nasal mucosa revealed a return to normal vascularity, a decrease in edema, and the absence of crusting or bleeding points. Epistaxes disappeared, and the patient became asymptomatic. She was discharged Jan. 5, 1951. Beginning Jan. 12 and continuing for the next week, the patient noted ankle edema and an increase in body weight. On Jan. 18 she was readmitted, and ethinyl estradiol was discontinued. An immediate diuresis occurred, and the patient remained asymptomatic until Jan. 27, when vaginal bleeding occurred. Epistaxes recurred on Feb. 2 and 4. On examination it was noted that the mucous membranes had returned to their pretherapy state. Ethinyl estradiol, 0.25 mg. per day, was given again on Feb. 5. The patient was discharged on March 1, while still receiving the drug, having been asymptomatic since Feb. 10.

Response to treatment continued until Sept. 17, 1951, when, on the advice of her local physician, the patient discontinued estrogen therapy because of low abdominal discomfort plus soreness and swelling of the right breast. The red blood cell count was 4,100,000. Heavy vaginal (estrogen deprivation) bleeding began Sept. 20 and continued through Sept. 23. On Sept. 26 epistaxes began and continued spasmodically for three days. At the first sign of nasal hemorrhage the patient resumed her daily medication of 0.25 mg. of orally administered ethinyl estradiol. Epistaxes disappeared Sept. 30 and have not recurred to the present writing.

Case 2.—A 60-year-old white man was first seen at Memorial Center on May 31, 1951. A history of repeated epistaxes of 25 years' known duration was elicited. His mother, two brothers, a son, and a daughter had all been subject to frequent and severe epistaxes. The mother and one brother died following severe hemorrhage. Three sisters of the patient had no signs or symptoms of this disease. Epistaxes began in the patient at the age of 35. They were mild, spontaneous, and recurrent every few days and were not troublesome until six years prior to his admission to Memorial Center, when they gradually became more frequent and more profuse. Local medical and surgical measures, as well as radium application to the nasal mucosa, were unsuccessful in controlling the hemorrhages.

On admission, physical examination revealed many telangiectases distributed over the face, trunk, upper extremities, tongue, and mouth. There was a large anterior perforation of the nasal septum due to previous radiation therapy. The mucous membrane of the nose was pale and edematous, with small hemorrhagic spots on the turbinates. Laboratory findings showed a hemoglobin level of 10.7 gm. per 100 cc., a red blood cell count of 3,800,000, and 247,000 platelets. The bleeding, clotting, and prothrombin times were within normal limits. A urinary gonadotropin assay was positive for 104 mouse units and negative for 208 mouse units per 24 hours, an abnormally high value for men. The 17-ketosteroids (total neutral fraction) were 9.4 and 8.0 mg. per 24 hours. Blood chemistry studies, including serum chloride, sodium, potassium, total protein, cephalin flocculation, and thymol turbidity tests, were all within normal limits.

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[4] Peluse, S.: Gingival Hemorrhage with Oral Manifestations in Hereditary Hemorrhagic Telangiectasia: Successful Control with a Sclerosing Agent Sylnasol: Effect of Vitamin P and Nicotinic Acid: Role in Case Reported Which Was Observed 5 Years, Arch. Otolaryng. 44: 668-685 (Dec.) 1946.

[5] Method of Kleinfelter, Albright, and Griswold in Smith, P. H.; Albright, F., and Dodge, E.: Modifications in Methods for Precipitation and Assay of Increased Amounts of Pituitary Gonadotropic Substances in Urine, J. Lab. & Clin. Med. 28: 1761-1766, 1943.

Administration of ethinyl estradiol, 0.25 mg. daily, was started June 7, 1951. There was no change in the frequency or severity of the patient's epistaxes until June 16, when a severe epistaxis occurred, and the patient was admitted to the University of Pennsylvania Hospital for nasal packing and transfusion. At that time an examination revealed a decrease in the size of the facial telangiectases. There was no change in the nasal mucous membranes.

The patient was readmitted to Memorial Center June 25, at which time the dose of ethinyl estradiol was increased to 0.5 mg. daily. Laboratory studies revealed a hemoglobin level of 12.1 gm. per 100 cc., a red blood cell count of 4,300,000, and a white blood cell count of 9,700 with a normal differential. Liver function studies, including cephalin flocculation, thymol turbidity, total protein, and albumin-globulin ratio, were within normal limits. On June 28 the nasal packing was removed without bleeding. The nasal mucous membranes were pink, glistening, and without evidence of telangiectases. The patient continued to be asymptomatic and was discharged on June 29, 1951, while still receiving therapy. One week later epistaxes recurred, then subsided spontaneously with no further recurrence.

A reexamination Aug. 20 revealed tenderness and swelling of both breasts, with loss of libido. Treatment was therefore changed to orally administered ethinyl estradiol, 0.5 mg. plus 5 mg. of methyltestosterone daily. Breast discomfort disappeared, and libido improved. On Oct. 22 the platelets were 86,000; by Dec. 10, 1951, they had increased to 170,000 without change in medication. The patient has remained asymptomatic to the present writing.

Case 3.—A 47-year-old white woman was admitted to Memorial Center in March, 1951, complaining of frequent epistaxes and melena accompanied by abdominal cramps and weakness. Both her grandmother and mother had telangiectases of the face and finger tips and also frequent episodes of epistaxis. The patient had occasional epistaxes in childhood. Her menarche occurred at 12 years of age. No unusual bleeding episodes were noted during the years of regular cyclic ovarian function. In 1936, when she was 32 years of age, a hysterectomy and bilateral salpingo-oophorectomy were performed for uterine fibroids, following which menopausal symptoms appeared. Shortly after this procedure, facial and thoracic telangiectases developed.

The patient was first seen at Memorial Center in 1939, at which time a diagnosis of Osler-Rendu disease was made and the facial lesions were treated with dry ice. Nasal hemorrhages were not incapacitating until 1947, when very frequent epistaxes developed that were refractory to all medical and surgical measures. In 1949 irregular gastrointestinal bleeding developed. The patient was readmitted to Memorial Center in February, 1951, because of incapacitating nasal and gastrointestinal bleeding. Following discharge, this patient was referred to the experimental hematology section for consideration of estrogen therapy.

On March 7, 1951, physical examination revealed the typical telangiectases of Osler-Rendu disease. They were distributed over the face, trunk, and upper extremities. The nasal mucous membrane was pale, atrophic, and crusted with blood. Examination was, otherwise, noncontributory. Laboratory studies on March 7 revealed a hemoglobin level of 12.3 gm. per 100 cc., a red blood cell count of 4,500,000, and a white blood cell count of 6,300 with a normal differential count. The platelet count was 360,000. The bleeding, clotting, and prothrombin times were within normal limits. A urinary gonadotropin assay on March 30 was positive for 104 mouse units and negative for 208 mouse units, a normal value for a postmenopausal woman.

On March 13, therapy with orally administered ethinyl estradiol, 0.25 mg. daily, was started. Within 10 days after institution of hormone therapy, epistaxes and gastrointestinal bleeding diminished. In one month a distinct reduction in number, size, and erythema of cutaneous telangiectases was evident. In May, 1951, two months after the institution of hormone therapy, a cyclic pattern in treatment response was noted. While receiving orally administered ethinyl estradiol, 0.25 mg. daily, the patient was in comparatively good health for three weeks. With no change in dosage, spontaneous diuresis occurred, followed in a few hours by exacerbation of bleeding and increased prominence of the telangiectases. The episode lasted for approximately one week and was followed by a three week period of favorable response. The patient was hospitalized from June 18 through June 27 for a trial period without treatment. There was prompt return of severe bleeding and telangiectases.

In August, 1951, an episode of diuresis recurred. At the same time increased epistaxes and increased prominence of telangiectases were noted. The dose of ethinyl estradiol was temporarily increased to 0.25 mg. twice daily, with prompt amelioration of these signs and symptoms. Ethinyl estradiol, 0.25 mg. daily, was resumed and was effective for the subsequent three weeks. The patient has been taught to recognize this cyclic change in response, and she varies the dosage promptly, with resultant minimal signs and symptoms.

Case 4.—A 53-year-old white man had three or four episodes of epistaxis in childhood. During the 15 years preceding admission the epistaxes had become increasingly severe, with episodes one to three times daily. About 10 years preceding admission he had had radium treatment to the nasal cavity. Since then the nasal hemorrhages had been treated by chemical and electrical cauterizations and, more recently, by ligations of the anterior ethmoidal and external carotid arteries. None of these measures had reduced the bleeding. Most of his hemorrhages occurred at night and were preceded by prodromes of nuchal pain and fulness in the frontal region. No family history of bleeding tendency was elicited.

Physical examination revealed typical stigmas of hereditary hemorrhagic telangiectasia of the face and violaceous lesions of the tongue and hard palate. The nasal mucosa was edematous and pale, with scattered lesions on the septum and turbinates and marked crusting. The heart was enlarged both clinically and roentgenologically, although the lung fields were clear. Abdominal examination revealed a palpable liver edge 2 cm. below the costal margin in the right midclavicular line (though dietary history was adequate and alcoholic intake was denied). Rectal examination revealed a symmetrically enlarged, boggy prostate. Laboratory studies prior to therapy revealed a red blood cell count of 4,200,000, a white blood cell count of 5,300, a platelet count of 256,200, and a hemoglobin level of 11.7 gm. per 100 cc. Bleeding time was 2 minutes; clotting time, 10 minutes; prothrombin time, 16.1 minutes. Liver function studies showed a sulfobromophthalein (bromsulphalein®) sodium retention of 23%, while cephalin flocculation was negative in 24 hours and 1+ in 48 hours. Thymol turbidity was 1.29 units. Total protein was 7.0 gm., with a normal albumin-globulin ratio. Acid phosphatase was 0.2 Bodansky units, and the alkaline phosphatase was 13.6 Bodansky units. Urinary gonadotropin assay was positive for 6.5 and negative for 13 and 52 mouse units per 24 hours. (This is a normal value for the male by this method.[5]) The 17-ketosteroids (total neutral fraction) were 7.4 mg. per 24 hours.

On July 10, 1951, therapy with orally administered ethinyl estradiol, 0.25 mg. daily, was started. There was no change in the incidence or severity of epistaxes during the first two weeks of treatment. During the next two weeks epistaxes gradually diminished in volume and by Aug. 6 had ceased. At that time, however, the patient complained of lassitude, loss of libido, breast tenderness, dyspnea, and ankle edema. Treatment was changed on that date to ethinyl estradiol, 0.5 mg. plus methyltestosterone 5.0 mg. daily. In addition, he was placed on a low salt diet. He was next seen on Aug. 20, by which time breast tenderness, dyspnea, ankle edema, and loss of libido had disappeared. Also, a decrease in the size and number of telangiectases was noted. There was no change in the size of the liver, the edge of which was still palpable 2 cm. below the costal margin.

of ampullary carcinoma have been studied. The serum amylase determination was of value in the diagnosis of carcinoma of the head of the pancreas in three out of four cases (table 3). In two cases there was a significant amylase elevation, while in one there was a significant depression of the amylase titer, compatible with extensive atrophy of the acinar cells, which was confirmed by histiological examination. In cases of carcinoma of the body and tail, serum amylase values proved to be of no diagnostic value. In one case of ampullary carcinoma there was an elevated amylase level (case 10, table 3).

Table 3.—*Serum Amylase Levels in Cases of Carcinoma of the Pancreas and Ampulla of Vater*

	Case No.	Maximum Serum Amylase Level (Mg./100 Cc.)	Comment
Carcinoma of the head	1	1,426	Jaundiced; surgery confirmed diagnosis
	2	959	Jaundiced; autopsy confirmed diagnosis
	3	147	Jaundiced; lesion localized to head without obstruction of pancreatic duct
	4	82	Jaundiced; inoperable
	5	48	Jaundiced; extensive atrophy of pancreas
Carcinoma of the body or tail	6	115	Not jaundiced; confirmed at surgery (body)
	7	206	Not jaundiced; autopsy revealed lesion of tail
	8	100	Not jaundiced; surgery revealed lesion of body
	9	292	Not jaundiced; autopsy revealed lesion of body
Carcinoma of the ampulla of Vater	10	973	Jaundiced; confirmed at surgery
	11	107	Jaundiced; confirmed at surgery
	12	87	Jaundiced; confirmed at surgery

It appears to me, after studying these 12 cases, that serum amylase determinations may be of some diagnostic value in the early detection of neoplasms of the head of the pancreas and of the ampulla of Vater. However, a normal serum amylase level in a patient with obstructive jaundice contributes nothing to the perplexing problem of differentiating between a benign and a malignant lesion as the basis for the obstruction.

Peptic Ulcer.—The intimate anatomic and physiological relationship of the gastroduodenal tract to the pancreas readily explains the elevated amylase levels that are frequently encountered in cases of penetrating and perforated peptic ulcers. In 1934, Hinton [21] suggested that the study of pancreatic enzymes could be of diagnostic value in cases of duodenal ulcers penetrating into the pancreas. A few years later, Probstein and coworkers [6a] reported four cases of peptic ulcer involving the pancreas in which the blood amylase was found to be moderately elevated. Subsequent reports [22] have established the diagnostic value of elevated amylase values in penetrating peptic ulcers.

My interest in the serum amylase value in cases of penetrating peptic ulcer was stimulated by the need for a simple and reliable test to confirm the clinical impression that a previously uncomplicated duodenal ulcer was penetrating into the pancreas, as strongly suggested by the change in the character of the pain. Since this study was undertaken, nine cases of penetrating peptic ulcer have been collected in which the serum amylase level was found to be elevated. The clinical diagnosis was confirmed at surgery in six of these cases. In a fairly large number of controls (uncomplicated, active peptic ulcer) serum amylase values consistently were found to be within normal limits.

Acute gastroduodenal perforation, without pancreatic involvement, occurring within a few hours after a meal, is frequently associated with an elevated amylase level.[23] This was demonstrated by Pemberton [6c] in his experimental work on dogs. The source of this elevated serum amylase appears to be the pancreatic amylase, which has spilled from the intestine into the peritoneum and reabsorbed into the circulation. My experience with serum amylase values in cases of duodenal perforations is very meager. One such case was studied in which an erroneous diagnosis of acute hemorrhagic pancreatitis was made on the basis of a serum amylase level of 2,000 mg. per 100 cc. Postmortem examination revealed a duodenal perforation with bile peritonitis, with a normal pancreas grossly and microscopically. The common bile duct and pancreatic duct were patent and free from calculi.

Diseases of the Salivary Glands.—It has been conclusively demonstrated that disease of the salivary glands, especially mumps, are associated with elevated serum amylase levels.[24] Such cases rarely present diagnostic problems, and the elevated serum amylase is of interest only because of its source. A few cases of mumps were studied, and in all there were elevated serum amylase levels, ranging from 578 mg. to 950 mg. per 100 cc. These elevations usually reached a maximum concentration by the fourth day of the illness. It was of interest to note that none of the patients with elevated amylase levels had abdominal symptoms.

Renal Disease.—One of the mechanisms that partakes in the "regulation" of the normal amylase level of the serum is renal excretion of amylase. When renal function is impaired, there is retention of amylase in the blood as reflected by elevated values, along with diminished excretion by the kidneys.[25] The level of serum amylase in cases of renal insufficiency seldom reaches the elevations seen in acute pancreatic lesions. My experience in a small number of cases of renal disease with azotemia has shown the serum amylase to be moderately elevated, ranging between 370 and 480 mg. per 100 cc. It was of interest

Table 4.—*Serum Amylase Levels in Penetrating Peptic Ulcer*

Patient	Maximum Concentration of Serum Amylase (Mg./100 Cc.)
V. R.	976
W. B.	462
C. M.	540
J. B.	598
D. H.	327
U. G.	543
R. C.	425
J. F.	439
S. M.	300

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21. Hinton, J. W.: Chronic Pancreatitis Associated with Peptic Ulcer, Arch. Surg. **28**: 580 (March) 1934.

22. Heifetz, Probstein, and Gray.[6b] Polowe, D.: Blood Amylase, Am. J. Clin. Path. **13**: 288 (June) 1943.

23. Pemberton, Grindlay, and Bollman.[6c] Musgrove.[6d]

24. Dunlop, G. A.: The Diastatic Index in Acute Parotitis, Lancet **2**: 183 (July 22) 1933. Candel, S., and Wheelock, M. C.: Serum Amylase and Serum Lipase in Mumps, Ann. Int. Med. **25**: 88 (July) 1946.

25. Myers, V. C., and Killian, J. A.: Studies in Animal Diastasis: The Increased Diastatic Activity of the Blood in Diabetes and Nephritis, J. Biol. Chem. **29**: 179 (March) 1917. Harrison, G. A., and Lawrence, R. D.: Diastase in Blood and Urine as a Measure of Renal Efficiency, Lancet **1**: 169 (Jan. 27) 1923. Gray, S. H., and Somogyi, M.: Relationship Between Blood Amylase and Urinary Amylase in Man, Proc. Soc. Exper. Biol. & Med. **36**: 253 (April) 1937.

to note in these cases that as the serum amylase rose there was a parallel rise in the blood urea nitrogen.

Adequate evaluation of moderate elevations of serum amylase requires consideration of the status of renal function. The temporary impairment of renal function frequently encountered in acute hemorrhagic pancreatitis does not alter the significance of the elevated value, for in this acute disease the amylase values are well over 1000 mg. per 100 cc. In cases of chronic renal insufficiency, the serum amylase value usually is under 500 mg. per 100 cc. and rarely poses any problem in the interpretation of this moderate elevation.

Table 5.—*Serum Amylase Levels in Cases of Liver Disease*

Patient	Diagnosis	Initial Fasting Serum Amylase Level	Range of Amylase Levels (Serial Determinations)	
			Minimum Concentration	Maximum Concentration
M. M.*	Toxic hepatitis	262	152	254
A. S.	Cirrhosis	158	...	...
A. G.*	Cirrhosis	125	97	132
C. L.	Cirrhosis	130	68	157
P. W.	Fatty infiltration	80	80	276
E. D.*	Hepatitis	109	81	113
C. F.*	Cirrhosis	91	64	157
A. M.	Fatty infiltration	80	80	110
O. D.	Cirrhosis	145	71	167
H. F.*	Cirrhosis	176	126	150
R. H.*	Cirrhosis	152	145	199
C. M.*	Cirrhosis	256	245	309
L. T.	Cirrhosis	232	181	260

* Jaundiced.

Diseases of the Hepatobiliary Tract.—In view of the varied reports [26] on the significance of serum amylase values in cases of hepatic disease, bile duct calculi, cholecystitis, and cholelithiasis, it seemed desirable to evaluate the diagnostic value of serum amylase determination in such conditions.

Liver Disease: Comfort [26a] found normal values for serum amylase in 22 cases of hepatic disease. Gray and co-workers [26c] reported that impaired hepatic function due to liver disease of various types is usually associated with a subnormal level. It seems difficult to offer a valid explanation for a subnormal amylase level in cases of liver disease, unless severe pancreatic insufficiency, either functional or organic, is concomitantly present.

Thirteen cases of liver disease were studied, and my observations are of interest only in that they confirmed the impression that serum amylase levels are within normal limits in cases of disease of the liver. In all cases there was normal pancreatic function as determined by function tests. The serum amylase values observed in this small series of cases of liver disease are expressed in table 5. The serum amylase levels seen in these cases did not confirm the belief that liver disease is associated with low or subnormal amylase values.

Cholecystic Disease: Cholecystic disease is also considered to be associated with a subnormal or low amylase value. This appears most unlikely; if any deviation from normal is to be found, one would anticipate an elevation in light of the frequent associated cholecystic disease in cases of acute and chronic recurrent pancreatitis. Normal serum amylase values were obtained in four cases of obstructive jaundice due to common duct calculi and in two cases of common duct stricture secondary to surgical intervention. One patient (E. J., table 6) with a common duct calculus had a serum amylase level of 333 mg. per 100 cc.; the pancreas was reported by the surgeon to have a "normal" appearance.

Twenty-eight patients with cholecystic disease had repeated serum amylase determinations while in the hospital for a painful attack. The significant serum amylase values obtained are shown in table 6. The clinical diagnosis was confirmed at surgery in 24 cases and by postmortem examination in one case. Clinical and roentgenologic evidence established the diagnosis in the remaining three cases. Serum amylase values in all but one case (E. J.), were within normal limits; the lowest value obtained was 64 mg. per 100 cc.

The amylase values obtained in these 28 cases do not reflect any tendency toward "subnormal" values as is generally believed to be found in cholecystic diseases. However, I am of the opinion that serum amylase determinations in patients with known cholecystic disease and repeated episodes of acute painful seizures may be of diagnostic value in the detection of recurrent pancreatitis. Frequently, the diagnosis of chronic recurrent pancreatitis is missed, and in these cases the condition is usually mistaken for some form of biliary tract disease.

Table 6.—*Serum Amylase Values in Cases of Cholecystic Disease*

Chronic Cholecystitis with Cholelithiasis		Chronic Cholecystitis with Common Duct Calculi		Chronic Cholecystitis Without Calculi	
Patient	Amylase Level*	Patient	Amylase Level*	Patient	Amylase Level*
S. N.	202	A. B.	133	O. B.	129
F. J.	144	L. M.	260	D. P.	211
F. R.	160	O. F.	68	H. S.	115
C. G.	255	E. J.	333		
M. S.	120	M. W.	110		
E. K.	190				
D. K.	64				
D. M.	89				
M. I.	195				
C. F.	140				
V. F.	152				
V. C.	102				
C. A.	156				
R. H.	150				
M. C.	102				
L. S.	99				
N. S.	112				
M. J.	191				
M. B.	206				
M. C.	159				

* Milligrams per 100 cc.

Recently, an interesting observation was reported by Gross and co-workers,[27] on a small series of patients with biliary dyskinesia. They noted an elevation in the serum amylase level after the administration of opiates

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26. (*a*) Comfort and Osterberg.[3f] Heifetz, Probstein, and Gray.[6b] (*b*) Somogyi, M.: Blood Diastase as an Indicator of Liver Function, Proc. Soc. Exper. Biol. & Med. **32**:538 (Dec.) 1934; Diastatic Activity of Human Blood, Arch. Int. Med. **67**:665 (March) 1941. (*c*) Gray, S. H.; Probstein, J. G., and Heifetz, C. J.: Clinical Studies on Blood Diastase: Low Blood Diastase as an Index of Impaired Hepatic Function, Arch. Int. Med. **67**:805 (April) 1941. (*d*) Branch, C. D., and Zollinger, R.: The Value of Blood Diastase in the Diagnosis of Common Duct Stone, Am. J. Surg. **41**:233 (Aug.) 1938.

27. Gross, J. B.; Comfort, M. W.; Mathieson, D. R., and Power, M. H.: Elevated Values for Serum Amylase and Lipase Following the Administration of Opiates: A Preliminary Report, Proc. Staff Meet., Mayo Clin. **26**:81 (Feb. 28) 1951.

to such patients. The mechanism for this is the resulting spasm of the sphincter of Oddi induced by administration of morphine or codeine.[27] I have had no opportunity to study the serum amylase response in patients with biliary dyskinesia, but I have seen one patient with biliary tract disease in which spasm of the sphincter of Oddi apparently was induced by meperidine (demerol®) hydrochloride resulting in a significant rise in serum amylase (714 mg. per 100 cc.). When meperidine was not given, subsequent serum amylase values remained within normal limits. At this hospital, we do not administer morphine and allied compounds to patients undergoing serum amylase determinations, especially to those patients with abdominal pain and suspected biliary tract and/or pancreatic disease.

SUMMARY AND CONCLUSIONS

The usefulness and importance of serum amylase determination in acute pancreatic lesions has had clinical recognition since Elman's tremendous contribution to this field. Despite the diagnostic value of serum amylase in pancreatic disease, this simple informative test is frequently not utilized.

Significant elevations of serum amylase occur within a matter of hours in cases of acute pancreatitis, and maximum concentrations are observed within 12 to 24 hours. There appears to be no constant relationship between the height of the amylase level and the severity of the attack; however, it appears that serial serum amylase determinations furnish invaluable information as to the activity and duration of the acute process in the pancreas. The serum amylase test has proved to be invaluable in cases of chronic recurrent pancreatitis. Twenty-five cases of chronic recurrent pancreatitis with hyperamylasemia during an acute exacerbation were collected and are reported in this communication.

The diagnostic value of the serum amylase test in cases of carcinoma of the pancreas and ampulla of Vater was determined in a small series. It was found to be of some value in cases of carcinoma of the head and of the ampulla. In cases of carcinoma of the body and tail, serum amylase levels were of normal value, as one would anticipate in view of their location and the uninvolvement of the main pancreatic duct.

Certain types of gastroduodenal lesions are associated with elevated serum amylase levels, especially in cases of duodenal perforations without pancreatic involvement. One such case was studied and is herein reported. Penetrating peptic ulcers frequently involve the pancreas and are readily diagnosed clinically on the basis of the moderate elevations in serum amylase. Nine such cases were collected and are included in this report. Other clinical conditions associated with elevated serum amylase values are diseases of the salivary glands and renal insufficiency. From the observation of elevated serum amylase levels in these conditions one readily realizes that elevated amylase values are not pathognomonic of primary pancreatic disease. However, critical evaluation of the clinical findings, along with the height of amylase elevation, will enhance the usefulness of the serum amylase test and not detract from its diagnostic value.

The previous belief that liver disease and cholecystic disease are associated with "low" or "subnormal" serum amylase levels could not be confirmed. Serum amylase determinations in the cases herein reported almost consistently revealed values within normal limits.

I am of the opinion that significant elevations reflect a pathological increase of either pancreatic amylase or salivary ptyalin because of disease of the pancreas or salivary gland. The elevated values encountered in cases of various abdominal conditions other than primary pancreatic disease are indicative of associated or secondary pancreatitis or excessive absorption of the "exocrine" pancreatic amylase, such as occurs in duodenal perforations. The slight to moderate elevations encountered in cases of renal insufficiency are readily understood.

Serum amylase determinations routinely should be made in all patients with acute abdominal pain. Elevated values in such patients invariably are of diagnostic value when correlated with other clinical findings. Critical evaluation of an elevated serum amylase enhances the diagnostic value of this test.

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CHROMIUM INTOXICATION

WITH SPECIAL REFERENCE TO HEPATIC INJURY

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Although chromium compounds have been known for many years to be toxic to various organs of the body, primary hepatic injury has not been a conspicuous clinical finding. The organs most frequently injured by these agents appear to be those that come in direct contact with them, either by external exposure or by excretion, namely, the skin, the mucous membranes, and the kidneys.

A variety of surface lesions have been ascribed to contact with soluble chromium compounds. As early as 1827, Cumin[1] described the characteristic "chrome hole" lesions. These occur on exposed parts of the skin, especially the hands, and are punched out, deeply penetrating, painless ulcers.[2] Macular erythema, scarlatinoid

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Dr. C. D. Proctor performed the polarographic determinations of chromium in the urine specimens.

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[1] Cumin, W.: Remarks on the Medicinal Properties of Madar, and on the Effects of Bichromate of Potass on the Human Body, Edinburgh M. J. 28:295-302, 1827

and pustular eruptions, and eczematoid lesions of the skin due to contact with these substances have also been reported.[3] Ulceration and perforation of the nasal septum due to inhalation of plating solution fumes have been described frequently.[4] There is little surrounding mucosal inflammation, but there may be pronounced hyperemia. Painless inflammation of the oral mucous membranes, perforating ulcers of the palate, and ulcers of the eyelids, conjunctiva, and cornea also occur.[5] Inhalation of chromium compounds has been implicated in the pathogenesis of certain cases of pneumoconiosis, allergic asthma, and respiratory tract carcinoma.[6] Inflammation of the respiratory tree has been produced in animals by inhalation of chromic acid dust.[7]

Ingestion of chromium compounds, usually potassium bichromate, leads to severe vomiting, thus preventing much absorption. Nevertheless, instances of systemic poisoning due to absorption of chromium from the gastrointestinal tract and even from the skin have been reported in humans and produced in experimental animals.[8] Once absorbed, the compounds are eliminated by the kidneys primarily and in the bile and feces. Experimentally, it has been shown that 60% of a single dose of chromium is eliminated in the urine within the first eight hours.[8g] This would account for the frequency of uremia in cases of acute poisoning.[9] In humans the tubules are the primary site of injury, whereas in animals glomerular as well as tubular damage has been produced.[10] Other regions reported to be injured by chromium are the central nervous and hematopoietic systems.[11] Hepatic abnormalities have been reported only occasionally, for the most part in accidental human poisonings due to unusual exposure to chromates or in the experimental intoxication of animals. Hepatomegaly without jaundice was noted in a person poisoned acutely, and fatty livers have been found at autopsy in cases of acute and chronic intoxication.[12] An experimental study of chronic chromium poisoning in rabbits demonstrated liver cell degeneration, inflammatory cell infiltration, Kupffer cell proliferation, and large amounts of chromium in the liver.[8g]

The existence of hepatic injury due to the usual industrial exposure to chromium has not been recognized. Surveys of persons in contact with these compounds, for example, in the electroplating, aircraft, and shipbuilding industries, have emphasized the skin and mucous membrane lesions but have not found clinical evidence of systemic poisoning.[13] Blair [4g] stated, "There is no evidence, from the literature or otherwise, that any injury from contact with chromic acid solution or spray in chromium plating develops into a constitutional disease."

We recently had the occasion to study an employee of a chromium plating plant, who had been admitted to the Cook County Hospital because of jaundice. Urinary chromium excretion was found, and the results of hepatic tests and findings on liver biopsy were compatible with the diagnosis of acute hepatitis. In an attempt to determine whether there is a relation between industrial exposure to chromium and liver injury, eight fellow workers were screened; in four, significant urinary chromium excretion was found. The four were studied by a series of hepatic tests and, but for one, by needle biopsy of the liver. Because of the widespread industrial use of chromium at present, these cases are reported to call attention to the possibility of liver injury due to subtle intoxication with these compounds.

METHODS

Twenty-four hour urine specimens were digested with nitric and sulfuric acids and evaporated to dryness. The residue was quantitatively analyzed for chromium, as chromate, by polarography.[14] The hepatic tests performed are listed in tables 1 and 3. Liver biopsy was performed by use of the Vim-Silverman needle. The tissue was fixed in formaldehyde solution, and sections were stained with hematoxylin-eosin, aniline blue, trichrome, iron, hemofuchsin, and silver stains.

REPORT OF CASES

CASE 1.—A. J., a 36-year-old Negro woman, was admitted to Cook County Hospital on Feb. 9, 1951. She had been employed in a chromium plating factory continuously for five years, working next to a chromic acid bath. For the past year she experienced intermittent abdominal swelling. Three weeks prior to admission, she noted pruritus and anorexia, which became increasingly severe. Two weeks later icterus appeared, and the stools became lighter and the urine darker in color. These symptoms persisted until admission. There was no history of alcoholism or exposure to other known toxins.

She was well developed physically, in no apparent discomfort, and afebrile. The skin was excoriated mildly, especially over the back. The scleras were moderately icteric. A few lymph nodes were palpable in the neck, axillas, and groin. The nasal mucosa was intact. The lungs and cardiovascular system were normal. The level of the abdomen was 2 cm. above the xiphopubic line. Neither liver nor spleen was palpable, and ascites was not demonstrable.

Urinalysis revealed a specific gravity of 1.025, no albumin or sugar, 2+ bilirubin, and 2+ urobilinogen. Within two days bilirubin was absent from the urine and urobilinogen excretion increased to 4+; by the 16th day, the latter had returned to nor-

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5. Schwartz.[2b] Sutton and Sutton.[3e]

6. (*a*) Letterer, E.; Neidhardt, K., and Klett, H.: Chromatlungenkrebs und Chromatstaublunge, Arch. f. Gewerbepath. u. Gewerbehyg. **12**: 323-361 (Sept.) 1944. (b) Lukanin, W. P.: Zur Pathologie der Chromat-Pneumokoniose, Arch. f. Hyg. **104**: 166-174, 1930. (c) Joules, H.: Asthma from Sensitisation to Chromium, Lancet **2**: 182-183 (July 23) 1932. (*d*) Machle, W., and Gregorius, F.: Cancer of the Respiratory System in the United States Chromate-Producing Industry, Pub. Health Rep. **63**: 1114-1127 (Aug. 27) 1948.

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mal. The hemogram showed 80% hemoglobin, 4,210,000 red blood cells per cubic millimeter, 6,100 white blood cells per cubic millimeter, 76% polymorphonuclear leukocytes, 1% eosinophils, 14% lymphocytes, 9% monocytes, and a few target cells. The blood Wassermann reaction was negative. The chest x-ray was normal.

TABLE 1.—*Results of Hepatic Tests in Case 1*

Test *	2/12/51	2/19/51	2/26/51	3/6/51	3/19/51
Icterus index (4-6 units)	95	25	12	28	17
Cephalin cholesterol flocculation (0-1+)	2+	4+	2+	0	0
Thymol turbidity (1.0-4.0 units)	20.8	9.4	6.3	6.8	5.4
Total protein (6.5-8.0 gm./100 ml.)	7.2	7.6	6.7	7.0	8.2
Albumin (4.0-5.5 gm./100 ml.)	4.0	5.2	..	5.0	5.2
Globulin (1.5-3.4 gm./100 ml.)	3.2	2.5	..	2.0	3.0
Gamma globulin † (0.6-1.25 gm./100 ml.)	1.89	2.02	1.85	1.4	1.57
Total cholesterol (150-250 mg./100 ml.)	258	270	262	205	265
Cholesterol ester/total cholesterol 0.50-0.75	..	..	0.41	..	..
Alkaline phosphatase (1.5-4.0 Bodansky units)	4.8	3.8	2.0	3.1	1.9
Inorganic phosphorus (3.0-5.0 mg./100 ml.)	4.0	3.4	3.2	4.1	3.5
Nonprotein nitrogen (20-40 mg./100 ml.)	30	30	..	..	..
Sulfobromophthalein retention, 2 mg./kg. dose (0-6%)	..	..	..	..	30

* Normal values in our laboratory are given in parentheses.

† de la Huerga, J., and Popper, H.: Estimation of Serum Gamma Globulin Concentration by Turbidimetry, J. Lab. & Clin. Med. 35: 459-465 (March) 1950.

The hospital course was one of gradual improvement, with disappearance of visible icterus in 16 days. Twice the patient had episodes of abdominal distention, vague abdominal discomfort, and headache, which lasted 24 to 48 hours and subsided without therapy other than analgesics. X-ray of the abdomen showed moderate amounts of gas in the large bowel during these episodes. Initial gastrointestinal x-rays showed findings suggestive of irritable colon, which later disappeared. The gallbladder was normal.

The results of the hepatic tests are tabulated in table 1. Initially the icterus index was 95 units, the thymol turbidity 20.8 units, and the cephalin-cholesterol flocculation 2+ (later 4+). Serum globulin, especially the gamma fraction, was moderately elevated. Serial determinations showed gradual return toward normal. Five weeks after admission sulfobromophthalein retention was 30%. Qualitative examination of the 24-hour urine on the fourth hospital day was strongly positive for chromium. Subsequent quantitative analyses of 24-hour urine specimens, before and after the use of dimercaprol injection, are recorded in table 2.

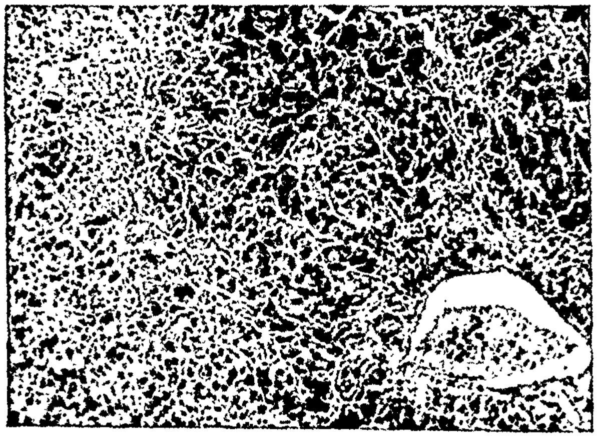

Fig. 1 (case 1).—There is marked centrolobular necrosis and Kupffer cell proliferation. The enlarged portal field is infiltrated with lymphoid and histiocytic cells and shows moderate bile duct proliferation. (× 120)

Biopsy of the liver was performed on the eighth hospital day. Histologically, there was necrosis of the liver cells of the central zone with dilatation of the sinusoids. The few liver cells preserved in the zone had eosinophilic cytoplasm, and the nuclei were markedly pyknotic or absent. The Kupffer cells were increased in number and size. There were focal accumulations of lymphoid type cells within the sinusoids or in place of liver cells. Bile pigment was noted chiefly in liver cells in the central zone, but occasionally elsewhere. The portal fields were moderately enlarged and showed lymphocytic and histiocytic infiltration and a moderate degree of mature bile duct and capillary proliferation. No fat was seen (fig. 1 and 2).

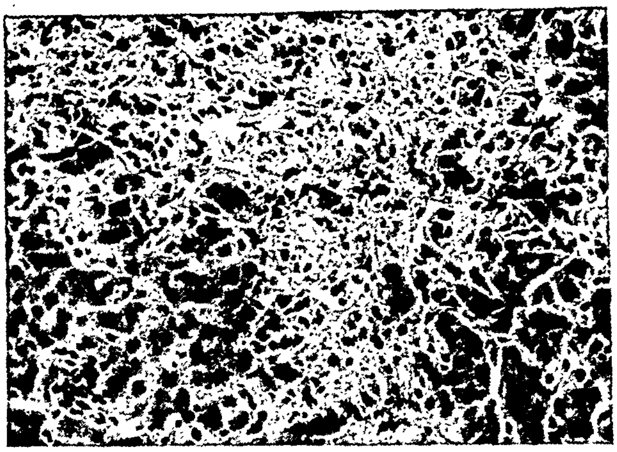

Fig. 2 (case 1).—There is necrosis of the liver cells in the center of the lobule. The cells that remain show variation in size and in nuclear and cytoplasmic staining. Marked proliferation of Kupffer cells and focal accumulations of lymphoid and histiocytic cells can be seen. (× 240)

TABLE 2.—*Amount of Chromium Recovered in Urine of Five Chromium Workers*

Case No.	Date	Chromium in Urine, Mg./24 Hr.
1	2/13/51	++++ ‡
	2/24/51	0.000
	3/14/51	0.739
	3/18/51	1.480
	3/19/51 *	0.630
	3/20/51 †	0.135
2	3/ 4/51	2.880
3	3/ 4/51	2.840
	3/18/51	0.186
4	3/ 4/51	0.495
5	3/ 4/51	0.184

* The patient received 300 mg. of dimercaprol injection intramuscularly during collection of this specimen.

† The patient received 200 mg. of dimercaprol injection intramuscularly during collection of this specimen.

‡ Qualitative only.

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8. (*a*) Philipson: A Case of Poisoning by Bichromate of Potash, Lancet **1**: 138-139 (Jan. 16) 1892. (*b*) Brieger, H.: Zur Klinik der akuten Chromatvergiftung, Ztschr. f. exper. Path. u. Therap. **21**: 393-408 (Nov.) 1920. (*c*) Major, R. H.: Case of Chromic Acid Nephritis, Bull. Johns Hopkins Hosp. **33**: 56-61 (Feb.) 1922. (*d*) Goldman, M., and Karotkin, R. H.: Acute Potassium Bichromate Poisoning, Am. J. M. Sc. **189**: 400-403 (March) 1935. (*e*) Sander, J. F., and Camp, C. D.: Chromium Poisoning in Infancy, ibid. **198**: 551-554 (Oct.) 1939. (*f*) Lewin, L.: Ueber die gewerbliche Vergiftung mit Chromverbindungen, Ztschr. f. Gewerbehyg. **15**: 159-162, 1908. (*g*) Bianchi, G.: Sulla intossicazione da cromo, Turin, Attualita di Scienze Mediche, 1941, p. 48.

9. Brieger.[8b] Major.[8c] Goldman and Karotkin.[8d]

10. Galloro.[7] Brieger.[8b] Hunter, W. C., and Roberts, J. M.: Experimental Study of the Effects of Potassium Bichromate on the Monkey's Kidney, Am. J. Path. **9**: 133-148 (Jan.) 1933. Hasegawa, M.: Studies on the Detoxicating Hormone of the Liver (Yakriton): Effect of Yakriton upon Chromic Nephritis, Tohoku J. Exper. Med. **32**: 163-176 (Jan. 30) 1938. Ohta, F.: Studies on the Detoxicating Hormone of the Liver (Yakriton): Contribution to the Usage of Yakriton against Experimental Chromate Nephritis, ibid. **39**: 37-46 (Sept. 30) 1940.

11. Brieger.[8b] Sander and Camp.[8e] Bianchi.[8g]

12. Letterer, Neidhardt, and Klett.[6a] Brieger.[8b] Goldman and Karotkin.[8d] McNally, W. D.: Toxicology, Chicago, Industrial Medicine, 1937.

13. Hall.[3c] Zvaifler.[4a] Bloomfield and Blum.[4e] Blair.[4g] Vaccaro, L.: Chromium Poisoning, Indust. Med. **10**: 246-247 (June) 1941. Brown, E. W.: Health Hazards from Lead Paint and Zinc Chromate Paint: Incidence in Modern Construction, Maintenance and Scrapping of Ships, Occup. Med. **5**: 739-758 (June) 1948.

14. Proctor, C. D.: To be published.

CASE 2.—J. R., a 28-year-old Negro, worked next to a chromium plating tank for four years. For the past year he noticed a metallic taste in his mouth. Six months previously nasal ulcers developed, which were successfully treated with petrolatum applications. There was no history of alcoholism or exposure to other known toxins. Abnormal physical findings included injection of the nasal and pharyngeal mucosa and tenderness over the area of liver dulness. Urinalysis, hemogram, chest x-ray, and gallbladder visualization gave normal results. The results of the hepatic tests and quantitative analyses of the urine for chromium are recorded in tables 2 and 3. Prior to liver biopsy, the prothrombin activity was 65% of normal. After 40 mg. of synthetic vitamin K was given parenterally, no significant change in prothrombin activity occurred.

Microscopically, the liver cells around the central vein contained pyknotic nuclei and a smudgy, or homogeneous, eosinophilic cytoplasm. Elsewhere, the nuclei showed moderate pleomorphism, and the cytoplasm contained fat and hydropic vacuoles. A moderate amount of bile pigment was found in the liver cells. The Kupffer cells were slightly increased in number, and scattered focal necroses were seen. The portal fields were enlarged and very cellular, and moderate capillary, bile duct, and fibroblast proliferations were noted (fig. 3).

CASE 3.—A. L. J., a 31-year-old Negro, worked in the chromium plating industry for four years. For the past two years he periodically noted soreness and redness of the nasal mucosa and had muscular aches. There was no history of alcoholism or exposure to other known toxins. There was injection of the nasal mucosa. The results of the remainder of the physical examination, as well as the urinalysis, hemogram, chest x-ray, and barium enema, were normal. The results of the hepatic tests and quantitative examination of the urine for chromium are recorded in tables 2 and 3. In the section a few of the liver cells about the central vein contained fine, granular bile pigment, and an occasional focal necrosis was noted. Otherwise, the liver was normal.

TABLE 3.—*Results of Hepatic Tests, Cases 2 to 5*

Test *	Case 2 3/7/51	Case 2 3/19/51	Case 3 3/7/51	Case 3 3/22/51	Case 4 3/7/51	Case 4 3/27/51	Case 5, 3/7/51
Icterus index (units)	9	6	11	19	6	11	6
Cephalin cholesterol flocculation	0	0	0	0	0	0	0
Thymol turbidity (units)	2.3	3.0	5.2	6.3	4.2	3.2	3.0
Total protein (gm./100 ml.)	7.0	7.6	6.7	7.2	7.9	8.1	7.2
Albumin (gm./100 ml.)	4.1	4.7	4.0	4.9	4.8	4.6	4.4
Globulin (gm./100 ml.)	2.9	2.9	2.7	2.3	3.1	3.5	2.8
Gamma globulin † (gm./100 ml.)	1.05	1.40	1.26	1.40	1.24	1.14	0.97
Total cholesterol (mg./100 ml.)	200	205	200	205	150	160	178
Cholesterol ester/total cholesterol	..	..	0.42	..	..	..	..
Alkaline phosphatase (Bodansky units)	2.4	1.8	2.4	1.9	2.0	..	2.6
Inorganic phosphorus (mg./100 ml.)	3.0	3.0	3.4	3.5	4.2	..	3.8
Sulfobromophthalein retention (%)	15.7	..	24.0	25.3	9.0	0.0	8.0

* For normal values in our laboratory, see table 1.
† de la Huerga and Popper (see reference in table 1).

CASE 4.—C. R., a 27-year-old Negro, worked in a chromium plant for one year, periodically at the chromic acid bath. He had an epistaxis one month before examination but was otherwise symptom free. Except for hyperemia of the nasal mucosa, physical findings were normal. The results of urinalysis, hemogram, chest x-ray, and gallbladder visualization were normal. The results of the hepatic tests and the quantitative examination of the urine for chromium are recorded in tables 2 and 3.

Morphologically, the cells immediately around the central veins showed smudginess and eosinophilia of the cytoplasm, pyknotic nuclei, and occasional karyorrhexis. The Kupffer cells were increased in number and often increased in size. There were foci of lymphocytes within the sinusoids and occasionally in place of liver cells. The portal fields were small and contained a slight amount of lymphocytic infiltration. A few liver cells contained granular or diffuse bile pigment.

CASE 5.—R. E. J., a 22-year-old Negro was removed from the plating to the mechanic section after six months of employment because of the development of nasal ulcers. He had no subsequent symptoms. The results of the hepatic tests and analyses of the urine are recorded in tables 2 and 3. Liver biopsy was not done in this case.

COMMENT

Because of their protein-precipitating and oxidizing properties, chromium trioxide, chromates, and bichromates are potent protoplasmic poisons. Chromic oxide, trivalent chromium salts, and metallic chromium are much less toxic.[15] Chromium has been reported to be present in the teeth but has not been found in other tissues normally.[16] In intoxications, however, it has been found widely distributed in most organs and secretions, and its appearance in the urine has been reported in several patients.[17] Exposure to these substances is common, because of their widespread use in industries such as printing, tanning, photography, dyeing, and electroplating.[18] In the last process, the metal to be plated with chromium is immersed in a solution of chromic acid, through which is passed an electric current. Owing to the liberation of oxygen and hydrogen gases at the electrodes, a fine spray of chromic acid arises over the bath, carrying chromium trioxide into the air. The maximum safe concentration of chromium trioxide has been considered to be 0.1 mg. per cubic meter of inhaled air. Unless proper ventilation is present, the mist from chromic acid baths may lead to concentration of chromium trioxide of 2.5 to 6.5 mg. per cubic meter of air.[4c]

All our patients worked in a chromium plating plant for one to five years, four of the five patients constantly next to the chromic acid bath. Four had a history or findings, or both, of the commonest type of injury in the chromium plating industry, namely, nasal hyperemia or ulceration. All were eliminating significant amounts of chromium when examined. Two showed clinical evidence of hepatic involvement: A. J. (case 1) was jaundiced, and J. R. (case 2) had definite hepatic tenderness but was otherwise well. The other patients had no physical evidence of systemic disease.

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[15] (*a*) Heyroth, F. F.: The Metals (Except Lead), in Patty, F. A.: Industrial Hygiene and Toxicology, New York, Interscience Publishers, Inc., 1949, vol. 2, pp. 675-738. (*b*) Akatsuka, K., and Fairhall, L. T.: The Toxicology of Chromium, J. Indust. Hyg. **16**: 1-24 (Jan.) 1934. (*c*) Conn, L. W.; Webster, H. L., and Johnson, A. H.: Chromium Toxicology: Absorption of Chromium by the Rat When Milk Containing Chromium Was Fed, Am. J. Hyg. **15**: 760-765 (May) 1932.

[16] Heyroth.[15a] Lowater, F., and Murray, M. M.: Chemical Composition of Teeth: V. Spectrographic Analysis, Biochem. J. **31**: 837-841 (May) 1937.

[17] Letterer, Neidhardt, and Klett.[6a] Brieger.[6b] Goldman and Karotkin.[6d] Bianchi.[6g] Chromium Nephritis and Detection in Urine, Queries and Minor Notes, J. A. M. A. **110**: 1060 (March 26) 1938.

[18] Chromium Poisoning: Cause and Prevention, Industrial Health Series, No. 5, United States Department of Labor, 1943.

Each patient had abnormalities in the results of the hepatic tests. A. J., whose clinical course was one of hepatitis, had grossly abnormal results of hepatic tests initially, followed by gradual improvement. The liver biopsy showed centrolobular necrosis, marked Kupffer cell proliferation, enlarged portal fields with lymphocytic and histiocytic infiltration, and bile duct proliferation. J. R., having liver tenderness on physical examination, had diminished prothrombin activity, which did not respond to adequate parenteral administration of vitamin K; a borderline elevation of the icterus index; and abnormal retention of sulfobromophthalein. Here, the findings on biopsy were focal necroses, moderately increased cellularity of the portal spaces, moderate damage of the cells around the central veins, and bile pigment imbibition. A. L. J. (case 3) had an elevated icterus index, reduced cholesterol ester percentage, and abnormal retention of sulfobromophthalein. In this instance the only abnormal finding on microscopic examination of the liver was the finely granular bile pigment imbibition. C. R. (case 4) had an elevated icterus index on one occasion (normal on another), elevated serum globulins, and abnormal retention of sulfobromophthalein. The biopsy showed minimal central degenerative changes combined with bile pigment imbibition. R. E. J. (case 5), most removed from the chromic acid bath and found to be eliminating the least amount of chromium, had borderline retention of sulfobromophthalein as the only abnormal result of the hepatic tests. Biopsy was not done in this case.

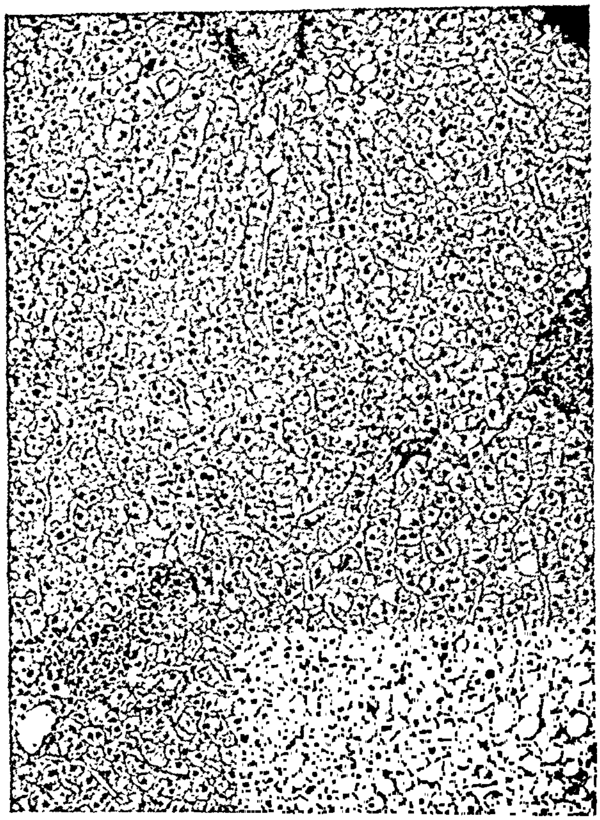

Fig. 3 (case 2).—The cells around the central vein show alterations in nuclear and cytoplasmic staining. Scattered fat and hydropic vacuoles are seen. The portal fields are enlarged and very cellular. (× 125)

Of the four biopsies, two showed pronounced and two slight abnormalities of hepatic structure. The histological changes found in the patient in whom jaundice developed were strikingly similar to those observed in experimental chromium poisoning in rabbits.[6g]

The indictment of chromium in these cases as the causative hepatotoxin seems justified in view of the history of exposure to a toxic form of chromium (chromium trioxide from chromic acid mist), the absence of exposure to other toxins (including alcohol), the finding of chromium in the urine in significant amounts, abnormalities in the results of hepatic tests, and the fact that the pathological findings in the liver did not resemble those seen in viral or other infective hepatitis, but rather those seen in the so-called toxic hepatitis.[19]

If it be true that industrial exposure to the toxic chromium compounds may cause systemic as well as surface injury in the human, the health hazard in plants using these compounds is not as negligible as suggested in the literature. Prophylactic measures, such as adequate ventilation and protective covering, should be taken in all factories to reduce exposure to a minimum. Skin and mucous membrane lesions should be treated promptly to prevent chronic systemic absorption of these compounds.[20] The effect of dimercaprol injection could not be evaluated in our patient because she was recovering at the time it was used.

The clinical well-being of most of our patients despite the presence of hepatic dysfunction as evidenced by hepatic tests and biopsy suggests that similar cases may have been overlooked because of the lack of more recent techniques for hepatic examination. The incidence of systemic poisoning in industries using chromium compounds, therefore, may require further investigation.

SUMMARY

Acute hepatitis with jaundice was observed in a person employed in the chromium electroplating industry. Chromium was identified in the urine on several examinations. Liver biopsy showed histological changes resembling those found in "toxic" hepatitis. In four other electroplating workers, found to be excreting significant amounts of chromium in the urine, hepatic tests and liver biopsy showed mild to moderate abnormalities. These workers had no symptoms related to the liver or other internal organs but did have the nasal lesions frequently attributed to chromium intoxication. The appearance of hepatitis in a chromium worker, as well as the existence of hepatic dysfunction in workers with no constitutional complaints, suggests that subtle systemic intoxication of employees of industries using chromium may be a definite health hazard.

629 S. Wood St.

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[19] Popper, H., and Franklin, M.: Viral Versus Toxic Hepatic Necrosis, Arch. Path. 46: 338-376 (Oct.) 1948.

[20] Zvaifler.[4a] Bloomfield and Blum.[4e] Blair.[4g] Footnote 18.

CLINICAL NOTES

FATAL VENTRICULAR FIBRILLATION FOLLOWING PROCAINE AMIDE HYDROCHLORIDE THERAPY

REPORT OF A CASE

John M. Read, M.D., Elko, Nev.

This is a report of death immediately following an intravenous injection of procaine amide ("pronestyl") hydrochloride. The case is reported because it is felt that instances of important untoward as well as the favorable effects of a drug should be generally known. Except for deaths in moribund patients and/or following exceedingly large, rapidly administered doses [1] no instances of death following the routine, cautious use of procaine amide have come to my attention. Aside from an increasingly cautious tone in the more recent articles [2] and an isolated report of agranulocytosis following oral administration,[3] most authors have mentioned the relative lack of serious toxic effects.[4]

REPORT OF A CASE

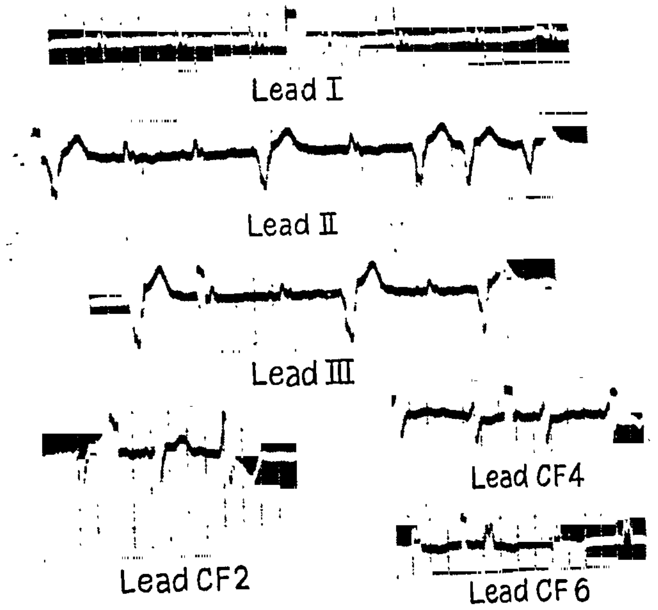
A 61-year-old widow was admitted to the Elko General Hospital in June, 1951, after a third attack of acute weakness and dyspnea in 48 hours. She had had rheumatic fever at 11 years. In 1935 she was admitted to the Elko General Hospital and a diagnosis of mitral stenosis and congestive failure was made. Since then she had been admitted to several hospitals in the Western states and had taken digitalis preparations irregularly. Prior to this recent admission she had taken no digitalis for one week. For the past several years she had noted an irregular "jumpy" heart from time to time but had noted no lasting spells.

On admission she appeared tired. The temperature and respirations were normal. A slight, but definite, malar flush was present. The ocular fundi were normal, and the neck veins not distended. The lungs showed only a few scattered rales at both bases posteriorly. The apex beat was palpable in the sixth intercostal space at the anterior axillary line, and the area of cardiac dulness corresponded to this. The second pulmonic sound was accentuated, and the loud apical first sound was preceded by a rumbing murmur. A harsh, grade 3 systolic murmur was heard over the precordium. The blood pressure was 120/80. An occasional premature beat was heard. A slightly tender, smooth liver edge descended 3 cm. below the right costal margin. There was no sacral edema. ankle edema present, graded as 1+. There was

The blood cell count, urine, sedimentation rate, and stool were normal. The result of the test with VDRL antigen was negative. The chest roentgenogram showed hazy, diffuse densities extending from both hili and a greatly enlarged heart with a typical "mitral" configuration. The electrocardiogram showed slurred ventricular complexes and diphasic T waves in the limb leads. The QRS time was 0.14 seconds. The R-T segment was slightly elevated in CF_1, CF_2, and CF_3 and slightly depressed with inverted or diphasic T waves in CF_4, CF_5, and CF_6. Successive, rapid premature beats, apparently arising in the right ventricle, were present (see the accompanying figure).

On the second day the patient complained of a smothering feeling, palpitation, and heaviness in the chest. That evening the nurse noted the pulse rate to be 130 and called the patient's physician. When seen at 11:30 p. m., she was apprehensive, cold, and orthopneic. She complained of palpitation, which she believed to have been present since 9 a. m. (14 hours). The neck veins were engorged. The apical and radial rates were equal and varied between 162 and 164. There was marked variation in the intensity of the first sound as heard over the apex and precordium, but the rhythm seemed regular. The blood pressure was 110/74. Carotid sinus pressure was not applied.

In the light of the bedside findings and the recent electrocardiographic observations, it was felt that ventricular tachycardia was present. Because of the patient's condition, prompt treatment seemed imperative. Accordingly 200 mg. (2 cc.) of procaine amide hydrochloride was given slowly into the left antecubital vein. The injection required three minutes, and a stethescope was held in place over the precordium during and after the injection. The rate and rhythm remained unchanged until just after the needle was withdrawn, when, for a few cycles, the rhythm became irregular; then the rate abruptly rose and no count could be made. At this time the patient uttered a faint cry and ceased breathing for a moment. The heart continued to beat wildly and very rapidly for 15 to 20 seconds; then the intensity of the heart sounds diminished and became inaudible. Slow, irregular respirations continued for two and a half minutes after the cessation of heart sounds.



Electrocardiogram taken 36 hours before death.

COMMENT

The close temporal relationship between the administration of the drug and sudden death was so striking as to exclude the possibility of coincidental death from an inevitable ventricular fibrillation. Though electrocardiographic and blood pressure controls were lacking, there seems little doubt that death was due to ventricular fibrillation. The remainder of the bottle was sent to the manufacturer's laboratory; the contents were reported as procaine amide hydrochloride without impurities.

From the Elko Clinic.

1. (*a*) Kayden, H. J.; Steele, J. M.; Mark, L. C., and Brodie, B. B.:
Use of Procaine Amide in Cardiac Arrhythmias, Circulation **4**: 13 (July)
1951. (*b*) Berry, K.; Garlett, E. L.; Bellet, S., and Gefter, W. I.: Use of
Pronestyl in the Treatment of Ectopic Rhythms, Am. J. Med. **11**: 431
(Oct.) 1951.

2. (*a*) Berry, Garlett, Bellet, and Gefter.[1b] (*b*) McCord, M. C., and
Taguchi, J. T.: A Study of the Effect of Procaine Amide Hydrochloride in
Supraventricular Arrhythmias, Circulation **4**: 387 (Sept.) 1951.

3. Miyoko, I.; Millar, J., and Townsend, J. H.: Agranulocytosis Fol-
lowing Maintenance Dosage of Pronestyl: Report of Severe Case with
Recovery, J. A. M. A. **147**: 652 (Oct. 13) 1951.

4. Mark, L. C., and others: The Action of Procaine Amide (N^1-[2-Di-
ethylaminoethyl] p-Aminobenzamide) on Ventricular Arrythmias, abstract,
J. Pharmacol. & Exper. Therap. **98**: 21 (Jan.) 1950. Kinsman, J. M.;
Clay, H. L.; Coe, W. S., and Best, M. M.: Procaine Amide (Pronestyl)
in the Treatment of Disorders of Cardiac Rhythm, Kentucky M. J.
48: 509 (Nov.) 1950. Joseph, S. I., and Rovenstine, E. A.: Cardiac
Arrhythmias: Prophylaxis and Therapy with Procaine Amide During
Cyclopropane Anesthesia for Thoracic Surgery, abstract, J. Pharmacol.
& Exper. Therap. **101**: 19 (Jan.) 1951. Joseph, S. I., and others: Procaine
Amide for Prophylaxis and Therapy of Cardiac Arrhythmias Occurring
During Thoracic Surgery, Surg., Gynec. & Obst. **93**: 75 (July) 1951.

Knowledge of the pharmacology of procaine amide hydrochloride is far from complete, but one may theorize on possible causes for this fatal outcome. The hypotensive effect of intravenous procaine amide has been well described.[5] Death in this instance from such a cause seems highly improbable.

Another known effect is the widening of the QRS complex, and, in some instances, bundle branch block has developed. Recently McCord and Taguchi [2b] noted an increase in ventricular rate when procaine amide hydrochloride was administered to patients with auricular fibrillation. Such quinidine-like effects [6] might more easily cause ventricular fibrillation in a heart in which bundle branch block was already established. Caution in the use of quinidine in the presence of bundle branch block has been advised for years, but in practice this is honored more in the breach than the observance. It may be that such will be the case with procaine amide hydrochloride.

SUMMARY

A case of ventricular fibrillation with death immediately following an intravenous injection of procaine amide is reported. It is emphasized that caution is necessary in the use of this drug; increased caution is suggested in cases of bundle branch block.

P. O. Box 269.

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5. *Miyoko, Millar, and Townsend.*[3] Procaine Amide Hydrochloride, Council on Pharmacy and Chemistry: New and Nonofficial Remedies, J. A. M. A. **144**: 1465 (Dec. 23) 1950. Stearns, N. S., and Callahan, E. J.: A Clinical Study of Intravenous Procaine Amide (Pronestyl): Its Value and Hazards, abstract, J. Clin. Investigation **30**: 676 (June) 1951. Edmonds, B. W.; *Comer, W. H.;* Kennedy, J. D., and Taylor, I. B.: Intravenous Use of Procaine in General Anesthesia, J. A. M. A. **141**: 761 *(Nov. 12) 1949.*

6. Gold, H.: Quinidine in Disorders of the Heart, New York, Paul B. Hoeber, Inc., 1950.

VENTRICULAR ACCELERATION FOLLOWING PROCAINE AMIDE HYDROCHLORIDE THERAPY

John L. Denney, M.D.
Harold Miller, M.D.
George C. Griffith, M.D.
and
Morris H. Nathanson†, M.D., Los Angeles

Procaine amide ("pronestyl") hydrochloride has been recently introduced for the treatment of cardiac arrhythmias; its use has been found effective especially in rhythms of ventricular origin.[1] The drug appears to be relatively innocuous, and, aside from occasional mild to moderate hypotensive reactions, no serious untoward effects have been observed. It is known that cardiac depressant and antifibrillatory drugs, although effective in the elimination of ectopic rhythms, may at times induce ectopic ventricular rhythms, including ventricular fibrillation. Such cardiac effects have been observed during the course of quinidine sulfate therapy.[2] They have been observed with greater frequency after the administration of the potent antifibrillatory drug alpha-fagarine and related compounds.[3] In 55 patients with various auricular and ventricular arrhythmias, continuous electrocardiograms were made during and after the intravenous administration of procaine amide hydrochloride.[1d] We found no indication of drug-induced ectopic ventricular activity in this group. However, in three patients with complete heart block the intravenous administration of relatively small doses of procaine amide hydrochloride was followed by the appearance of frequent multifocal ectopic ventricular beats in two cases and a transient ventricular fibrillation in one.[4] It has been suggested that ectopic ventricular activity and ventricular fibrillation induced by quinidine sulfate therapy depends on an impaired function of the conducting system. The following report concerns a patient in whom pronounced widening of the QRS wave and a paroxysm of ventricular acceleration developed during and after the intravenous administration of procaine amide hydrochloride.

REPORT OF A CASE

The patient was a 69-year-old white woman, admitted to the hospital on Nov. 6, 1951. Her complaints were weakness, shortness of breath, and palpitation. She had had a myocardial infarction two years previously and had made an uneventful recovery. Since that time she had had dyspnea on mild effort. On the evening of admission, while walking to the grocery store, she experienced a sudden onset of palpitation, associated with weakness and shortness of breath, and was taken to the hospital.

On admission, she showed the following significant physical changes. She was moderately dyspneic, and there was a slight degree of cyanosis of the lips and finger tips. There was a pronounced distention of both jugular veins. Moist rales were present at both lung bases posteriorly. On auscultation, the heart rate was very rapid but appeared to be regular. The sounds were distant, and there were no murmurs. The blood pressure was 90 systolic and 70 diastolic. The abdomen was obese, but there were no palpable organs or masses. There was no edema of the extremities. The diagnosis was ectopic tachycardia and acute coronary occlusion as a possible basis for the arrhythmia.

An electrocardiogram was made shortly after admission, which showed a ventricular tachycardia, rate 210 per minute (*A* in the accompanying figure). The QRS wave was 0.1 second in duration, and there was a slurring of the R wave on the descending limb. Procaine amide hydrochloride, 500 mg., was injected by vein slowly at the approximate rate of 100 mg. per minute. This was followed by a gradual widening of the QRS; at one minute after the onset of injection, the QRS was 0.14 second and a notching appeared on the descending limb of the R wave (*D* in the accompanying figure). As the injection of the drug was continued, there was a gradual reduction in heart rate and a widening and increased notching of the QRS (*E* through *K* in the figure. Strip *K* taken four and a half minutes after the start of the injection, shows the rate reduced from 210 to 160 per minute and the QRS wave widened to 0.2 second with pronounced notching on the descending limb. Five minutes after the start of the injection of the drug, a series of rapid regular ventricular oscillations appeared (*L* in the figure). There was a gradual reduction in the amplitude of these oscillations. This type of complex may be considered to represent a ventricular tachycardia from a new focus or a ventricular flutter. Such oscillations have been observed preceding the irregular oscillations of smaller amplitude, which are characteristic of ventricular fibrillation.[5] This was followed by ventricular complexes of approximately normal configuration, rate 140, a nodal rhythm (*M* in the figure). Auricular waves preceding the QRS gradually appeared, and restoration of sinus rhythm occurred eight minutes after the start of the injection (*P* in the figure). The patient was given 500 mg. of procaine amide hydrochloride every four hours. She had no further attacks, and subsequent observation showed no evidence of myocardial infarction.

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† Dr. Nathanson died on April 24, 1952.

From the University of Southern California School of Medicine and Medical Service, Los Angeles General Hospital. Resident in Medicine (Dr. Denney); Research Associate, Department of Medicine, Cardiology, (Dr. Miller).

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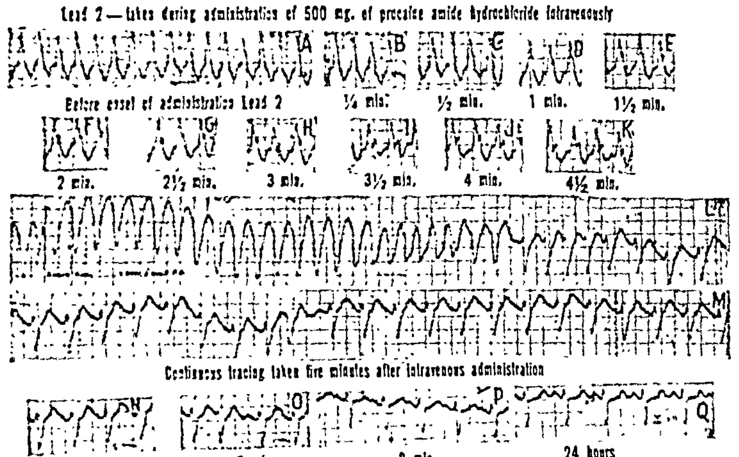
COMMENT

Widening of the QRS wave has been observed during quinidine sulfate therapy and is considered as an indication for discontinuance of the use of the drug. Gold [2b] described a case of fatal syncope during the administration of quinidine sulfate, presumably due to cardiac standstill or ventricular fibrillation. An electrocardiogram taken after 5 gm. of the drug had been administered showed that the QRS, which had been normal before treatment, was prolonged to 0.16 second. In discussing the treatment of ventricular tachycardia, Gold stated that it is not wise to continue quinidine sulfate therapy if the QRS has been prolonged to 50% above the control. He expressed the belief that a widening of the QRS may represent a precursor to ventricular fibrillation. Sokolow,[2d] in a recent review, stated that he did not continue quinidine sulfate therapy after the increase in QRS time exceeded 50% of the control value and he used particular caution when the increase exceeded 25%. Kayden and co-workers [1b] noted that continuous intravenous infusion of procaine amide hydrochloride until death in the experimental animal produced the following events: prolongation of the P-R interval, widening of QRS complexes, wandering pacemaker, nodal and ventricular premature contractions, and ventricular tachycardia and fibrillation.


Lead 2—taken during administration of 500 mg. of procaine amide hydrochloride intravenously
A
B
C
D
E
Before onset of administration Lead 2
¼ min.
½ min.
1 min.
1½ min.
F
G
H
I
J
K
2 min.
2½ min.
3 min.
3½ min.
4 min.
4½ min.
L
M
Continuous tracing taken five minutes after intravenous administration
N
O
P
Q
5½ min.
6 min.
8 min.
24 hours

Electrocardiographic changes during the intravenous administration of 500 mg. of procaine amide hydrochloride to a patient with ventricular tachycardia.

Davis and Sprague [6] and Davis [7] have emphasized the significance of the integrity of the conducting tissue in the prevention of ectopic ventricular rhythms. An intact conducting system distributes impulses quickly and uniformly to all regions of the ventricular myocardium. This not only insures harmony of contraction but further produces a uniform state of refractoriness. Such a generalized state of refractoriness protects against reentrant excitation. When there is impairment of ventricular conduction, the stimulus is delayed to portions of the heart and subsequently there is a variable state of refractoriness in the ventricular myocardium. The stimulus may then be blocked at hypoexcitable areas, continue along responsive paths, and reenter the blocked portion when it becomes responsive. This re-entry mechanism is considered to be the basis for ventricular rhythms, including ventricular fibrillation. For this reason it has been emphasized that, during the administration of such drugs as *quinidine sulfate and digitalis, conduction defects* are a warning sign of the onset of serious ventricular rhythms. In the present instance, the ventricular arrhythmia that occurred during the treatment was of short duration and had no untoward effects. However, it represents a prefibrillation state such as is frequently seen before the onset of fatal ventricular fibrillation. It would appear that, as with quinidine sulfate, widening of the QRS during procaine amide hydrochloride therapy is an indication for discontinuance of the use of the drug.

CONCLUSION

A case of ventricular tachycardia treated with procaine amide ("pronestyl") hydrochloride is reported in which the following events occurred: (*a*) progressive and pronounced widening and notching of the QRS wave of the electrocardiogram, (*b*) a short paroxysm of ventricular acceleration of a prefibrillation type, and (*c*) restoration of sinus rhythm. It is suggested that, as with quinidine *sulfate therapy*, a widening of the QRS wave during the administration of procaine amide hydrochloride may represent a precursor to ventricular fibrillation and is an indication for discontinuance of the use of the drug.

1200 N. State St. (Dr. Miller).

Basal Metabolic Rate.—Either hypothyroidism or hyperthyroidism may be present in the face of a basal metabolic rate that falls within the normal range. Moreover, a low basal metabolic rate does not always indicate hypothyroidism, but may represent hypometabolism without hypothyroidism. Contrariwise, an elevated basal metabolic rate does not invariably mean hyperthyroidism. In the absence of thyrotoxicosis high values may be encountered in such diverse states as Paget's disease, polycythemia, anemia, leukemia, lymphosarcoma, Hodgkin's disease, essential hypertension, aortic stenosis, coarctation of the aorta, and dyspnea due to cardiac or pulmonary disease. Elevations of the basal metabolic rate may also result when the patient is inadequately rested or fasted, overexcited, or has a tremor or a punctured tympanic membrane.—J. L. Gabrilove, M.D., and W. R. Dorrance, M.D., Tests of Endocrine Function. *The Medical Clinics of North America*, May, 1952.

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FEVER DUE TO PROCAINE AMIDE HYDROCHLORIDE THERAPY

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and
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Procaine amide ("pronestyl") hydrochloride is a drug widely used for the elimination and prevention of ectopic ventricular rhythms. Its oral use at times is maintained for long periods to prevent recurrent premature beats or paroxysmal ventricular tachycardia. Toxic side-effects have been infrequent and usually not serious. Minor toxic affects have been gastrointestinal irritation with anorexia, nausea, and vomiting, usually occurring with a high dosage (6.0 to 8.0 gm. a day).[1]

A major toxic effect has been the production of granulocytopenia following bone marrow depression with death.[2] We recently observed a patient in whom chills, fever, generalized muscle pains, malaise, dermatitis, and mental depression developed during the administration of the drug. The fever disappeared abruptly when use of the drug was discontinued and recurred on two subsequent occasions after readministration of the drug. The causative relation of the drug to the fever seemed definite. We had not previously encountered a similar instance, but, during the preparation of this report, we found an article by Leibowitz [3] describing a patient in whom chills and fever developed after oral ingestion of procaine amide hydrochloride. He predicted, "As experience with procaine amide increases it is likely that similar reactions will be encountered." In our patient, much laboratory work was done in an attempt to explain the cryptogenic fever before the drug was suspected as causative. It is probable that other instances are occurring and are unrecognized, especially if the patient is ambulatory and not under close observation. Chills, fever, muscle aches, and malaise could easily be ascribed to current viral infections or other plausible causes. Procaine amide hydrochloride must be suspected as a possible cause of any febrile reaction occurring during its administration.

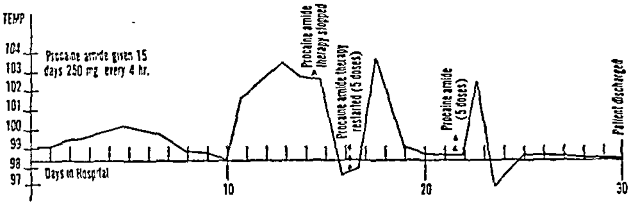
REPORT OF A CASE

A 58-year-old unmarried woman was first seen in the office Jan. 22, 1952, complaining of bilateral anterior chest and shoulder pain, with extension to both arms and to the interscapular region, of six days' duration. An electrocardiogram demonstrated unequivocal evidence of acute posterior myocardial infarction. The patient was immediately hospitalized. Anticoagulant treatment was started, with intravenous administration of heparin and oral administration of bishydroxycoumarin (dicumarol®). Procaine amide hydrochloride, 250 mg. every four hours day and night, was given on admission because of the presence of frequent premature ventricular beats. The patient's temperature on admission was 98.2 F.

During the following 10 days the oral temperature fluctuated diurnally, reaching a high of 100.4 F (see the accompanying figure). This low grade febrile response was accepted as commensurate with cardiac infarction.

On the 11th hospital day, the oral temperature rose to 101.8 F. The patient became nauseated and vomited. She was irritable and apprehensive. During the succeeding four days the temperature remained elevated, reaching a maximum of 104 F. The white blood cell count was 5,000. The cause of the sudden rise in temperature and the clinical picture of malaise, nausea, and vomiting was not clear. Septicemia, genitourinary infection, pulmonary infarction, brucellosis, typhoid, and peripheral thrombophlebitis were considered and investigated with appropriate laboratory studies. After five days of continuous fever, although no one was familiar then with reports of fever due to the use of bishydroxycoumarin or procaine amide hydrochloride, it was thought that the reaction might be caused by one of these.

On the 15th day at 6 a. m., the administration of both bishydroxycoumarin and procaine amide was discontinued. The temperature began to drop and 24 hours later was normal; it remained so during the following 36 hours. Improvement in the patient was immediate and dramatic. The aching and malaise disappeared; the mental irritability and apprehension was succeeded by cheerfulness. The nurses' record said, "She has no complaints." Two days later the procaine amide therapy was started again, 250 mg. every four hours for five doses. The first dose was given at 10 a. m. By 4 o'clock in the afternoon the patient felt distinctly uncomfortable and mentally irritable and by 11 o'clock the temperature was 104 F. The next morning she was nauseated, mentally irritable, and depressed, talking incessantly and wanting to go home. The following day the temperature was normal; she felt much better, appeared normal, and listened with interest to the radio. For five days she felt well and the temperature was normal.



Fever following oral administration of procaine amide hydrochloride. The fever subsided with discontinuance of the use of the drug and recurred when it was readministered on two occasions.

After five days procaine amide therapy was started again. After the third dose the patient recognized the effect of the drug and said, "It is those little yellow pills that are upsetting me." The temperature again rose to 104 F; the next day a maculopapular, itching rash appeared on the extensor surfaces of the arms, feet, thighs, and dorsa of both feet. Again, after discontinuance of the use of the drug, the temperature became normal the next day, and the rash faded. During the remainder of her hospital stay she had no further trouble.

SUMMARY

An instance of fever, chills, rash, and malaise following oral administration of procaine amide ("pronestyl") hydrochloride is reported.

1930 Wilshire Blvd. (5) (Dr. Askey).

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ALLERGY TO PROCAINE AMIDE

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The therapeutic similarity between procaine amide and quinidine is recognized; however, whereas the toxic effects of quinidine are well known, procaine amide ("pronestyl") has not yet been used widely enough to permit evaluation of its ability to cause toxic reactions. The following case of a true allergic reaction to procaine amide is, therefore, discussed.

REPORT OF A CASE

A 53-year-old man with known hypertension for the past 20 years was first seen on Nov. 11, 1951, with sudden onset of severe substernal oppression. An electrocardiogram revealed acute anteroseptal myocardial infarction. On Dec. 11, 1951, the heart rate, which had been 85 per minute, increased to 115, and numerous extrasystoles, subsequently shown by electrocardiograph to be auricular premature contractions, were noted. The patient was given 1.5 gm. of procaine amide orally; this was followed by 250 mg. every four hours. After the third dose, when a total of 1 gm. had been taken, a generalized maculopapular erythematous rash developed, accompanied by severe itching, congestion of the nose, slight generalized adenopathy, and a fever of 102 F. Cessation of procaine amide therapy and institution of diphenhydramine (benedryl®) hydrochloride, 50 mg. every 4 hours, was followed by complete subsidence of all symptoms within 24 hours.

Inasmuch as the patient was receiving other medication at the time of this reaction, it was decided to test him with procaine amide alone. Consequently, on Feb. 10, 1952, he was placed on 250 mg. of procaine amide every four hours. Following the fourth dose, the identical clinical picture noted above developed; this condition again promptly responded to diphenhydramine hydrochloride. A stained blood smear at the height of the reaction showed 7% eosinophils as compared with ½ to 1% in the control smear before beginning the procaine amide test. A subsequent patch test with the drug was negative.

COMMENT

Hitherto, reported toxic effects of procaine amide had been limited to the circulatory and gastrointestinal systems.[1] Recently, reference has been made to three cases of agranulocytosis [2] and two cases of urticaria [3] following its use. Although it is not unusual to find a patient who is sensitive to quinidine and who has fever and a skin eruption following even small doses, such a reaction to procaine amide has not been previously reported.

923 Fifth Ave.

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HUMAN POISONING BY CHLORDANE

REPORT OF A CASE

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and
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The newer insecticides have proved their value in control of insects.[1] Since the products have been made available to the public, however, accidental poisonings have occurred. We are reporting our experience in such a circumstance.

Chlordane, a chlorinated hydrocarbon having the empirical formula $C_{10} H_6 Cl_8$, is in the pure state a viscous, amber-colored, odorless liquid. It can be formulated for use in varying strengths as an oil solution, an emulsion concentrate, a dust, or a wettable powder.[2] Human toxicity of these preparations depends on the type of vehicle as well as the concentration of chlordane. Some of the toxic manifestations ascribed to chlordane preparations may be due to the vehicle alone.[3] Contamination of the skin with certain petroleum distillates has produced anesthesias and paresthesias that may be due to the fat solvent properties of the distillates.[4]

The insecticide enters the body by absorption through the intact skin, by inhalation of dusts or sprays, and by ingestion. Skin and intestinal absorption is enhanced by the presence of fats, oils, or organic solvents.[5] Chlordane is moderately irritating to the skin in concentrated form, but in the diluted solutions commonly used this skin warning fails to appear. As little as 2.4 gm. in solution contacting the skin daily may be dangerous to man.[6] Acute fatal poisoning may result from a dose of 5 to 60 gm.[7] The onset of symptoms may occur as early as 45 minutes following ingestion of chlordane. Death may result within 24 hours, but most commonly not until the third or fourth day. If survival continues to the sixth day, recovery usually follows. Irritability of the central nervous system is the earliest sign and symptom. This irritability progresses into convulsions alternating with periods of depression.[3] Death occurs following deep central nervous system depression with or without a terminal seizure.[7] Chronic poisoning produces predominately inanition, probably due to the severe degenerative changes in the liver, the most severely damaged of the vital organs. These changes are similar to those produced by other chlorinated hydrocarbons such as chloroform.[8] Degenerative changes in the proximal convoluted tubules of the kidney [9] and subserosal hemorrhages in the intestines and heart have been demonstrated in animals.[10]

Prophylaxis should be directed toward preventing acute and chronic exposures to the poison through the skin and respiratory tract in the manufacture, in formulation of commercial compounds, and in the ultimate use by the public. This and a decrease in the accidental ingestion may be brought about by stressing the human toxicity of this substance. Actual treatment involves removal of the poison from the gastroenteric tract and skin as rapidly as possible by any means available. Convulsions should be controlled by sedatives or general anesthesia. Stimulants, and other supportive measures such as oxygen, should be used in the phases of central nervous system depression.

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REPORT OF A CASE

C. J. E., 15-month-old daughter of one of the authors, accidentally drank a small amount, probably not more than a mouthful, of a chlordane suspension. This occurred at about 3 p. m. Although the child spit out some of the suspension, the mother promptly induced vomiting by administering sodium bicarbonate solution and gagging the child. This was accomplished within 2 to 3 minutes following ingestion. The chlordane suspension was made using 1 tablespoon of a wettable powder (50% chlordane) suspended in 1 qt. (1 liter) of water. The distributor of the commercial preparation, the county agricultural agent, and a biochemist were consulted. All were of the opinion that the amount taken was probably not toxic, although all admitted there was not sufficient information available to them concerning chlordane's toxicity.

About three hours after ingestion (6 p. m.) a generalized tremor developed, and the child became incoordinate and ataxic, falling several times. Convulsions soon ensued, tonic phases alternating with short clonic phases, until they were controlled by ethyl chloride inhalations about 40 minutes later. On examination when the convulsions began her temperature was 99.6 F rectally, the pupils were dilated but reacted to light, her face was flushed; the respirations were irregular, and the pulse rate was 140 per minute. Risus sardonicus, opisthotonos, bilateral ankle clonus, and generalized hyperactive reflexes were present. Cyanosis was noted only during administration of ethyl chloride. After the convulsions were controlled by ethyl chloride inhalations, amobarbital (amytal®) sodium was given (0.06 gm. intravenously and 0.06 gm. subcutaneously) to facilitate gastric lavage with magnesium sulfate saturated solution. The strong odor of chlordane suspension was apparent in all washings. Some of the magnesium sulfate was left in the stomach. At 7:30 p. m. (4½ hours after ingestion of chlordane) the convulsions seemed controlled, and the patient was admitted to a hospital. Thirty minutes later generalized convulsions recurred and were not controlled until 0.345 gm. of amobarbital sodium was administered intravenously, and 0.06 gm. subcutaneously. Oxygen and fluids intraveneously were given continuously. For the next eight hours respirations were irregular in rate and shallow in depth except for an occasional deep sigh. We were unable to determine whether the respiratory depression was due to the chlordane ingested or to the large amount of amobarbital sodium given. Thirteen hours after chlordane ingestion respirations resumed normal character, and 16 hours after chlordane ingestion the child roused. Examination at this time revealed irritability, dilated pupils, and some ataxia. For the next few hours the child was constantly moving, unaware of her surroundings, and irritable when disturbed. The pupils resumed normal size, and respirations continued to be regular.

During the second day the consulting neurologist found only moderate ataxia and irritability. This irritability prevented the obtaining of an electroencephalogram; however, an electrocardiogram and chest roentgenogram were made, and the patient was discharged from the hospital. The ataxia and irritability progressively diminished and finally disappeared after two to three weeks.

Past History.—The child had had no convulsions previously. She had always been in good health, and her development had been normal.

Laboratory Data.—On the day following admission a blood count revealed only mild hypochromic anemia. On this day and the following day her urine contained an occasional white blood cell and red blood cell in a centrifuged specimen. The electrocardiogram was within normal limits, and chest roentgenogram revealed no abnormality of the heart or lungs. An attempt was made to obtain an electroencephalogram three weeks later but was unsuccessful, owing to lack of cooperation by the patient.

COMMENT

Individual susceptibility to chlordane varies within wide limits, probably even more than is at present determined. In our case the quantity ingested unfortunately could not be determined exactly, but it is our impression that the amount of chlordane ingested by this child weighing 9 kg. did not exceed 100 mg. Because of the individual susceptibility, treatment should be immediate and directed toward the prompt removal of chlordane from the body, no matter how small the amount. Careful gastric lavage in our case was not done immediately because the information we obtained indicated that the amount ingested was too small to be of concern. As Lehman has well stated, "Absolute toxicity of any given insecticide is of relatively minor interest, but the real criterion of utility must ultimately be based on the hazards to man. . . . Little effort is directed towards the study of long-range effects on plant and animal life before the insecticide is introduced into commerce."[11]

SUMMARY

A case of poisoning due to a new popular commercial insecticide, chlordane, demonstrates the signs and symptoms of toxicity. The need for adequate investigation and dissemination of information concerning the dangers of insecticides to man is stressed.

2300 Caroline St. (Dr. Lensky).

[11] Lehman, A. J.: The Toxicology of the Newer Insecticides, Proceedings International Congress Tropical Medicine and Malaria 2: 1713, 1948.

MENINGITIS DUE TO PSEUDOMONAS AERUGINOSA

INTRATHECAL TREATMENT WITH STREPTOKINASE AND STREPTODORNASE AND INTRAMUSCULAR AND INTRATHECAL TREATMENT WITH NEOMYCIN

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Robert C. Hardy, M.D.

and

Juan Negrin Jr., M.D., New York

The over-all mortality from meningeal infection with Pseudomonas aeruginosa was found by Stanley to be 68% in a review of 90 cases reported to 1947.[1] The same author observed that, when the cases were divided into groups depending on whether the infection was primary in the meninges or whether it had developed secondary to foci elsewhere in the body, there was a pronounced difference in the case fatality rate. Patients in whom meningitis was the primary disease, comprised mostly of persons who were infected by lumbar punctures or after neurosurgical procedures, showed a mortality rate of 55%. Patients in whom the meningitis occurred as a complication of infection originating elsewhere in the body showed a mortality rate of 86%. From these data it is apparent that meningitis due to Ps. aeruginosa is a serious disease. Moreover, it is our opinion that published

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This study was supported in part by a grant from Charles Pfizer & Company, Brooklyn.

From the Department of Medicine and Neurosurgery, Cornell University Medical College and the Cornell Medical and Surgical Divisions, Bellevue Hospital.

[1] Stanley, M. M.: Bacillus Pyocyaneus Infections: A Review, Report of Cases, and Discussion of Newer Therapy Including Streptomycin, Am. J. Med. 2: 253, 1947.

reports tend to minimize the mortality figures, since it seems certain that more recoveries than fatalities are deemed worthy of reporting.

Antimicrobial treatment of Ps. aeruginosa meningitis has only occasionally been successful, with the most favorable results obtained by the use of polymyxin B. Tomlin,[2] Hayes,[3] and Jawetz,[4] and their respective associates, have reported the successful treatment of one patient each with this agent. Two of the patients experienced severe local pain at the time the intrathecal injections were administered, and the two who received the material intramuscularly exhibited transient signs of nephrotoxicity, consisting of casts in the urine and albuminuria. Two patients relapsed after initial control of the disease but were later successfully re-treated.

Although this appears to be the most effective antimicrobial agent available for treatment of meningitis due to Ps. aeruginosa, one must maintain some reservations concerning the use of an agent in which the toxic concentration is so close to the therapeutic concentration.

Another approach to the treatment of meningitis that has recently received some clinical appraisal is the intrathecal administration of streptococcic concentrates containing streptokinase and streptodornase. This treatment has proved highly beneficial in cases of empyema and infected hemothorax, and on occasion sterilization of chest cavities has resulted without concomitant antimicrobial therapy.[5] It has been demonstrated that the streptococcic concentrate may cause lysis of purulent and sanguineous exudate,[6] with the result that infecting bacteria may be more directly exposed to host defenses or antimicrobial therapy. Presumably this is the basis for the activity of these substances in meningitis.

Recently we had an opportunity to treat a patient with meningitis due to Ps. aeruginosa that had developed after an elective laminectomy. The treatment employed consisted of intrathecal injections of a solution of streptokinase and streptodornase and intrathecal and intramuscular injection of neomycin. The results were highly favorable, and details of the patient's course constitute the present report.

REPORT OF A CASE

The patient, a 26-year-old white housewife, was first admitted to Bellevue Hospital on Jan. 29, 1951, with a history and physical findings consistent with the presence of a herniated intervertebral lumbar disk. The defect could not be demonstrated by myelogram. On March 14 a right hemilaminectomy of the fifth lumbar vertebra was performed, and a small disk impinging on the nerve root was found. The protruded disk was excised, leaving the dura intact. Twenty-four hours after operation, the patient's temperature rose to 105 F and slight nuchal rigidity was noted. Examination of the wound revealed evidence of infection at its upper end. Separation of the skin margins permitted drainage of approximately 12 cc. of seropurulent material. The infection appeared to arise from the depths of the wound, which was explored down to the dura. No foreign body was found. Cultures of the wound revealed a diphtheroid bacillus, and for this reason penicillin was given intramuscularly in large doses. After five days the patient's temperature returned to normal. On the seventh postoperative day nuchal pain and rigidity were again noticed, and wound cultures at this time revealed Ps. aeruginosa. Despite the presence of this organism, healthy granulations gradually filled the wound, signs and symptoms subsided, and the patient was discharged with the wound virtually healed on May 3. Lumbar puncture was not done postoperatively.

Five days later, on May 8, the patient was readmitted to the hospital, complaining of headache, stiff neck, vomiting, chills, and fever. The operative wound appeared well healed, and there was no local tenderness. Lumbar puncture revealed cloudy xanthochromic fluid, which contained 1,420 white blood cells per cubic millimeter; cultures revealed Ps. aeruginosa. During the next six weeks, the patient received continuous antibiotic therapy in high dosage; penicillin, chloramphenicol, terramycin, and streptomycin were given in various combinations. In the course of the treatment she received three intrathecal injections of streptomycin, 0.05 gm., at two-day intervals. Despite this treatment, she continued to have daily temperature elevations to 101 or 102 F. Cerebrospinal fluid sugar concentrations were low, ranging from 15 to 34 mg. per 100 cc. The chloride level ranged from 80 to 141 mg. per 100 cc., and several protein determinations were approximately 75 mg. per 100 cc. About 1,500 white blood cells per cubic millimeter were consistently observed in the cerebrospinal fluid, and approximately 2,500 red blood cells were noted on several occasions. Several cultures of the cerebrospinal fluid revealed Ps. aeruginosa, and on one occasion this organism appeared in a culture of the blood.

In view of the unremitting infection we decided to explore the wound again. Accordingly, on June 3 a total laminectomy of the fifth lumbar vertebra was done, with exploration of the epidural space. Abscess, foreign body, or other abnormality was not found.

Drug susceptibility tests revealed the organism to be resistant to 25 µg per cubic centimeter of chloramphenicol, aureomycin, and streptomycin. It was found to be sensitive to neomycin (4 units per milliliter in 24 hours). As the patient's condition continued to deteriorate despite three months of intensive antimicrobial therapy, we decided to institute intrathecal treatment with streptococcic concentrate (streptokinase and streptodornase) and neomycin.[7] The patient was given 15,000 units of streptokinase and 3,750 units of streptodornase intrathecally at two-day intervals, with daily intrathecal administration of 5,000 units of neomycin. Neomycin was also given intramuscularly in a daily dose of 120,000 units. The patient continued to receive penicillin. Details of this portion of her course may be seen in the accompanying chart. Her temperature rose to 105.8 F 12 hours after the first intrathecal administration of streptokinase and streptodornase, despite the fact that treatment with corticotropin (ACTH), 75 mg. daily, had been started 24 hours previously. During intrathecal administration of the enzyme solution, and for about one hour thereafter, the patient complained of discomfort of the lower part of the back and saddle area and experienced a dull headache of moderate severity. Lumbar puncture six hours later, however, failed to reveal any significant changes over pretreatment findings. She felt nauseated for several hours. Twenty-four hours after the initial intrathecal medication the patient became semistuporous and disoriented, and there was pronounced increase in nuchal rigidity. The cerebrospinal fluid pressure at this time was 330 mm. of water, and the fluid appeared distinctly more turbid. Great relief followed the slow withdrawal of 25 cc. of cerebrospinal fluid. On the alternate days, in which neomycin was administered intrathecally without enzymes, no subjective discomfort was noted, and there was no temperature elevation or other objective evidence of untoward effect. Cerebrospinal fluid cultures were continuously sterile after the second day of treatment. Chills and temperature elevations to 103 to 105 F occurred 8 to 12 hours after the patient received the remaining two intrathecal injections of streptokinase and streptodornase, and cerebrospinal fluid pressure following the third administration was 520 mm. of water. Sufficient amounts of cerebrospinal fluid were withdrawn to lower the pressure either to normal or to half the initial pressure. Papilledema did not develop. Blood pressure remained fairly constant, while the pulse rate varied proportionately with temperature fluctuations. Urine output was good and remained free of albumin, casts, and blood cells. The plasma nonprotein nitrogen concentration was determined before, during, and after treatment and did not exceed normal limits. Cerebrospinal fluid turbidity rapidly diminished after the completion of treatment, and by the ninth day it was crystal clear. The patient gradually improved; by the time of her discharge, one month after the cessation of treatment, she was without significant abnormal physical signs and had no symptoms. At the time of writing, she had been followed for a period of nine months and had remained well.

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[2] Tomlin, C. E.: Pseudomonas Meningitis: Report of a Case with Recovery After Polymyxin B Therapy, A. M. A. Arch. Int. Med. 87: 863 (June) 1951.

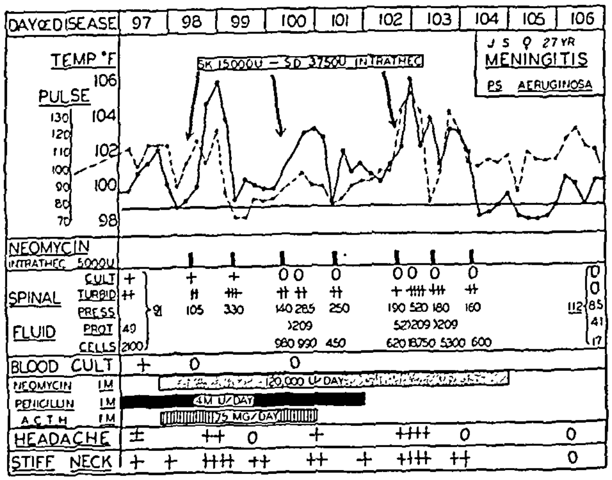
[3] Hayes, E. R., and Yow, E.: Meningitis Due to Pseudomonas Aeruginosa Treated with Polymyxin B, Am. J. M. Sc. 220: 633, 1950.

[4] Jawetz, E.: Infections with Pseudomonas Aeruginosa Treated with Polymyxin B, A. M. A. Arch. Int. Med. 89: 90 (Jan.) 1952.

[5] Tillett, W. S., and others: Streptococcal Enzymatic Debridement, Ann. Surg. 131: 12, 1950.

[6] Tillett, W. S., and Sherry, S.: The Effect in Patients of Streptococcal Fibrinolysin (Streptokinase) and Streptococcal Desoxyribonuclease on Fibrinous, Purulent, and Sanguineous Pleural Exudations, J. Clin. Investigation 28: 173, 1949.

[7] The neomycin used in this study, lot no. 3500424, was supplied by Dr. L. W. Smith, Commercial Solvents Corporation, New York.



Course of patient with meningitis due to Ps. aeruginosa during intrathecal treatment with streptokinase and streptodornase. Neomycin was concomitantly administered both intramuscularly and intrathecally. A severe toxic reaction followed each intrathecal injection of streptokinase and streptodornase, but sterilization of cerebrospinal fluid occurred promptly, followed by complete recovery.

COMMENT

It is difficult to assess the role of the agents used in the successful treatment of the patient. In vitro tests indicated that neomycin was highly active against the strain of Ps. aeruginosa in the cerebrospinal fluid, and its use may have contributed materially to the control of the infection. The effect of treatment with streptokinase and streptodornase is more difficult to evaluate. There are no satisfactory clinical signs or tests by which one can reliably measure the therapeutic effect of these substances in the subarachnoid space. Moreover, the presence or absence of febrile reactions and other evidences of toxicity do not appear to correlate with the therapeutic effect and, in fact, may represent in part chemical irritation by other substances in the streptococcic concentrate. In the evaluation of the treatment of this patient, two further points are worthy of comment. First, the fact that recovery has been reported in three cases in which polymyxin B alone was used (despite two relapses) indicates that antimicrobial therapy alone may be effective. Second, Tillett and his co-workers have reported the recovery of a patient with meningitis due to Ps. aeruginosa following one intrathecal injection of streptokinase.[8] The recovery occurred after several months of failure to cure the patient with intensive antimicrobial therapy (although neomycin or polymyxin B were not given). In this case there was little doubt that the use of streptokinase and streptodornase was responsible for the patient's recovery, whether by permitting exposure of the infecting organism to the previously ineffective antimicrobial agents or by assisting host defenses by lysis of the exudate. The further possibility that the streptococcic concentrates might directly affect Pseudomonas organisms cannot be entirely excluded, but in vitro tests have failed to show inhibitory activity by this substance. Moreover, there was no change of the inhibitory activity of chloramphenicol against a strain of Ps. aeruginosa when streptococcic concentrate was added to the test.[9]

From the case reported by Tillett and his associates, and the one presently under discussion, it appears that intrathecal injection of streptokinase and streptodornase may be of therapeutic value in the treatment of meningitis caused by Ps. aeruginosa.

The toxic reactions in the present case appeared to result from the enzyme therapy, since they occurred after each intrathecal injection and did not occur when neomycin was given alone. In fact, neomycin was well tolerated without apparent evidence of local or systemic toxicity. The chills, high fever, increased cerebrospinal fluid pressure, and lethargy appeared to be referable to a local action of the injected enzyme, and striking relief was obtained by the slow withdrawal of a few cubic centimeters of spinal fluid. The reactions could be explained on the basis of a rapid accumulation of cerebrospinal fluid and increased intracranial pressure after administration of the enzymes.

As has been mentioned previously, the severity of the toxic responses does not appear to be in proportion to the therapeutic effect, and conceivably the reactions could be minimized with further studies of dosage and method of administration of the agent. The toxicity may be serious, however, and in two patients with tuberculous meningitis death occurred a few hours after intrathecal injection of enzyme.[10] At present, a number of other patients with purulent meningitis have received intrathecal enzyme therapy and have tolerated the injections with a minimum of difficulty. The corticotropin was administered in an attempt to minimize the anticipated reaction to therapy, but it apparently had little effect. It does not seem possible to attribute an important role to corticotropin in the treatment of this patient.

The choice of neomycin in the present case was based on knowledge of its in vitro activity against the offending organism. So far as is known there would be no objection to combining treatment with polymyxin B or other antibiotics with intrathecal enzyme therapy. Such a combination might permit shorter periods of treatment with potentially toxic agents and, at the same time, forestall the occurrence of relapses, such as occurred in two of the three patients receiving polymyxin B alone.

SUMMARY

A patient with meningitis due to Pseudomonas aeruginosa, resistant to treatment with several antimicrobial agents, completely recovered after a course of intrathecal treatment with streptokinase and streptodornase and intrathecal and intramuscular treatment with neomycin.

First Ave. & 26th St.

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[8] Tillett, W. S.: Studies on the Enzymatic Lysis of Fibrin and Inflammatory Exudates by Products of Hemolytic Streptococci, Harvey Lect. 45: 149-210, 1952.

[9] Unpublished observations of this laboratory.

[10] Craddock, W. L., and Haddock-Suarez, J.: Terminal Hyperthermia Following Intrathecal Streptokinase Administration in a Case of Tuberculous Meningitis, Ann. Int. Med. 36: 168-171, 1952. Guthrie, T. C.: Unpublished data.

SIMPLE TRACTION DEVICE

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Recently, we were confronted with the problem of providing a method for mobilization of contractures in both hips and knees of a patient in whom none of the usual orthopedic or physical therapeutic techniques was applicable. The problem was successfully solved with the aid of a simple traction device that was built in a short time without the use of complicated machinery. The purpose of this paper is to make generally available the knowledge of this additional, easily constructed physical therapeutic aid.

The construction of this device is illustrated diagrammatically in figure 1. We used a masonite® board ⅜ in. by 16 in. by 18 in. and two pieces of plywood, each ½ in. by 6 in. by 6 in. The latter were made into two small carriages by attaching four small metal wheels to each. The wheels could spin freely because friction was low. The carriages moved easily with only slight pushing or pulling. Such pushing or pulling force was provided by the weight of the patient's legs while the device was in operation.

REPORT OF A CASE

A 40-year-old white salvage dealer sustained third degree burns while cutting up an old car with an acetylene torch. The gas tank had exploded, and burning gasoline had doused his legs. For nine weeks the patient was treated in various hospitals and at home. When he finally came to the hospital, he had lost 50 lb., and strong flexion contractures in both hips and knees had developed. Passive range of motion in these joints was about 10 degrees; active movements were almost completely lacking. Black leathery eschar covered part of the burned areas on both lower legs and feet. Greenish, purulent discharge came from beneath the fissures. Wet compresses were ordered to prepare the wounds for skin grafting. In the meantime the patient was referred to the department of physical medicine for restoration of normal function of the contracted joints and for strengthening of the severely atrophied muscles of both legs.

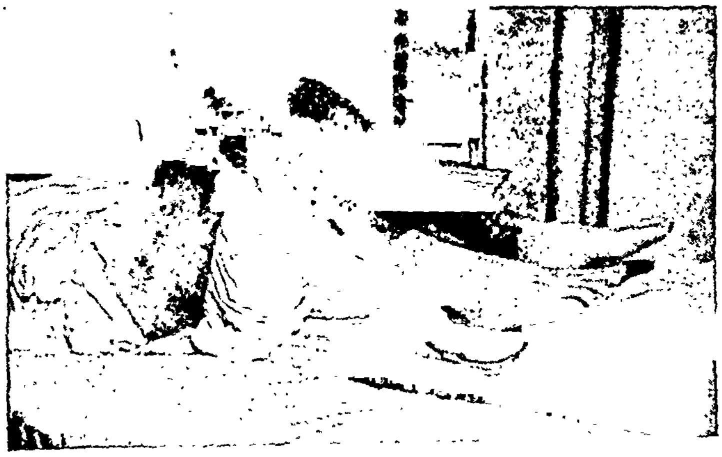

Fig. 2.—Position of patient's legs at beginning of traction therapy.

None of the usual orthopedic or physical therapeutic techniques for mobilization of hip or knee contractures could be used in this case. Adhesive tape traction requires healthy skin, of which the patient did not have enough left; skeletal traction could not be considered, because it would have been necessary to drill the Kirschner wire through denuded and infected skin, which could result in osteomyelitis; passive and assistive motion would have been too paniful, because the legs could not be gripped without including parts of the burned areas; manual traction could not be performed, because what healthy skin was left had become macerated from the wet packs and was too slippery to provide a firm grip. Nevertheless, the joints had to be moved by some method as soon as possible to prevent a seriously crippling condition to result from the contractures.

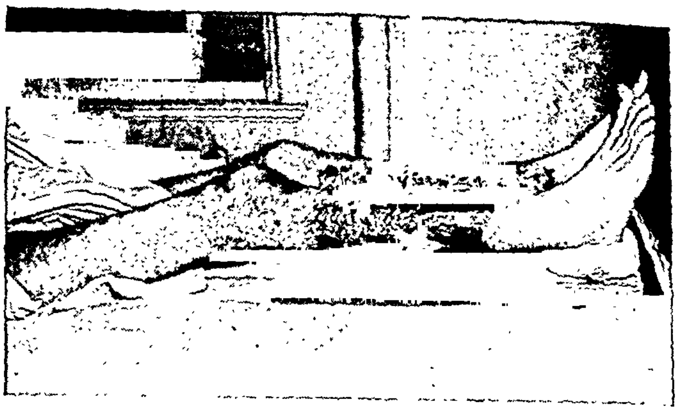

Fig. 3.—Position of patient's legs, with contractures corrected, 24 hours after traction therapy was instituted.

The problem was solved by use of the traction device described. The masonite® board and the two small carriages were put on the bed. Both heels were gently lifted onto gauze "doughnuts" and, with these, onto the carriages (fig. 2). A folded towel was placed under the foot end of the masonite® board to produce an inclined plane, with the foot end of the board higher than the head end. This was necessary because the patient's pain was too severe while the board was in the horizontal position. With the board inclined in the only position the patient could tolerate, it was noted that the carriages had a tendency to roll toward the head, thus increasing the flexion contractures; however, the weight of the legs not only nullified this tendency but even made the carriages move gradually against gravity toward the foot end of the board. It was hoped this would eventually straighten out the contractures. The patient was left in this position, since he did not complain of pain. To the surprise of all concerned, the patient's legs were almost straight after only 24 hours on the traction device (fig. 3). The patient then started active hip and knee exercises, using the device for assistive force as long as his muscles were too weak to perform these movements unassisted. When skin grafting was done, on one leg at a time, exercises were continued on the leg not being surgically treated. Exercises were resumed on the leg that had been operated on as soon as the graft had sufficiently taken. The subsequent hospital course was uneventful.

SUMMARY AND CONCLUSION

Multiple joint contractures were straightened out quickly, yet gently, by the use of a simple traction device. This device also provided assistance to the atrophied muscles for active motion. The same equipment can be used effectively for arthritic patients or postoperatively in cases in which an effective traction device is required. The tractive force is easily regulated by merely changing the incline of the board according to the patient's tolerance. The apparatus is inexpensive and can be improvised without difficulty.

From the Veterans Administration Hospital, Jefferson Barracks, Mo.

Because of space limitations, figure 1 has been omitted from THE JOURNAL and will appear in the authors' reprints.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

CONGENITAL THYROID HYPERPLASIA IN TWINS

REPORT OF A CASE FOLLOWING ADMINISTRATION OF THIOURACIL AND IODINE TO MOTHER DURING PREGNANCY

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and
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The purpose of this communication is to record the occurrence of hyperplasia of the thyroid in newborn twins following the administration of thiouracil and iodine to the mother during gestation. To our knowledge no other case of apparently induced hyperplasia of the thyroid in humans has been reported. Experimentally hyperplasia of the thyroid has been produced in puppies by thyroidectomy upon the mothers. In regions where endemic goiter prevails, the occurrence of spontaneous congenital enlargement of the thyroid is not rare.

REPORT OF CASE

History.—The first of normally delivered twin girls was stillborn. The mother, aged 36, who had previously borne six healthy children, was receiving treatment for thyrotoxicosis. For five months she had received strong iodine (Lugol's) solution, 10 drops three times daily, and propylthiouracil, 100 mg. four times daily, with amelioration of her symptoms and reduction of the basal metabolic rate from +36 to +11%.

External Examination.—The examination of the stillborn infant began one hour after delivery, Nov. 23, 1951. Permission to open the head was not obtained. Externally the cadaver appeared normal, except that there was a conspicuous smooth tumor, 3 cm. in diameter, at the middle of the neck. The body length (estimated) was 65 cm., the weight 3,000 gm. The entire skin surface was smooth and pink, with no lividity anywhere. There was no rigor mortis. The orifices were empty and without discoloration.

Internal Examination.—The single abnormal finding was the enlarged thyroid, which measured 4.5 by 3.5 by 2.5 cm. and weighed 32 gm. The cut surface was uniform, being slightly reddish, slightly firm, and homogeneous except for slender lobular septums. The lungs were totally unexpanded; the left myocardial wall was contracted. The thymus, the adrenal glands, and all other thoracic, abdominal, and pelvic viscera were normal.

Histological Study.—The sections of the thyroid were uniform and consisted of closely arranged empty follicles separated by loose reticular tissue in which there were numerous capillaries. The follicles varied only moderately in size and were lined by simple cuboidal epithelium, with no parathyroid tissue in any of the sections. There was no demonstrable lesion of the thymus, adrenals, kidneys, liver, pancreas, or spleen. The prenatal atelectasis of the lungs appeared uncomplicated.

The surviving twin sister has an enlarged thyroid but appears normal otherwise.

SUMMARY

The occurrence of congenital hyperplasia of the thyroid, apparently induced, in twins, one of them stillborn, is reported.

From the pathology, surgical, and obstetrical services, John D. Archbold Memorial Hospital.

COUNCIL ON PHARMACY AND CHEMISTRY

REPORT TO THE COUNCIL

The following abstract of recent nomenclature decisions of the Council is authorized for publication.

R. T. Stormont, M.D., *Secretary.*

NEW GENERIC AND BRAND NAMES RECOGNIZED BY THE COUNCIL

The Council collaborates with manufacturers in the selection of generic and brand names for marketed drug preparations presented for acceptance and also for new products still under development or clinical trial. The last report on such names appeared in The Journal, May 3, 1952, p. 63.

The following abstract lists the generic designation, the chemical name, where necessary for information, and the brand name or names simultaneously recognized for the stated firms. In general, a generic name should bear recognizable relation to the chemical name and conform as closely as consistent with brevity and practicality to existing systems of scientific nomenclature. The chief requirement for a brand name is that it should not be therapeutically suggestive.

The listing of a brand name is not to be construed as indicating Council acceptance of the product itself. Products accepted for inclusion in N. N. R. are announced separately.

Basic Aluminum Carbonate Gel for an aluminum hydroxide-carbonate complex: Baseljel (Wyeth, Inc.)

Bimethoxycaine Lactate for Bis[β-(*o*-methoxyphenyl)isopropyl]amine lactate: Isocaine Lactate (The Upjohn Company)

Calcium *p*-Aminosalicylate: Pasara Calcium (Smith-Dorsey)

Carbacrylamine Resins for the following combination of the component ion exchange resins: carbacrylic resin, potassium carbacrylic resin, and polyamine-methylene resin: Carbo-Resin (Eli Lilly and Company)

Carbacrylic Resin and Potassium Carbacrylic Resin for the hydrogen and potassium forms of cross-linked polyacrylic polycarboxylic ion exchange resin

Chlormerodrin for the mercurial diuretic compound 3-chloromercuri-2-methoxypropylurea: Neohydrin (Lakeside Laboratories, Inc.)

Chlorotrianisene for the synthetic estrogen tri-(*p*-anisyl)chloroethylene: Tace (The Wm. S. Merrell Company)

Cyanocobalamin for vitamin B_{12}

Iproniazid for 1-isonicotinyl-2-isopropyl hydrazine: Marsilid (Hoffmann-LaRoche, Inc.)

Isoniazid for isonicotinic acid hydrazid (isonicotinylhydrazine): Nydrazid (E. R. Squibb & Sons) and Niadrin (Endo Products, Inc.)

Neomycorsone for a mixture of neomycin sulfate and cortisone acetate: Neosone (The Upjohn Company)

Pentaerythritol Tetranitrate: Peritrate Tetranitrate (Chilcott Laboratories, Division of the Maltine Company)

Propylhexedrine for 1-cyclohexyl-2-methylaminopropane: Benzedrex (Smith, Kline & French Laboratories)

Testosterone Cyclopentylpropionate for testosterone betacyclopentylpropionate

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TREATMENT OF CANCER

The treatment of cancer, once limited to the use of surgery, x-rays, and radium, has been expanded considerably in recent years. New anticancer drugs have been developed, and hormones and radioactive isotopes have been applied to the therapy of malignant disease. These new agents represent valuable additions to the therapeutic program, but the optimistic publicity that has accompanied their use has tended to obscure several facts. In the first place, most of these substances have a relatively narrow range of therapeutic activity. The anticancer drugs, for example, are effective almost exclusively against the lymphomas, the sex steroids act only on a few types of carcinoma, and other substances are similarly limited in their action. Moreover, while these agents may prolong life and make it more comfortable, they are for the most part palliative rather than curative. Permanent cures are still achieved largely with surgery and/or external irradiation administered to patients with early lesions that have not yet metastasized widely. In spite of the many new techniques of treatment, most patients with advanced cancer still die of their disease. The search for truly curative anticancer agents with broad ranges of therapeutic activity continues, but, until such agents are found, surgery and irradiation remain the mainstays of therapy for most types of malignant lesions.

In cancer surgery, progress has been made along several fronts. More radical surgery, made practical by advances in anesthesiology, blood and electrolyte replacement, endocrine substitution, and control of infections, has expanded the concept of operability and made it possible to relieve symptoms and prolong life in many cases of cancer with extensive local spread. Total pneumonectomy for bronchogenic carcinoma, pelvic exenteration for advanced uterine, bladder, and rectal carcinoma, and radical resection of the mandible for carcinoma of the floor of the mouth are examples of this type of procedure. In general, surgical removal of metastatic lesions in distant organs has not proved effective, but in some cases apparent cure has followed the excision of solitary pulmonary lesions derived from uterine or rectal neoplasms, and removal of metastatic adenocarcinomas involving the central nervous system has been found to be a useful procedure. Many palliative operations have been developed for use when direct attack on a tumor is not possible. These include new and improved techniques for relief of malignant obstruction of the urinary, gastrointestinal, or biliary tracts, and operations designed to inhibit tumor growth by alteration of the hormone balance in patients with tumors of the breast and reproductive system. Castration and adrenalectomy are examples of the latter category. These procedures actually constitute an indirect form of chemical therapy.

The field of radiation therapy has also seen remarkable technical advances, many of them by-products of research on atomic energy. Although treatment was once limited to the use of medium voltage x-rays, radium, and radon, the radiotherapist now has at his disposal supervoltage x-ray generators, new types of radiation from the cyclotron, betatron, and other particle accelerators, and many new radioactive substances that can be applied to malignant lesions in a variety of ways. Unfortunately, clinical results have not been greatly improved by the application of these new techniques, although in some instances trial has been on too small a scale to permit final evaluation. Supervoltage (2,000 to 4,000 kv.) x-ray generators, with their increased depth dose per unit skin dose, have not given significantly better results in the treatment of deep-seated cancer than medium voltage (100 to 400 kv.) x-ray machines. The even more penetrating (10,000 to 70,000 kv.) x-rays that can be produced by the betatron have the practical disadvantage of a high skin dose at the point of exit of the beam from the body. It appears that the relative depth dose of x-rays can be increased more effectively and safely by modification of the technique of irradiation than by changes in the quality of the rays. Multiple field therapy, rotational therapy (rotation of the patient with the x-ray beam centered on the lesion), and the protection of the skin with grids or perforated shields have been effective in this regard. Among the newly available types of radiation produced by accelerators, the extremely high-energy, positively charged particles, notably protons, have shown the most promise in the treatment of deep lesions, because of the relatively low ionization that they produce at the surface of the body and the extremely dense ionization toward the end of their range. Neutron irradiation has not given results superior to those obtainable with x-rays. Neutron irradiation of tumors previously infiltrated with material that can be made radioactive by such bombardment is still in the experimental stage.

The use of radioactive isotopes has made the administration of radiation a much more flexible procedure but has not yielded types of radiation or biologic effects fundamentally different from those previously available. Radioactive isotopes in the form of solids, solutions, or colloidal suspensions may now be used not only topically and interstitially but also orally, intravenously, intra-arterially, by instillation into body cavities, and at a distance from the body. These procedures often permit better localization of radiation than has heretofore been possible. Localization may be metabolic, as when systemically administered radioactive iodine (I^{131}) is localized in thyroid tissue, or it may be physical, as when intraperitoneally administered radioactive gold colloids are deposited on the surface of the peritoneum involved by neoplastic implants or are carried to regional nodes via the lymphatics. Immunological localization has been attempted by preparing radioactive organ-specific antibodies, but none of these has been found useful in treatment. Mechanical localization can be achieved by infiltration of tissues with radioactive colloidal suspensions or by interstitial implantation of flexible wires or sutures containing radioactive substances such as cobalt 60 or gold 198. Effective irradiation of superficial lesions can be accomplished with surface applicators containing radioactive phosphorus or strontium, both of which give off beta rays free of penetrating gamma rays. Ruthenium 106 is also a useful source of beta rays. Gamma teletherapy is now possible with machines containing large quantities of radioactive cobalt, and units containing europium 152-154 or cesium 137 are being tested.

The chemical attack on cancer continues to be the subject of intensive research. This approach is based on the principle that the metabolic and mitotic activity of most neoplastic cells is greater than that of most normal cells and that this increased activity makes the neoplastic cell more susceptible to the cytotoxic action of radiation and many chemicals. The fact that some rapidly growing normal cells in the bone marrow and intestine are also damaged by these same agents indicates that the quantitative differential is not great. This, added to the fact that cancer cells, like bacteria, are capable of developing resistance to therapeutic agents, accounts for the many failures in the drug therapy of cancer. Qualitative metabolic differences in cancer cells that can be utilized in treatment have not been described.

Many anticancer drugs have been tested, but only a few have had wide clinical trial. Nitrogen mustards given intravenously have little effect on carcinomas but produce temporary remissions in a high percentage of patients with Hodgkin's disease, lymphatic leukemia, lymphosarcoma, giant follicular lymphoblastoma, and mycosis fungoides. Given intra-arterially, nitrogen mustard has produced local destruction of carcinoma of the tongue, lung, kidney, and other organs, but cures have not been obtained. Triethylene melamine (TEM) given orally or intravenously produces results similar to those obtained with nitrogen mustard in the lymphomas. Folic acid antagonists ("aminopterin" or "a-methopterin") are useful chiefly in acute leukemia of children. Urethan (ethyl carbamate) given orally produces clinical improvement in chronic leukemia, especially in the myelogenous variety.

Other, more questionable, treatments have been proposed. Fowler's solution (potassium arsenite) is effective particularly in Hodgkin's disease and chronic myelogenous leukemia, but treatment must be prolonged and the incidence of toxic manifestations is high. Stilbamidine (4,4′-stilbenedicarboxamidine) and related compounds produce symptomatic relief in multiple myeloma, but there is little objective improvement. A polysaccharide extracted from Serratia marcescens (Bacillus prodigiosus) produces hemorrhagic changes in animal tumors and occasionally a decrease in the size of lymphomatous or sarcomatous tumors in man, but it also produces fever and severe hypotension and is regarded as unpredictable and hazardous. Colchicine, resin of podophyllin, antireticular cytotoxic serum, K-R (an extract prepared from killed Trypanosoma cruzi), H. 11 (an extract of the urine of normal persons), and "krebiozen" are among the agents that have been advocated in some quarters but have not been generally accepted by experts in the cancer field.

Hormone therapy has been found beneficial only in certain kinds of cancer, and in these it is believed that the environment of the tumor rather than the tumor itself is affected. Estrogens produce regression of local and metastatic lesions in carcinoma of the prostate and may prolong life for many years. They are also of benefit in postmenopausal carcinoma of the breast and appear to have some effect on carcinoma of the bladder and chorioepithelioma. Androgens have a palliative effect in carcinoma of the breast with extensive metastasis. There is equivocal evidence that progesterone has value in carcinoma of the breast, cervix, and prostate. Cortisone and corticotropin have no consistent effect on the objective manifestations of carcinoma, but they do inhibit to some extent the growth of the malignant lymphomas. In the acute leukemias they produce spectacular remissions of short duration; in chronic leukemias the results are unpredictable, with aggravation of the disease sometimes occurring. The one feature of chronic leukemia that is regularly benefited, however, is the hypersplenic phase. When hemolytic, neutropenic, and thrombopenic crises occur, cortisone or corticotropin produces the same benefits as splenectomy, even when x-rays and other measures have failed to do so.

Other approaches to the therapy of cancer that have been employed experimentally include the use of viruses that selectively attack and destroy neoplastic cells, the attempt to stimulate immunity to cancer in the host by inoculation with extracts of tumor and other tissues, and the destruction of superficial tumors with ultrasonic waves. None of these approaches is yet of practical value in man.

In the current preoccupation with specific therapy, sight is often lost of the fact that the patient as well as the cancer must be treated. The patient's morale must be maintained, and general supportive treatment must be utilized. The relief of secondary infection with antibiotics, of disturbances in fluid, electrolyte, and nutritional balance with parenteral fluids, of any associated congestive heart failure with standard remedies, and of anemia with transfusions will often do more to make the patient comfortable than any specific measures available. It should be noted, however, that iron therapy has no value except in the treatment of hypochromic anemia following hemorrhage. Liver extract and supplementary vitamin B_{12} are equally valueless unless there is a specific deficiency of these substances.

The solution to the problem of specific anticancer therapy awaits a better understanding not only of the metabolism of cancer cells but of the mechanism of normal growth as well. For the acquisition of this knowledge unrestricted basic research may well be as important as "project" or restricted research. Support and coordination of research is being provided in the United States on an unprecedented scale by such organizations as the American Cancer Society, the American Association for Cancer Research, the National Cancer Institute, the Atomic Energy Commission, the Committee on Growth of the National Research Council, and the Committee on Research of the A. M. A.'s Council on Pharmacy and

Chemistry. An example of the type of activity sponsored by the last-named committee is the continuing cooperative study on the use of steroid hormones in the treatment of advanced breast, thyroid, and genitourinary carcinoma. The results of the breast carcinoma study, in which 45 clinics and 14 pharmaceutical firms participated, have been published in THE JOURNAL.[1]

The development of improved methods of cancer therapy seems certain. In the meantime, over-optimism concerning new remedies must be guarded against assiduously. Every effort must be made on the part of scientists, science writers, and pharmaceutical manufacturers to prevent the release to the lay press of overenthusiastic news stories of new "wonder drugs" or therapeutic procedures. The false hopes raised by such stories have caused patients to postpone urgent surgery or to refuse irradiation until their lesions have advanced beyond all hope of control. Persons in high government office and other responsible positions also have an obligation to the public to refrain from promoting new and inadequately tested therapeutic measures. Such misguided activity can result in incalculable harm to the health of the public.

1951 EXPENDITURES FOR MEDICAL CARE

The Bureau of Medical Economic Research has recently published another item in its series on personal consumer expenditures for medical care.[1] The basic data are from the July, 1952, issue of the *Survey of Current Business,* United States Department of Commerce. New data are given for 1951 and some slight revisions of the previously published data for 1949 and 1950.

Briefly stated, the percentages of total personal consumer expenditures spent for medical care and for each of the major items of medical care were about the same in 1951 as they were in 1950. The percentage spent for medical care was 4.3% in 1951 and 4.3%, according to revised data, for 1949 and 1950. The percentages ent for the services of physicians and of hospitals continue to be 1.2 and 1.0 respectively. The percentage of the medical care dollar spent on physicians' services in 1951 was 28.1, as compared with 28.3 in 1950 and 28.9 in 1949 according to the revised data for the two earlier years. On the other hand, the share of the medical care dollar going to hospitals reached an all-time high of 23.8% in 1951 and the revised data indicate 22.0% for 1949 and 23.4% for 1950. The dentists' share of the medical care dollar was 11.9% in 1949, 11.5% in 1950, and 11.0% in 1951.

Personal consumer expenditures for medical care rose from 7.8 billion dollars in 1949 to 8.4 billion dollars in 1950 to 9.0 billion dollars in 1951. Total personal consumer expenditures for all goods and services, however, rose slightly faster, from 180.6 billion dollars in 1949 to 194.3 billion dollars in 1950 to 208.0 billion dollars in 1951. Thus the American people continue to devote only slightly more than 4% of their total consumer expenditures to medical care items, among which hospital care bulks larger and larger.

GENERAL PRACTITIONER AWARD

The General Practitioner Award was presented for the first time in January, 1948, at the Association's meeting in Cleveland. Since then it has been awarded annually at the midyear session. It has been the practice for the Board of Trustees to select three names from those submitted by the state societies and present them to the House of Delegates for its choice. At its meeting in Chicago in June, 1952, the House approved the following communication from the Board of Trustees:

> It is believed that a much more dignified and fair approach would be to have the Chairman of the Board appoint an impartial committee, to remain anonymous until after the award is made, which would study the credentials and choose from the nominations submitted the general practitioner most qualified for the honor.

To meet the wishes of the House, biographical material submitted by various state medical societies for the General Practitioner Award should be in the office of the Secretary of the American Medical Association no later than Nov. 1.

SCIENTIFIC EXHIBIT, DENVER SESSION

The Scientific Exhibit will again be one of the features of the Clinical Session in Denver, Dec. 2 to 5, 1952. All activities of the session, except the House of Delegates, will be housed in the remodeled and enlarged Municipal Auditorium. The Scientific Exhibit will be located on the ground floor of the auditorium, together with facilities for colored television, motion pictures, and several of the meeting rooms.

Application blanks are now available for space in the Scientific Exhibit, and prospective authors of exhibits should make application as soon as possible. Attention is called to the fact that the Denver Session is a clinical meeting and hence emphasis is placed largely on the clinical aspects of medicine rather than on research activities.

The Committee on Scientific Exhibit is composed of three members of the Board of Trustees: L. W. Larson, M.D., Bismarck, N. D., chairman; Thomas P. Murdock, M.D., Meriden, Conn.; and James R. McVay, M.D., Kansas City, Mo. The subcommittee on Scientific Exhibit in Denver includes Dumont H. Clark, M.D., chairman; Thomas M. Van Berger, M.D.; and Marvin E. Johnson, M.D.

Application blanks for space may be obtained from the Director, Scientific Exhibit, American Medical Association, 535 N. Dearborn St., Chicago 10.

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[1] Estrogens and Androgens in Mammary Cancer, Report to the Council on Pharmacy and Chemistry, J. A. M. A. **140:** 1214 (Aug. 13) 1949. Proceedings of the First Conference on Steroid Hormones and Mammary Cancer, Chicago, A. M. A. (April) 1949. Current Status of Hormone Therapy of Advanced Mammary Cancer, Report to the Council on Pharmacy and Chemistry, ibid. **146:** 471 (June 2) 1951. Lazlo, D., and others: Effect of Testosterone on Patients with Bone Metastases: A Metabolic Study, Particularly of Breast Carcinoma, Report to the Council on Pharmacy and Chemistry, ibid. **148:** 1502 (April 26) 1952.

[1] Dickinson, F. G.: 1951 Expenditures for Medical Care, Publication M-69, Chicago, American Medical Association, 1952. The latest item in this series had been Medical Care Expenditures, Prices and Quantity, 1930-1950, J. A. M. A. **147:** 1354-1359 (Dec. 1) 1951, which was reprinted as Bulletin 87. Publication M-69 is really a small supplement to Bulletin 87. A copy of these two publications may be obtained from the Bureau of Medical Economic Research, American Medical Association, 535 N. Dearborn St., Chicago 10.

MEDICAL NEWS

CALIFORNIA

Warning—Epidemic Encephalitis.—Dr. Wilton L. Halverson, state director of public health, has issued a warning that encephalitis may become epidemic in the Central Valley of California this year unless intensified action against the mosquito that transmits the disease is taken by control agencies and individual householders alike. The threat of a mosquito-borne epidemic is indicated in reports of both human and equine cases of encephalitis in the San Joaquin Valley, coupled with the appearance of extremely large numbers of Culex tarsalis mosquitoes.

At a meeting of representatives of the vector control advisory committee, State Department of Public Health, and representatives of mosquito abatement districts in the Central Valley, preventive and remedial measures were suggested. Individual householders were urged to eliminate mosquito breeding sources around their premises, keep adult mosquitoes out of the house by careful screening of doors and windows, and use hand sprays or aerosol bombs to kill mosquitoes getting indoors. All persons, and particularly children, were advised to remain indoors as much as possible during evening hours, when Culex tarsalis usually attacks, and to use mosquito repellents when outdoors.

Health departments were asked to be on the alert, maintaining close contact with private physicians, in order to provide an up-to-the-minute statewide picture, with the earliest possible warning of epidemic conditions. Encephalitis cases in the high-prevalence areas of the Central Valley normally reach a peak in July, August, and September. Mosquito abatement districts were urged to intensify their control measures directed specifically at the Culex tarsalis species, and irrigation districts and individual ranchers were asked to support the control measures, giving particular attention to irrigation practices to prevent unnecessary standing water, which would create mosquito breeding places.

ILLINOIS

Health at the State Fair.—Many phases of health are emphasized at the Illinois Centennial State Fair, which is being held at Springfield Aug. 8-17. In addition to displays of health subjects throughout the fairgrounds, free movies, and dental and chest x-ray examinations are provided. Inside the Illinois Building are displayed improvements in sanitation during the past 100 years. As part of the tuberculosis case finding program, the State Department of Public Health, in cooperation with the Illinois Tuberculosis Association, is offering x-ray examinations of the chest. The Department of Public Health is also sponsoring a special "health tent" with exhibits and motion pictures on many health subjects.

Chicago

Fellowship in Pediatric Cardiology.—The Children's Division of Cook County Hospital in Chicago has an opening for a fellow in pediatric cardiology. Training in angiocardiography and catheterization will be available if desired. Information may be obtained from Dr. Benjamin M. Gasul, 700 S. Wood St., Cook County Children's Hospital, Chicago.

Dr. MacEachern Honored.—Dr. Malcom T. MacEachern, director of professional relations for the American Hospital Association, was presented with a badge bearing the coat of arms of the Comité des Hôpitaux du Quebec (the Committee of Catholic Hospitals of Quebec) and a citation in recognition of his work in the hospital field, during the session of the Comité in Quebec City, June 23-25. In July Dr. MacEachern was in Brazil, South America, where he gave a three week lecture course to students in hospital administration at the University of São Paulo. While in Brazil he also participated in an Institute for Hospital Administrators and served as consultant on local, regional, state, and national organization of hospitals. The trip was arranged under the auspices of the University of São Paulo.

IOWA

State Medical Society Election.—At its meeting in Des Moines, April 27-30, the Iowa State Medical Society elected Dr. Ben T. Whitaker, Boone, president; Dr. Robert N. Larimer, Sioux City, president-elect; Dr. Elias B. Howell, Ottumwa, first vice-president; Dr. Lester C. Kern, Waverly, second vice-president; Dr. Allan B. Phillips, Des Moines, secretary; and Dr. N. Boyd Anderson, Des Moines, treasurer.

Dr. Barnes Retires from Faculty.—Dr. Milford E. Barnes, since 1930 head of the department of hygiene and preventive medicine, University of Iowa, was honored at a testimonial dinner June 4. Dr. Barnes, whose retirement from the university faculty was recently announced, has served as director of the university's department of health since 1930; and since 1943 he has been consulting director of the Iowa State Hygiene Laboratory, which he served as director for many years.

Before coming to the university he was a field director under the international health division of the Rockefeller Foundation. A wrist watch and a morocco-bound book, containing more than 100 letters from former students and co-workers, was presented to Dr. Barnes. Greetings from individual friends, societies, and organizations in various countries were read.

MINNESOTA

Dr. Buie Receives Alumni Award.—Dr. Louis A. Buie, professor of surgery in the Mayo Foundation, University of Minnesota, Rochester, has been given the 1952 Alumni Honor Award and Gold Key by the Medical Alumni Association of the University of Maryland "for outstanding contributions to medicine and distinguished service to mankind." The presentation was made June 5, when Dr. Buie addressed the association on "The Fruit of Loyal Nature and of Noble Mind."

Before becoming chief of the department of proctology at the Mayo Clinic in 1919, Dr. Buie was affiliated with the University of Maryland hospital and James Lawrence Kernan Hospital and Industrial School for Crippled Children in Baltimore. He is a past president of the American Proctologic Society, secretary of the American Board of Proctology, chairman of the Council on the Constitution and By-Laws of the American Medical Association, and a member of the Judicial Council of the Association and the Committee on Education and Publications of the National Foundation for Infantile Paralysis, Inc.

NEW YORK

Genesee County Sesquicentennial.—Medicine played a prominent part in the week-long observance of Genesee County's Sesquicentennial celebration through the activities of the Genesee County Medical Society and the Woman's Auxiliary of the Genesee Memorial Hospital. The medical group had a float in the Batavia parade portraying the advance of medicine since the founding of Genesee County 150 years ago. Dr. Charles M. Graney, Batavia, was chairman of the County Medical Society's Exhibition Committee; Dr. Alfred L. George, Batavia, wrote and directed the pageant, which had a cast of 500 characters.

New York City

Dr. Wolf Appointed Dean in Vermont.—Dr. George A. Wolf Jr., director of the outpatient department of New York Hospital and assistant professor of clinical medicine at Cornell Medical College, has been appointed dean of the college of medicine of the University of Vermont, Burlington, and professor of clinical medicine. He succeeds Dr. William E. Brown, who retired June 30.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

Report on Tuberculosis.—In its annual report for the 50th year of an organized drive against tuberculosis, the New York Tuberculosis and Health Association points out that though fewer persons are dying of the disease today, the incidence continues at an alarmingly high level and out of proportion to the medical and social progress of the last half century. During the last 20 years the incidence has not materially changed: there were 17,645 cases of pulmonary tuberculosis in New York at the end of 1931 as compared to 17,911 cases on Dec. 31, 1951. A slow but steady increase of cases has marked the last five years. Of the 13 city hospitals caring for tuberculosis patients, 6 have more than 100% bed occupancy; 85% of capacity is the normal hospital occupancy, but according to the report none of the 13 operates at this figure.

OKLAHOMA

Insurance Company Cancels Malpractice Policy.—The London and Lancashire Indemnity Company of Hartford, Conn., has cancelled the master policy held with the Oklahoma State Medical Association for the writing of malpractice insurance for its members. The company stated that it was withdrawing from this field of insurance. Physicians whose malpractice insurance was with the London and Lancashire will find it necessary to place this coverage with companies that are now writing it in Oklahoma. The insurance committee of the association is attempting to negotiate a new contract with another company. Over 985 members of the association held contracts with the London and Lancashire.

PENNSYLVANIA

100 Years of Pennsylvania Medicine.—"A Century of Medicine, 1848-1948," a history of medicine in the Commonwealth of Pennsylvania, has just been published by the State Medical Society. Among the medical "firsts" in this country, which it lists for Pennsylvania, are the following: the first general hospital, first permanent medical periodical, and the first board of health. The first woman's medical school in the world was established in Philadelphia more than a century ago. The Medical Society of the State of Pennsylvania was the 16th state society to be organized, coming into being one year after the founding of the American Medical Association.

Philadelphia

Antivirus Research.—Schenley Laboratories, Inc., Lawrenceburg, Ind., has announced a grant of $17,000 to the Langbord Virus Laboratory at Hahnemann Medical College and Hospital, for antivirus research. The studies will be made by Dr. Bernard A. Briody, Ph.D., director of the virus laboratory.

Dr. Long Appointed to UNESCO Commission.—Dr. Esmond Ray Long was recently appointed a member of the U. S. National Commission of UNESCO, as representative of the National Academy of Sciences, National Research Council. He succeeds Detlev W. Bronk, Ph.D., president of Johns Hopkins University, Baltimore.

Director of the Henry Phipps Institute for Study, Treatment and Prevention of Tuberculosis, Dr. Long served as chief consultant on tuberculosis to the Surgeon General's Office, U. S. Army, during World War II, and later directed the tuberculosis section of public health in the U. S. zone in Germany. He has served as president of the National Tuberculosis Association, American Association of Pathologists and Bacteriologists, and the American Association of the History of Medicine.

SOUTH CAROLINA

Man of the Year.—Dr. Walter R. Mead, Florence, was the recipient recently of the "Man of the Year" civic service award, sponsored annually by the Rotary, Kiwanis, Optimist, Lions, and Exchange clubs of Florence.

Cancer Award.—Dr. Thomas A. Pitts, chairman of the Board of Trustees of the Medical College of the State of South Carolina, Charleston, was awarded the medal of the American Cancer Society for outstanding service in the cancer program for 1951. The citation was made by the South Carolina Division of the American Cancer Society, "in recognition of distinguished service in the control of cancer." He has been director of the South Carolina Baptist Hospital Cancer Clinic since it was organized and has also directed a cancer detection clinic there for the past four or five years.

VIRGINIA

Dr. Caudill Honored.—Dr. Walter Cleveland Caudill of Pearisburg was recently honored by his alma mater, the Medical College of Virginia in Richmond, which conferred on him the honorary degree of master of science in general medicine. Dr. Caudill was president of the Medical Society of Virginia (1950-1951), and in 1951 the House of Delegates of the Medical Society of Virginia selected him as "General Practitioner of the Year." For several years he has served in the Virginia State Legislature.

WISCONSIN

Health Day for Business Executives.—On July 30 the University of Wisconsin Industrial Management Institute sponsored an executive leadership program for business men. The faculty of the University of Wisconsin Medical School, Madison, cooperated in providing information on the symptoms of coronary thrombosis, nervous disorders, cancer, and tuberculosis. Dr. John W. Brown, director of the university's department of student health, who was in charge of the health day program, stated that the program was planned "to help the group understand how medical men diagnose and treat ailments that are especially prevalent among businessmen . . . to make available to business executives the most advanced scientific knowledge that will help lengthen their lives."

GENERAL

Dates Changed for Graduate Course in Urology.—The American Urological Association announces that the postgraduate course in urology, known as the South Central Section Seminar, which was scheduled to be given in Kansas City, Mo., Dec. 14-18 (The Journal, June 14, p. 698), will be given instead in the same city on Jan. 11-14, 1953. Information may be obtained from the headquarters of the association, 102-110 Second Ave., S.W., Rochester, Minn.

Proposed Nation-Wide Medical Code for Boxing.—Measures designed to protect young boxers and to promote the exchange of information on the medical effects of athletics were discussed at a symposium July 27 in New York, under the sponsorship of the State Athletic Commission. Dr. Marvin A. Stevens, New York, member of the Medical Advisory Board of the commission, was chairman.

A plea for establishing national standards to protect boxers was made by Dr. Frank R. Ferlaino, New York, former director, State Athletic Commission's medical advisory board. Among the steps suggested by Dr. Ferlaino were the establishment of a national medical sports association for interchanging information, recognized medical criteria for stopping bouts, standardized medical equipment, and safer gloves, mats, and mouthpieces for boxers. The National Boxing Association will hold a meeting on the Medical Aspects of Boxing in Montreal, Canada, Sept. 8-10.

Dermatologists Meet in San Francisco.—The Pacific Dermatologic Association will meet in San Francisco Aug. 29-30 under the presidency of Dr. Nelson P. Anderson, Los Angeles. On Friday the sessions at the Palace Hotel will open with the presidential address at 9 a. m., which will be followed at 9:20 by a talk on "The Diagnostic Pitfalls in Dermal Pathology" by Dr. Marcus R. Caro of Chicago, clinical professor of dermatology, University of Illinois. Brief reports of scientific activities will be given by the universities of Colorado, Utah, Washington, Oregon, Southern California, California at Los Angeles, and California, Stanford University, and the College of Medical Evangelists.

The Saturday session, which will be held in the Letterman Army Hospital, will open at 9 a. m. with a demonstration of clinical techniques. At 10:30 a. m. there will be a report from the International College of Dermatology in London under the chairmanship of Dr. Norman N. Epstein of San Francisco.

Discussion roundtable groups will be held during the luncheon at the hospital, which will be followed by a clinical meeting of the hospital.

On Sunday at 9 a. m. there will be a seminar in histopathology at Letterman Army Hospital.

Prevalence of Poliomyelitis.—According to the National Office of Vital Statistics, the following number of reported cases of poliomyelitis occurred in the United States and its territories and possessions in the weeks ended as indicated:

Area	July 19, 1952	July 12, 1952	July 21, 1951
New England States			
Maine	1	3	2
New Hampshire	..	..	1
Vermont	1	1	1
	3	6	17
	..	..	..
	6	2	9
Middle Atlantic States			
New York	68	26	52
New Jersey	16	10	9
Pennsylvania	18	8	16
East North Central States			
Ohio	69	41	26
Indiana	19	5	8
Illinois	46	33	52
Michigan	56	19	37
Wisconsin	26	5	29
West North Central States			
Minnesota	27	17	6
Iowa	82	72	17
Missouri	13	5	6
North Dakota	..	..	1
South Dakota	15	15	1
Nebraska	55	35	9
Kansas	37	19	9
South Atlantic States			
	3	..	..
	7	2	2
	1	5	..
Virginia	17	13	9
West Virginia	22	17	2
North Carolina	14	11	4
South Carolina	1	..	4
Georgia	25	18	34
Florida	22	38	17
East South Central States			
	73	18	12
	24	21	21
	20	13	31
	51	38	42
West South Central States			
Arkansas	14	9	19
	83	60	41
	46	47	21
	243	206	66
Mountain States			
Montana	6	5	1
Idaho	4	3	3
Wyoming	1	1	8
	10	10	43
	19	22	1
Arizona	8	7	7
Utah	4	..	19
Nevada	..	..	..
Pacific States			
Washington	38	18	5
Oregon	19	7	4
California	59	40	70
Territories and Possessions			
Alaska	..	..	4
Hawaii	12	11	..
Puerto Rico	1	2	..
Total	1,405	1,060	798

During the "disease year," which began with the week ended July 5, a total of 5,406 cases of poliomyelitis had been reported (week ended July 19) as compared with 3,351 for the same period in 1951; in 1949, the corresponding figure was 5,313.

FOREIGN

Congress of Therapeutics.—The International Congress of Therapeutics will be held in Madrid, Spain, Oct. 16-18. The main topics for discussion will be potassium, massive hormonotherapy, and radioisotopes. Among the speakers will be Paul F. Hahn, Ph.D., Nashville, Tenn., who will discuss "Tumor Therapy by Radioisotopes."

LATIN AMERICA

Dr. Kemeny Honored.—Dr. Esteban Kemeny, director of the School of Dietetics, Santiago, Chile, has been elected president of the Nutrition Society of Chile. He recently visited the United States under the sponsorship of the Institute of Inter-American Affairs for the purpose of observing American dietetic methods.

CORRECTION

Annual License Renewal Not Required in Florida.—On page 506 of the May 31, 1952, JOURNAL under the heading "Annual Registration" is a statement to the effect that a new Florida law requires annual renewal of licenses of practitioners of medicine. Annual renewal is not required. The statement should have been to the effect that the Florida law requires annual registration of such licenses with the state board of health.

MEETINGS

American Medical Association Public Relations Institute, Edgewater Beach Hotel, Chicago, Sept. 4-5. Mr. Leo E. Brown, 535 N. Dearborn St., Chicago 10, Director.

Alaska Territorial Medical Association, Anchorage, Aug. 21-23. Dr. William P. Blanton, P. O. Box 2569, Juneau, Secretary.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, The Homestead, Hot Springs, Va., Sept. 4-6. Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Texas, Secretary.

American College of Surgeons, Waldorf-Astoria, New York, Sept. 22-26. Dr. Michael L. Mason, 40 East Erie St., Chicago 11, Secretary.

American Congress of Physical Medicine, Roosevelt Hotel, New York, Aug. 25-29. Dr. Frances Baker, One Tilton Ave., San Mateo, Calif., Secretary.

American Hospital Association, Philadelphia, Sept. 15-18. Mr. George Bugbee, 18 East Division St., Chicago 10, Executive Director

American Medical Writers' Association, Jefferson Hotel, St. Louis, Oct. 1. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

American Physiological Socety, New Orleans, Sept. 4-6. Dr. E. F. Adolph, University of Rochester School of Medicine and Dentistry, Rochester, N. Y., Secretary.

American Roentgen Ray Society, Shamrock Hotel, Houston, Texas, Sept. 23-26. Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Secretary.

American Society of Maxillofacial Surgeons, Hotel del Coronado, San Diego, Calif., Sept. 10-13. Dr. Casper M. Epsteen, 25 East Washington St., Chicago 2, Secretary.

Annual Gulf Coast Regional Conference on Industrial Health, Rice Hotel, Houston, Tex., Sept. 24-27. Dr. W. H. Hamrick, P. O. Box 2371, Houston, Tex., Chairman.

Biological Photographic Association, Hotel New Yorker, New York, Sept. 10-12. Mr. Lloyd E. Varden, Pavelle Color, Inc., 533 W. 57th St., New York 19, Secretary.

Colorado State Medical Society, Stanley Hotel, Estes Park, Sept. 9-12. Mr. Harvey T. Sethman, 835 Republic Bldg., Denver 2, Executive Secretary.

Constantinian Society, Lake Placid Club, Lake Placid, N. Y., Oct. 1-4. Dr. C. F. Shook, P. O. Box 1035-36, Toledo 1, Ohio, Secretary.

Medical Society of Delaware, Rehoboth, Sept. 8-10. Dr. Andrew M. Gehret, 822 North American Bldg., Wilmington, Secretary.

Medical Society of the District of Columbia, Hotel Statler, Washington, Sept. 29-Oct. 1. Mr. Theodore Wiprud, 1718 M St. N.W., Washington, Secretary.

Medical Society of the State of Pennsylvania, Bellevue-Stratford Hotel, Philadelphia, Sept. 28-Oct. 2. Dr. Walter F. Donaldson, 500 Penn Ave., Pittsburgh 22, Secretary.

Medical Society of Virginia, Jefferson Hotel, Richmond, Sept. 28-Oct. 1. Mr. Robert L. Howard, 1105 West Franklin St., Richmond, Executive Secretary.

Michigan State Medical Society, Book-Cadillac Hotel, Detroit, Sept. 24-26. Dr. L. Fernald Foster, 606 Townsend St., Lansing 15, Secretary.

Mississippi Valley Medical Society, Jefferson Hotel, St. Louis, Oct. 1-3. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

Montana Medical Association, Florence Hotel, Missoula, Sept. 18-21. Mr. L. R. Hegland, 104 North Broadway, Billings, Executive Secretary.

National Medical Association, Chicago, Aug. 11-15. Dr. John T. Givens, 1108 Church St., Norfolk 10, Va., General Secretary.

Neurosurgical Society of America, The Cloister, Sea Island, Ga., Sept. 18-20. Dr. C. D. Hawkes, 22 North Manassas St., Memphis 5, Tenn., Secretary.

Nevada State Medical Association, Reno, Aug. 21-23. Dr William A. O'Brien III, 505 Chestnut St., Reno, Secretary.

New Hampshire Medical Society, Mt. Washington Hotel, Bretton Woods, Sept. 7-9. Dr. Deering G. Smith, 44 Chester St., Nashua, Secretary.

North Pacific Pediatric Society, Challenger Inn, Sun Valley, Idaho, Sept. 19-20. Dr. S. Gorham Babson, 1107 S.W. 13th Ave., Portland 5, Ore., Secretary.

North Pacific Society of Internal Medicine, Sun Valley, Idaho, Sept. 19-20. Dr. Robert L. King, 1115 Terry Ave., Seattle 1, Wash., Secretary.

North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, Texas, Sept. 17. Dr. E. Aubrey Cox, Hamilton Bldg., Wichita Falls, Texas, Chairman.

Piedmont Proctologic Society, Savannah Beach, Ga., Aug. 23. Dr. B. Richard Jackson, 224 Hillsboro St., Raleigh, N. C., Secretary.

Reno Surgical Society *Conference*, Reno, Nevada, Aug. 21-23. Dr. Olin C. Moulton, 130 N. *Virginia St.*, Reno, Nevada, Chairman.

Tri-State Medical Society of Texas, Louisiana and Arkansas, Texarkana, Texas, Oct. 2-3. Dr. Albert M. Hand, 619 Main St., Texarkana, Texas, Secretary.

U. S. Chapter, International College of Surgeons, Conrad Hilton Hotel, Chicago, Sept. 2-5. Dr. Arnold S. Jackson, 1516 Lake Shore Drive, Chicago, Executive Secretary.

Utah State Medical Association, Salt Lake City, Sept. 4-6. Dr. T. C. Weggeland, 42 S. Fifth East St., Salt Lake City, Secretary.

Vermont State Medical Society, Mt. Washington Hotel, Bretton Woods, N. H., Sept. 7-9. Dr. James P. Hammond, 128 Merchants Row, Rutland, Secretary.

Washington State Medical *Association*, *Olympic* Hotel, Seattle, Sept. 13-17. Dr. Bruce *Zimmerman*, 338 White-Henry-Stuart Bldg., Seattle, Secretary.

INTERNATIONAL

Aero Medical Association Interim Meeting, Paris, France, Sept. 26-28. Dr. Armand Robert, 2 Rue Marbeuf, *Paris 8e, France*, Secretary.

American Congress of Industrial *Medicine*, Rio de Janeiro, Brazil, Sept. 20-28. Prof. Dr. Jose Pedro Reggi, Calle *Arenales No.* 981 Buenos Aires, Argentina, Director General.

Australasian Medical Congress, Melbourne Victoria, Australia, Aug. 22-30. Dr. C. H. Dickson, Medical Society Hall, 426 Albert St., East Melbourne, Victoria, Australia, General Secretary.

Congress of International *Institute* of Embryology, University of Utrecht, *Zoological* Laboratory, Utrecht, Netherlands, Aug. 27-31. Dr. M. W. Woerdeman, Amsterdam, Holland, President.

European Congress of Cardiology, University of London, Bloomsbury, W.C.1, England, Sept. 10-12. Dr. K. Shirley Smith, 35 Wimpole St., London, W.1, England, Secretary.

European Society of Cardiovascular Surgery, Strasbourg, France, Oct. 3-4. Sir James Learmonth, Department of Surgery, University New Buildings, Edinburgh 8, Scotland.

Interamerican Cardiological Congress, Buenos Aires, Argentina, Aug. 31-Sept. 7. Dr. Blas Moia, Larrea 1132, Buenos Aires, Argentina, Secretary-General.

Inter-American Congress of Public Health, Havana, Cuba. Sept. 26-Oct. 1. Pan-American Sanitary Bureau, 2001 Connecticut Ave., Washington, D. C

Inter-American Congress of Radiology, Mexico City, Mexico, Nov. 2-8. Dr. Guido Torres Martinez, Marsella No. 11, Mexico, D.F., Secretary-General.

International Congress of Anthropology and Ethnology, Vienna, Austria, Sept. 1-8. For information write: Secretaries, Royal Anthropological Institute, 52 Upper Bedford Place, London, W.C.1, England.

International Congress on Diseases of the Chest, Rio de Janeiro, Brazil, Aug. 24-30. Mr. Murray Kornfeld, 112 East Chestnut St., Chicago 11, Illinois, Executive Secretary.

International Congress of Haematology, Mar del Plata, Argentina, Sept. 21-26. Dr. Carlos Reussi, Anchorena 1710, Buenos Aires, Argentina, Secretary.

International Congress of the History of Medicine, Nice-Cannes, France, and Monaco, Sept. 8-14. Dr. F. A. Sondervorst, 124 Avenue es Allies, Louvain, Belgium, Secretary-General.

ernational Congress on Hydatid Disease, Santiago, Chile, Nov. 21-24. Organizing Committee, P. O. Box 9183, Santiago, Chile.

International Congress of Internal Medicine, Friends House, London, N.W.1, Eng. Sept. 15-18. Sir Harold Boldero, 12 Pall Mall East, London, S.W.1, England, Secretary.

International Congress of Medical Press, Venice, Italy, Oct. 4-5. Dr. Bruno de Biasio, S. Marco 4646, Venice, Italy, Secretary.

International Congress on Medical Records, London, England, Sept. 7-12. Miss Gwen Perkins, 120 C Street S.E., Washington, D. C., Chairman.

International Congress of Military Medicine and Pharmacy, Montevideo, Uruguay, March 1-7, 1953. Dirreccion General del Servico de Sanitad Militar, 8 de Octubre y Mariano Moreno, Montevideo, Uruguay, Secretariat of the Congress.

International Congress on Neuropathology, Rome, *Italy*, *Sept.* 8-13. Dr. Armando Ferraro, 722 W. 168th St., New York, N. Y., U. S. A., Secretary General.

International Congress of Otorhinolaryngology, Amsterdam, Netherlands, June 8-15, 1953. Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam, Netherlands, Secretary.

International Congress of Radiology, Copenhagen, Denmark, July 19-25, 1953. Professor Flemming Norgaard, 10 Oster Voldgade, Copenhagen K., Denmark, Secretary General.

International Congress of Therapeutics, Madrid, Spain, Oct. 16-18. Professor Armijo, Instituto de *Farmacologia*, Cuidad Universitaria, Madrid, Spain, Secretary.

International Congress of Urology, The Waldorf-Astoria, New York, N. Y., U. S. A., Sept. 15-18. Dr. John A. *Taylor*, 2 East 54th St., New York, N. Y., U. S. A., Secretary-General.

International Hospital Congress, *London*, England, May 25-30, 1953. Capt. J. E. Stone, 10 Old Jewry, London, EC2, England, Hon. Secretary.

International Study Conference on Child Welfare, Bombay, India, Dec. 5-12. For information write: All India Save the *Children* Committee, 5 Carmichael Road, Bombay, India.

International Union Against Tuberculosis, Rio de Janeiro, Brazil, Aug. 24-27. Prof. Etienne Bernard, 66 Blvd. St. Michel, Paris 6e, France, Secretary-General.

Medical Women's International Association, Vichy, France, Sept. 12-16. Dr. Margaret M. Wurts, 27 East Wellesley Road, Montclair, N. J., *U.* S. A., National Secretary.

Neuroradiological Symposium, Stockholm, Sweden, Sept. 17-20. Docent Ake Lindbom, Symposium Neuroradiologicum, Serafimerlasarettet, Stockholm K., Sweden, Secretary.

Pan American Congress of the Medical Press, Buenos Aires, Argentine, July 12-16, 1953. Secretaria del Congress, 763 Uriburu, Buenos Aires, Argentine.

World Congress of *Jewish Physicians*, Jerusalem, Israel, Aug. 10-14. Secretariat: ℅ Israel *Medical* Association, Rehov Mesilat Yesharim, Beth Kovatz, Jerusalem, Israel.

World Congress of Medicine, *Montecatini*, Italy, Oct. 16-18. Secretariat: Corso Trieste, 65, *Rome*, *Italy*.

World Federation of Mental Health, Brussels, Belgium, Aug. 25-Sept. 1. Dr. J. R. Rees, 19 Manchester St., London, W.1, England, Secretary.

World Medical Association, Athens, Greece, Oct. 12-16. Dr. Louis H. Bauer, 2 East 103d St., New York 29, N. Y., Secretary General.

EXAMINATIONS AND LICENSURE

NATIONAL BOARD OF MEDICAL EXAMINERS

National Board of Medical Examiners: *Part I*, Sept. 8-10. All centers *where there* are *five* or more candidates. Exec. Sec., Dr. John P. Hubbard, 225 S. 15th St., Philadelphia 2.

EXAMINING BOARDS IN SPECIALTIES

American Board of Anesthesiology: *Oral.* Swampscott, Mass., Sept. 28-*Oct.* 1. Sec., Dr. C. B. Hickcox, 80 Seymour St., Hartford 15.

American Board of Dermatology and Syphilology: *Written.* Various Centers, September 1953. Final date for filing *applications* was May 1. All candidates are now required to take the written examination. *Oral.* Philadelphia, October 1953. Ex. Sec., Miss Janet Newkirk, 66 East 66th St., New York 21.

American Board of Internal Medicine: *Written.* Various centers and overseas, Oct. 20. Closing date for the acceptance of applications for the written examination was May 1. *Oral. San Francisco*, Sept. 3-6; *Philadelphia*, Nov. 13-14 and Washington, D. C., Nov. 17-18. Exec. Sec.-Treas., Dr. William A. Werrell, 1 West Main Street, Madison 3.

American Board of Neurological Surgery: *Written.* New Haven, Sept. 19-21. *Oral.* Chicago, May or June 1953. Final date for filing application for the oral examination is Jan. 15, 1953. Sec., Dr. W. J. German, 789 Howard Ave., New Haven 4, Conn.

American Board of Obstetrics and Gynecology: *Written.* Various Centers, Feb. 6, 1953. Final date for filing applications is Nov. 1. Sec., Dr. R. L. Faulkner, 2105 Adelbert Road, Cleveland 6.

American Board of Ophthalmology: *Written.* Various Centers, January 1953. Final date for filing applications was July 1. *Practical.* Chicago, Oct. 6-10 and New York City, June 6-10, 1953. Sec., Dr. Edwin B. Dunphy, 56 Ivie Road, Cape Cottage, Maine.

American Board of Orthopaedic Surgery: The deadline for the receipt of applications for the 1953 Part II examinations is Aug. 15. Sec., Dr. Harold A. Sofield, 122 S. Michigan Ave., Chicago.

American Board of Otolaryngology: *Written.* Chicago, Oct. 6-10. *Oral.* Chicago, Oct. 6-10 and New Orleans, April 21-25, 1953. Sec., Dr. Dean M. Lierle, University Hospital, Iowa City.

American Board of Pathology: *Written and Oral.* Pathologic Anatomy and Clinical Pathology examination. Chicago, Oct. 9-11. Sec., Dr. William B. Wartman, 303 E. Chicago Ave., Chicago 11.

American Board of Pediatrics: *Oral.* Chicago, Oct. 24-26; Boston, Dec. 5-7. Exec. Sec., Dr. John McK. Mitchell, 6 Cushman Road, Rosemont, Pa.

American Board of Plastic Surgery: *Oral and Written.* New York, Sept. 26-28. Final date for filing of case reports is Aug. 15 Sec., Dr. Bradford Cannon, 4647 Pershing Ave., St. Louis 8.

American Board of Preventive Medicine and Public Health: Cleveland, Oct. 18-20. Sec., Dr. E. L. Stebbins, 615 N. Wolfe St., Baltimore.

American Board of Proctology: *Oral and Written.* Philadelphia, Sept. 27-28. Sec., Dr. Louis A. Buie, 102-110 Second Ave., S.W., Rochester, *Minn.*

American Board of Psychiatry and Neurology: New York City, Dec. 15-16. Final date for filing applications is Sept. 15. Sec.-Treas., Dr. David A. Boyd, Jr., 102-110 Second Ave. S.W., Rochester, Minn.

American Board of Radiology: *Oral.* Dec. 1-7. Final date for filing application was June 1. *Oral.* Tampa, April 8-15. Sec., Dr. B. R. Kirklin, 102-110 Second Ave., S.W., Rochester, Minn.

American Board of Surgery: *Written.* Various Centers. October 1952. Final date for filing application was July 1. *Written.* Various Centers, March 1953. Final date for filing application is Dec. 1. Sec., *Dr.* J. Stewart Rodman, 225 S. 15th St., Philadelphia.

American Board of Urology: *Written.* Various Centers, December. *Oral.* Chicago, Feb. 7-11. Sec., Dr. Harry Culver, 30 Westwood Road, Minneapolis 16, Minn.

Board of Thoracic Surgery: *Written.* Various Centers, Sept. 5. Sec., Dr. William M. Tuttle, 1151 Taylor Ave, Detroit 2.

DEATHS

Gill, William Davis ⊕ San Antonio, Texas; born in Dallas, Texas, Nov. 24, 1895; Vanderbilt University School of Medicine, Nashville, Tenn., 1917; specialist certified by the American Board of Ophthalmology and American Board of Otolaryngology; member of the Southern Medical Association, Texas Ophthalmological and Otolaryngological Society, American Academy of Ophthalmology and Otolaryngology, American Laryngological Association, American Laryngological, Rhinological and Otological Society, and the Association for Research in Ophthalmology; fellow of the American College of Surgeons; past chairman of the city board of health; served in the medical corps of the regular army from 1919 to 1927; awarded the French Medaille d'Honneur for services rendered during World War I; member of the staff, Nix Memorial Hospital, Medical Arts Hospital, and Santa Rosa Hospital, where he died May 24, aged 56, of chronic myocarditis.

Tedder, James William ⊕ New Orleans; born in Magee, Miss., April 1, 1908; Tulane University of Louisiana School of Medicine, New Orleans, 1932; specialist certified by the American Board of Dermatology and Syphilology; member of the American Academy of Dermatology and Syphilology; formerly on the faculty of Louisiana University Medical Center; served during World War II, and was awarded the Bronze Star for heroic and meritorious service to survivors of a torpedoed, sunken ship in Langayen Gulf during the invasion of Luzon in the Philippines in January, 1945; on the staffs of Baptist and Charity hospitals and Touro Infirmary; died in Veterans Administration Hospital May 5, aged 44, of subdural hematoma and bronchopneumonia.

Garvin, Albert Henry ⊕ Buffalo; born in Joliet, Ill., in 1881; Yale University School of Medicine, New Haven, 1903; an Associate Fellow of the American Medical Association; member of the American College of Chest Physicians, American Trudeau Society, and the American Clinical and Climatological Association; president of the Buffalo and Erie County Tuberculosis Association; consultant in tuberculosis at Millard Fillmore Hospital; at one time physician in charge of the New York State Hospital for the Treatment of Incipient Pulmonary Tuberculosis at Ray Brook, N. Y., and superintendent of the Spring Hills Sanatorium in Northville, Mich.; died May 28, aged 71, of cerebral hemorrhage.

Barrera, Severo Eugene ⊕ Albany, N. Y.; Columbia University College of Physicians and Surgeons, New York, 1929; served as professor of neurology and psychiatry at Albany Medical College and psychiatrist in chief and neurologist at Albany Hospital; formerly on the faculty of his alma mater, and principal research psychiatrist at the New York State Psychiatric Institute in New York; specialist certified by the American Board of Psychiatry and Neurology; member of the American Neurological Association, American Association of Neuropathologists, and the American Psychiatric Association; died in the Ellis Hospital, Schenectady, May 25, aged 51, of chronic cardiovascular disease.

Layman, Daniel Wunderlich ⊕ Indianapolis; born in Putnamville, Ind., June 1, 1872; Columbia University College of Physicians and Surgeons, New York, 1898; clinical professor emeritus of otorhinolaryngology at Indiana University School of Medicine; specialist certified by the American Board of Otolaryngology; member of the American Laryngological, Rhinological and Otological Society; fellow of the American College of Surgeons; served on the staffs of the Robert W. Long Hospital and James Whitcomb Riley Hospital for Children; died in Methodist Hospital April 21, aged 79, of arteriosclerotic heart disease and nephrosclerosis.

Allen, Lee Gilbert ⊕ Litchfield, Ill.; Marion-Sims-Beaumont Medical College, St. Louis, 1902; past president of the Montgomery County Medical Society; on the staff of St. Francis Hospital; died June 10, aged 75, of coronary thrombosis.

Barrett, Edward William ⊕ Medford, Mass.; Jefferson Medical College of Philadelphia, 1902; school physician; at one time city physician; on the staff of Lawrence Memorial Hospital where he died May 17, aged 82, of arteriosclerotic heart disease and bronchopneumonia.

Barthmaier, Othmar Frank, Philadelphia; Hahnemann Medical College and Hospital of Philadelphia, 1906; formerly on the faculty of his alma mater; served during World War I; affiliated with Hahnemann Hospital and St. Luke's and Children's Hospital, where he died May 9, aged 67, of arteriosclerosis.

Bates, George ⊕ Kingston, Mich.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1890; died in Cass City May 13, aged 91.

Baum, E. Eldon ⊕ Oklahoma City, Okla.; University of Oklahoma School of Medicine, Oklahoma City, 1928; served on the staffs of the Mercy and Wesley hospitals; died May 16, aged 56, of cerebral and kidney infarcts and bilateral tumor of adrenals.

Beeler, Bruce Henry, Evansville, Ind.; University of Louisville (Ky.) School of Medicine, 1915; served during World War I; affiliated with Deaconess Hospital and St. Mary's Hospital, where he died May 23, aged 64, of cancer and cirrhosis of the liver.

Bellak, Asher-Seelig, Philadelphia; Medico-Chirurgical College of Philadelphia, 1908; formerly affiliated with St. Agnes Hospital and the Mount Sinai Hospital, where he died May 19, aged 66, of tuberculosis.

Berger, Max Milton ⊕ Oakland, Calif.; Rush Medical College, Chicago, 1935; served during World War II; assistant chief of urology, Veterans Administration Hospital; died April 30, aged 44, of coronary thrombosis.

Carleton, Elmer Howard ⊕ Hanover, N. H.; Dartmouth Medical School, Hanover, N. H., 1897; formerly on the faculty of his alma mater; member of the New England Otolaryngological Society; served on the staff of the Dartmouth Eye Institute; in 1939 received the honorary degree of doctor of science from Bowdoin College, Brunswick, Me.; past president of the Dartmouth Medical School Alumni Association; died June 1, aged 83.

Clarke, William Thomas ⊕ Los Angeles; University of Toronto Faculty of Medicine, Toronto, Canada, 1899; L.R.C.P., L.R.C.S., Edinburgh, 1904, and L.F.P.S., Glasgow, 1904; served during World War I; an Associate Fellow of the American Medical Association; died in California Lutheran Hospital May 11, aged 75, of cerebral thrombosis.

Collins, Elmer E. G. ⊕ Chicago; Jenner Medical College, Chicago, 1910; formerly chief physician for Swift & Company, packers; died May 16, aged 68, of coronary thrombosis.

Colthorp, Ralph Weldon, Dallas, Texas; University of Texas School of Medicine, Galveston, 1942; specialist certified by the American Board of Psychiatry and Neurology; lieutenant in the U. S. Navy from 1942 to 1947; formerly director of the Boys Industrial School in Topeka, Kan.; consulting psychiatrist for the city school system; director of the Child Guidance Clinic; died May 25, aged 35, of septicemia.

Connell, John ⊕ Jersey City, N. J.; New York Homeopathic Medical College and Hospital in New York, 1889; died in the Medical Center April 1, aged 87, of bronchopneumonia.

Cooper, Jacques ⊕ Chicago; Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin, Prussia, Germany, 1917; affiliated with Holy Cross Hospital; died May 9, aged 62, of heart disease.

Cress, Peter Joseph ⊕ Marysville, Calif.; Wisconsin College of Physicians and Surgeons, Milwaukee, 1908; died in Garmisch, Germany, May 30, aged 70, of coronary occlusion.

Cummings, Joseph Leo ⊕ Poughkeepsie, N. Y.; Columbia University College of Physicians and Surgeons, New York, 1934; specialist certified by the American Board of Psychiatry and Neurology; served during World War II; formerly member of

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⊕ Indicates Member of the American Medical Association.

the city board of health; neuropsychiatrist consultant, Veterans Administration Hospital in Castle Point; affiliated with St. Francis and Vassar Brothers hospitals; died May 28, aged 45, of metastatic carcinoma of the liver.

Duckworth, William Henry ⊕ St. Clair, Mo.; National University of Arts and Sciences Medical Department, St. Louis, 1918; died in the Jewish Hospital May 3, aged 58, of heart disease.

Eckhardt, Peter, Rock Island, Ill.; Rush Medical College, Chicago, 1896; died in St. Anthony's Hospital May 9, aged 92.

Epstein, William George ⊕ Chicago; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1910; formerly on the faculty of Loyola University School of Medicine; member of the senior surgical staff, St. Bernard's Hospital, where he died June 16, aged 65, of myocardial infarction.

Erskine, James Pendleton, Lake Worth, Fla.; Columbia University College of Physicians and Surgeons, New York, 1903; formerly practiced in New York, where he was affiliated with New York Hospital, New York Eye and Ear Infirmary, and the French Hospital; died in West Palm Beach, May 25, aged 78, of cancer.

Evans, Samuel Davidson ⊕ Pittsburgh; University of Pittsburgh School of Medicine, 1925; specialist certified by the American Board of Ophthalmology; member of the American Academy of Ophthalmology and Otolaryngology and the Association for Research in Ophthalmology; instructor in ophthalmology at his alma mater; affiliated with Eye and Ear Hospital; died in St. Joseph's Hospital June 1, aged 51, of pneumonia and ulcerative colitis.

Harris, Henry, New York; Fordham University School of Medicine, New York, 1917; died recently, aged 59, of coronary occlusion.

Hasking, Arthur Perry ⊕ Jersey City, N. J.; Columbia University College of Physicians and Surgeons, New York, 1903; past president and secretary of the Hudson County Medical Society; member of the American Psychiatric Association; for many years county physician; affiliated with Medical Center of Jersey City; died in Monmouth Memorial Hospital in Long Branch, May 29, aged 72, of cerebral hemorrhage and hypertension.

Haworth, Dillon Benjamin, Kansas City, Mo.; Kansas City (Mo.) Medical College, 1901; served during World War I; died May 22, aged 80.

iller, Frank Brooks ⊕ Pinckneyville, Ill.; St. Louis College of hysicians and Surgeons, 1910; owner of a hospital bearing his name; died in Veterans Administration Hospital in Marion, May 6, aged 71, of coronary thrombosis.

Hirst, Virginius Bitzer ⊕ New York; University of Virginia Department of Medicine, Charlottesville, 1918; associate clinical professor of otorhinolaryngology at Post Graduate Medical School of the New York University-Bellevue Medical Center; served during World War I; fellow of the New York Academy of Medicine and American College of Surgeons; affiliated with New York Eye and Ear Infirmary; died May 9, aged 61.

Hoenes, Andrew John ⊕ Salt Lake City, Utah; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1889; an Associate Fellow of the American Medical Association; died May 29, aged 91, of arteriosclerotic heart disease.

Hoenig, Edward J. ⊕ New York; Cornell University Medical College, New York, 1910; specialist certified by the American Board of Obstetrics and Gynecology; fellow of the American College of Surgeons; consulting gynecologist, and for two terms president of the medical board, Jewish Memorial Hospital; died May 3, aged 70, of coronary thrombosis.

Holt, Charles Scott ⊕ Fort Smith, Ark.; St. Louis University School of Medicine, 1906; fellow of the American College of Surgeons; affiliated with St. Edward's Hospital and Sparks Memorial Hospital, where he died June 7, aged 72, of carcinoma of the prostate.

Kidd, James William, Fort Wayne, Ind.; Baltimore University School of Medicine, 1897; died in the Methodist Hospital May 29, aged 78, of arteriosclerotic heart disease.

Kissel, Julius Peter ⊕ Centralia, Ill.; St. Louis University School of Medicine, 1910; served during World War I; died June 3, aged 63, of cerebral hemorrhage.

Kopelowitz, Jonas Clarence ⊕ Los Angeles; Washington University School of Medicine, St. Louis, 1913; specialist certified by the American Board of Internal Medicine; served during World War I; affiliated with Cedars of Lebanon Hospital; died May 4, aged 60, of hypertensive heart disease.

Kosciolek, Stephen Frank ⊕ Lowell, Mass.; Boston University School of Medicine, 1934; drowned in Northwood Lake, N. H., April 30, aged 45.

Kroll, Louis Joseph ⊕ Baltimore; University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, 1936; died in Johns Hopkins Hospital, recently, aged 39, of glomerulonephritis.

Laessle, Henry Adolph ⊕ Philadelphia; Jefferson Medical College of Philadelphia, 1902; also a graduate in pharmacy; specialist certified by the American Board of Otolaryngology; assistant professor of otolaryngology at Graduate School of Medicine, University of Pennsylvania; member of the Philadelphia Laryngological Society; died May 13, aged 80, of coronary thrombosis.

Lamb, Gerald E., Farwell, Mich.; Detroit College of Medicine, 1899; for many years county coroner; president of the Farwell State Savings Bank; died in the Henry Ford Hospital, Detroit, May 28, aged 76.

Lange, Sidney, Cincinnati; Medical College of Ohio, Cincinnati, 1903; professor emeritus of radiology, University of Cincinnati College of Medicine; specialist certified by the American Board of Radiology; member and past president of the American Roentgen Ray Society; affiliated with Good Samaritan, St. Mary's, and Cincinnati General hospitals; died in Jewish Hospital May 9, aged 72, of cerebral hemorrhage following a fall.

Levine, Emanuel ⊕ Elizabeth, N. J.; University of Vermont College of Medicine, Burlington, 1938; served during World War II; died May 8, aged 37, of injuries received in an automobile accident.

Lewis, Harry Samuel ⊕ Washington, D. C.; George Washington University School of Medicine, Washington, D. C., 1910; fellow of the American College of Surgeons; died May 14, aged 72, of coronary thrombosis.

Lichtenwalner, Howard Adams ⊕ Bloomsburg, Pa.; Hahnemann Medical College and Hospital of Philadelphia, 1921; served during World War I; on the staff of the Bloomsburg Hospital, where he died May 12, aged 57, of cerebral vascular encephalopathy.

Light, Ralph Allison, Chanute, Kan.; University Medical College of Kansas City, Mo., 1898; veteran of the Spanish-American War; formerly secretary of the state board of registration and examination; for many years county coroner; retired surgeon of the Santa Fe Railroad; formerly member of the city council and school board; died in Neosho Memorial Hospital, June 3, aged 80, of hypostatic pneumonia.

Loefflad, Paul Aloysius ⊕ Upper Darby, Pa.; Georgetown University School of Medicine, Washington, D. C., 1925; physician for many years of Villanova (Pa.) College; on the staff of the Fitzgerald Mercy Hospital in Darby, and Misericordia Hospital in Philadelphia, where he died May 18, aged 51, of bleeding duodenal ulcer.

Long, Homer Garrison, Copley, Ohio; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1897; died May 20, aged 80.

Lorenzo, Frank Alexander ⊕ Punxsutawney, Pa.; Columbia University College of Physicians and Surgeons, New York, 1904; fellow of the American College of Surgeons; consulting surgeon, Elk County General Hospital, Ridgway, and the Adrian Hospital; died May 30, aged 72.

Love, Travis Drennon, Mendenhall, Miss.; Birmingham Medical College, 1915; died May 25, aged 67.

Marston, Charles Benjamin, San Rafael, Calif.; Hahnemann Medical College of the Pacific, San Francisco, 1908; past president of the board of education; on the staff of the San Rafael General Hospital, where he died May 19, aged 79, of cerebral accident and Parkinson's disease.

Martin, George Henry, Chicago; Northwestern University Medical School, Chicago, 1905; died June 27, aged 69.

Martinek, Jaroslav ⊕ Phoenix, Ariz.; University of Illinois College of Medicine, Chicago, 1946; died in Los Angeles May 28, aged 34, of bilateral pneumothorax and bronchial asthma.

Metzger, Karl Fenton, Belmar, N. J.; Hahnemann Medical College and Hospital of Philadelphia, 1933; died in Fitkin Memorial Hospital, Neptune, April 28, aged 45.

Mitchell, Allie Long, East Aurora, N. Y.; University of Buffalo School of Medicine, 1898; died in a Buffalo nursing home May 30, aged 97, of coronary thrombosis.

Nachtigall, Hans W., Chicago; Chicago College of Medicine and Surgery, 1913; for many years on the staff of St. Joseph Hospital; died May 24, aged 65, of coronary thrombosis.

O'Connor, Donald Charles ⊕ Buffalo; University of Buffalo School of Medicine, 1922; superintendent of the Edward J. Meyer Memorial Hospital, where he died May 25, aged 56, of hypertensive cardiovascular disease.

O'Donnell, John Joseph ⊕ Erie, Pa.; College of Physicians and Surgeons, Baltimore, 1904; specialist certified by the American Board of Psychiatry and Neurology; member of the American Psychiatric Association; served during World War I; affiliated with Hamot and St. Vincent's hospitals; died May 29, aged 70, of carcinoma.

Owen, William Arthur, Columbus, Ohio; Ohio Medical University, Columbus, 1897; died April 15, aged 78, of coronary thrombosis.

Pettit, James Kenneth ⊕ Atlanta, Ga.; Atlanta Medical College, 1914; served in France and Scotland during World War I; affiliated with the Veterans Administration; formerly on the staffs of the Georgia State Hospital for Insane in Milledgeville, Letchworth Village in Thiells, and Mansfield State Training School in Mansfield Depot, Conn.; died in Veterans Administration Hospital, Augusta, May 22, aged 63, of carcinomatosis.

Pinson, Fred Edward, Vicksburg, Miss.; Meharry Medical College, Nashville, Tenn., 1915; died May 26, aged 63, of injuries received in an automobile accident.

Plamondon, James Dillon, Lake Grove, Ore.; Willamette University Medical Department, Salem, 1898; served during World War I; formerly assistant superintendent of the Eastern Oregon State Hospital in Pendleton and Oregon State Hospital in Salem; served on the medical examining board of the Veterans Administration in Portland; died in Veterans Administration Hospital, Vancouver, Wash., May 15, aged 76, of a cerebrovascular accident.

Porter, MacGuyer ⊕ Elnora, Ind.; Eclectic Medical Institute, Cincinnati, 1897; died May 12, aged 85, of angina pectoris.

Posner, Benjamin Herman, Boston; Middlesex College of Medicine and Surgery, Cambridge, Mass., 1935; served during World War II; died April 5, aged 43, of coronary disease.

Praglin, Daniel Frank, Monterey Park, Calif.; Tulane University of Louisiana School of Medicine, New Orleans, 1929; served during World War II; died May 15, aged 49, of coronary thrombosis.

Pugh, Thomas Bryan, Napoleonville, La.; Charity Hospital Medical College, New Orleans, 1875; for many years coroner and sheriff of Assumption Parish; died May 4, aged 99.

Reilly, Maurice John ⊕ Arthur, Ill.; Chicago Medical School, 1931; served during World War II; died May 21, aged 47, of a heart attack.

Renaud, Joseph Edward ⊕ Syracuse, N. Y.; Syracuse University College of Medicine, 1927; medical director of the Easy Washing Machine Corporation; member of the Industrial Medical Association; affiliated with St. Joseph's Hospital, where he died May 9, aged 48, of cerebral hemorrhage.

Richmond, Oscar Lee, Corbin, Ky.; Vanderbilt University School of Medicine, Nashville, 1917; died May 27, aged 58.

Roberts, Daniel Stillwell, West Point, Ky.; Kentucky School of Medicine, Louisville, 1905; local surgeon for the Illinois Central Railroad; died May 18, aged 80, of diabetes mellitus.

Roberts, William James, Logansport, Ind.; Georgia College of Eclectic Medicine and Surgery, Atlanta, 1895; died in St. Joseph Hospital March 14, aged 81, of coronary occlusion.

Scott, Walter Sidney ⊕ Denver; Columbian University Medical Department, Washington, D. C., 1896; died March 6, aged 78.

Seitz, George William, Buffalo; University of Buffalo School of Medicine, 1902; died May 6, aged 77.

Shay, Francis Leo ⊕ New Haven, Conn.; Tufts College Medical School, Boston, 1925; member of the American Academy of General Practice; affiliated with Grace Unit, Grace-New Haven Community Hospital and Hospital of St. Raphael, where he died April 24, aged 53, of coronary thrombosis and virus pneumonia.

Sheridan, Joseph Morton, Baton Rouge, La.; Fordham University School of Medicine, New York, 1910; served during World War I; formerly affiliated with the New York City Farm Colony, West New Brighton, Staten Island, N. Y., New York City Cancer Institute Hospital, Welfare Island, New York, Morrisania and Gouverneur hospitals in New York; died May 4, aged 64.

Starzynski, Theophilus A., Pittsburgh; Western Pennsylvania Medical College, Pittsburgh, 1904; served during World War I; died June 9, aged 73.

Stevenson, Benjamin J. ⊕ Sparta, Ill.; Homeopathic Medical College of Missouri, St. Louis, 1898; died March 8, aged 78, of carcinoma of the prostate.

Stuart, Clyde George ⊕ Los Angeles; College of Medical Evangelists, Loma Linda and Los Angeles, 1938; certified by the National Board of Medical Examiners; public health physician with county health department for many years; affiliated with White Memorial Hospital; died May 8, aged 49, of hypertensive heart disease.

Summers, Charles Raymond ⊕ Oak Park, Ill.; University of Illinois College of Medicine, Chicago, 1929; affiliated with West Suburban Hospital; died in Veterans Administration Hospital, Hines, June 11, aged 55, of chronic glomerulonephritis with uremia.

Tillotson, Harry Trudeau, Chicago; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1886; died June 15, aged 89, of coronary insufficiency.

Tittle, Lloyd Clay ⊕ Dallas, Texas; Baylor University College of Medicine, Dallas, 1918; died May 6, aged 61, of surgical shock with acute hepatic insufficiency.

Vander Bogart, Harry Eugene ⊕ Goshen, Ind.; the Hahnemann Medical College and Hospital, Chicago, 1914; served during World War I; member of the board of directors of the Goshen Hospital; died May 23, aged 68, of carcinoma of the ascending colon.

Voorhis, Anna E. Harvey ⊕ Mount Vernon, N. Y.; Woman's Medical College of the New York Infirmary for Women and Children, New York, 1893; formerly practiced in Yonkers, where she was affiliated with St. John's Hospital; died June 9, aged 85, of hypertensive arteriosclerotic disease.

West, Edward W., North Little Rock, Ark.; University of West Tennessee College of Medicine and Surgery, Memphis, 1904; died in the Provident Hospital, Chicago, May 25, aged 76, of cerebral hemorrhage and essential hypertension.

Wiener, Jacob Jesse ⊕ New York; Columbia University College of Physicians and Surgeons, New York, 1916; member of the American Academy of Pediatrics; affiliated with Hospital for Joint Diseases; died in Miami Beach, Fla., May 26, aged 58, of carcinoma of the colon.

Winne, William Ray ⊕ North Syracuse, N. Y.; Syracuse University College of Medicine, 1917; served during World War I; formerly county coroner; affiliated with Crouse Irving Hospital in Syracuse; died May 28, aged 61, of coronary disease.

GOVERNMENT SERVICES

ARMY

Lieut. Hopkins Awarded Soldier's Medal.—First Lieut. William C. Hopkins of Chicago, medical officer with the 25th Infantry Division in Korea, has been awarded the Soldier's Medal for "unselfish heroism and dauntless courage." The citation reads in part as follows: "On March 7, 1952, a helicopter crashed while attempting to evacuate two wounded soldiers from a foggy, windswept hill position near Sohuiri, Korea. With utter disregard for his personal safety, Lieutenant Hopkins ran through a known mine field to the crashed helicopter and aided in righting the flaming aircraft, rescuing one of the wounded soldiers and attempting to free the other man still trapped in the wreckage. He persisted in his efforts until the heat became unbearable and he was forced to move away from the craft.

"The helicopter pilot dashed into the flaming wreckage and tried again to rescue the trapped man. Realizing that the gas tank would soon explode, Lieutenant Hopkins ran into the inferno and forced the pilot away from the craft, saving him from permanent disfigurement and possible death. Lieutenant Hopkins' unselfish heroism and dauntless courage are in keeping with the highest traditions of the United States Army."

Lieutenant Hopkins is the son of Percy E. Hopkins, M.D., of Chicago, a former president of the Illinois State Medical Society.

DEPARTMENT OF DEFENSE

Korean Society News.—Col. Walter M. Bartlett, Decatur, Ga., Medical Consultant at Eighth Army Headquarters, has been elected president of the "38th Parallel Medical Society of Korea," and Capt. Inger Schulstad, a woman doctor with the Norwegian Surgical Hospital, secretary. They succeed Lieut. Col. Charles E. Mason, Alhambra, Calif., I Corps surgeon, the outgoing president, and Capt. Ruth Dickson, Vinton, Va., chief nurse of the 8,055th Mobile Army Surgical Hospital, former secretary.

The new vice-presidents are Col. Erling F. Hjort, commanding officer of the Norwegian Surgical Hospital; Col. Morgan Smith, Canada, newly-appointed surgeon of the First Commonwealth Division; Capt. Louis Kirkpatrick, USN, Reagan, Texas, First Marine Division Surgeon; Lieut. Col. Claude B. Knight, Wewoka, Okla., 45th Division Surgeon; Lieut. Col. Sin Jai Kuh, commanding officer of the 5th Republic of Korea Mobile Army Surgical Hospital; Lieut. Col. A. G. Rangaraj, commanding officer of the 60th Indian Field Ambulance Unit, and Lieut. Col. H. Haskell Zipperman, Mount Holly, N. J., commanding officer of the 8,063d Mobile Army Surgical Hospital. This unique society, which is composed of medical personnel in I Corps and adjacent IX Corps areas, meets twice a month on the 38th parallel. Speakers at the most recent meeting were Colonel Hjort of the Norwegian Hospital and Capt. Edward Jahnke, Pittsburgh, Pa., from Walter Reed Army Hospital, Washington, D. C.

VETERANS ADMINISTRATION

New GI Bill for Veterans.—The President has signed a new GI Bill for veterans who served in the armed forces any place in the world since the start of the Korean conflict on June 27, 1950. The new law provides five benefits, all designed to assist veterans in their return to civilian life. The benefits include education and training; guaranteed or insured loans for homes, farms, and businesses; unemployment compensation; mustering-out pay; and job finding help.

The education and training provisions allow a veteran one and one-half days of training for each day in service after the outbreak of the Korean fighting, regardless of where the service was performed, up to a maximum of 36 months; however, veterans who have previously trained under earlier veterans' training laws, the World War II GI Bill or Public Laws 16 or 894 for the disabled, may get up to 48 months, minus whatever time they have already spent in training under those earlier programs. A veteran may train in school or college, on the job or on the farm, as long as the school or training establishment has been approved by an appropriate state approving agency and meets other qualifications of the law.

Veterans in GI Bill training will receive an education and training allowance each month from the government to meet part of the expenses of their training and living costs. Tuition, fees, books, supplies, and equipment will not be paid by the government; instead, they will be paid out of the monthly allowance. Rates for veterans in full-time training in schools and colleges are $110 a month if they have no dependents, $135 if they have one dependent, and $160 if they have more than one dependent. Those in training less than full time will receive lower monthly rates. Top monthly amounts for on-the-job trainees are $70 without dependents, $85 with one dependent, and $105 with more than one dependent. The maximal allowances for institutional on-farm trainees are $95, $110, and $130, respectively. The law requires that on-job and on-farm rates be reduced, at four month intervals, as the training progresses and the veteran's own earnings increase. Veterans were reminded that the last three benefits of the new GI Bill, unemployment compensation, mustering-out pay, and job-hunting aid, are not administered by the Veterans Administration. Veterans should not write to the VA about them if they want further information or have questions to be answered; instead, they should correspond directly with the agency concerned. Education and training, for post-Korea veterans, must be started by Aug. 20, 1954, or two years after release from active duty, whichever comes later. No training may be given beyond either seven years after discharge or seven years after the end of the current emergency, whichever is later.

MISCELLANEOUS

Science Foundation to Compile Research Information.—Federal agencies supporting scientific research and development programs have been asked to supply the National Science Foundation with statistical and fiscal information about their programs for the fiscal years 1951 and 1952. The foundation is compiling such information at the request of the Bureau of the Budget, which has gathered similar information in previous years. The final report, which it is hoped will be available before Jan. 1, 1953, will be of value also to Congress and to the agencies, themselves, in the administration and planning of their programs. The information will be of value to the foundation, also, in fulfilling its statutory responsibilities with respect to scientific research and its studies concerning the effect of present support and policies on research and education in the institutions of the country.

The information will be compiled under various functional classifications, including basic research, applied research, development, and increase of research and development plant, and subject classifications including biologic, medical, agricultural, physical, mathematical, engineering, and social sciences. Typical examples of nonprofit institutions for which information will be gathered are colleges and universities, research institutes, research foundations, experiment stations, hospitals, libraries, museums, observatories, and corporations organized specifically to do scientific research and development for the federal government on a nonprofit basis. Information on direct research activities of agencies of the federal government will be excluded, but the report will include information on federal support to agencies of state, local, or foreign governments.

FOREIGN LETTERS

BRAZIL

Colon Resection with Immediate Open Anastomosis.—Dr. E. da Silva Bastos, professor of surgery at the São Paulo State University, recently reported a series of 72 patients on whom he performed resection of the colon followed by immediate open anastomosis. Except for four of the cases, two with tuberculomas of the cecum and two with inflammatory strictures from diverticulitis, all were due to cancer of the colon, the cecum, and the rectum. In the 72 cases, 12 with Mikulicz resection, 23 with closed anastomoses, and 37 with open anastomoses, there were four deaths (5.53%), one from generalized peritonitis after an ileocolic closed anastomosis, one from hypoproteinemia as a consequence of a hematoma, one from uremia, and one from cerebral thrombosis. In the cases in which closed anastomosis was performed there was one death (4.35%), and in the cases with open anastomosis there was also one death (2.70%). There were 5 complications in 12 cases of resection by the Mikulicz technique, the most frequent of which were abscesses of the intestinal wall and eventrations. In only one case of eventration was there no accompanying subcutaneous abscess. In the group of closed anastomoses, the most frequent complication was stricture, which occurred in five cases and probably also in two cases of dehiscence of the suture with abscess or fecal fistula. Closed anastomosis, used in two cases of resection of the rectum, caused stricture with fecal fistula in both. In one of the cases, the fistula was very wide, remaining so until the death of the patient 18 months after the operation. Open anastomosis, used in 16 cases without colostomy, caused fewer complications. There were four cases of fecal fistula, all of them in previous resections of the rectum. All fistulas were of little importance, disappearing spontaneously within a short time. Only one fistula occurred in a patient without colostomy; in the other three patients a minimal colostomy had been done. The author concludes that the open anastomosis is superior to the closed anastomosis for the following reasons. 1. It is a simpler technique, not needing special instruments. 2. The suture is easier; the stitches are made directly through the walls of the intestine under direct vision. 3. The suture is tighter and the approximation of the intestinal layers more perfect.

Hemagglutination with Human Red Blood Cells and BCG Tuberculin.—Dr. Laerte de Andrade, of the Oswaldo Cruz Institute, and Dr. Candido Silva, of the division of leprosy, National Department of Health, have just reported the results of their extensive investigations on the hemagglutination test of Middlebrook and Dubos and its use in connection with tuberculosis and leprosy. In the introduction of their report the authors state that, owing to the difficulty of obtaining in Brazil specific antigens prepared with the polysaccharide fraction of the tubercle bacillus or the Lederle tuberculin (O.T.) recommended by American investigators, they tried to use several tuberculin preparations made in the country. The only one that gave good results was prepared by cultivation of BCG on a Sauton medium. The authors performed 271 tests on serums from blood donors as well as from tuberculous and leprous patients. Human O Rh-negative red blood cells sensitized by BCG tuberculin were used for the test. A qualitative test very useful for routine work was performed in capillary tubes, as an adaptation of Chown's test for Rh antibodies.

The 271 serums examined included 68 from tuberculous patients, 116 from leprous patients, and 87 from donors from the blood bank of the Rio de Janeiro Hospital dos Servidores. There were 16 negative serums (23.5%) among the tuberculous patients, 22 (18.9%) among the leprous patients, and 82 (94.3%) among healthy blood donors. Of the 52 tuberculous patients with positive results, 19 showed results with titers of 1:32 or less and 33 showed results with titers from 1:64 to 1:1024. Of the 94 leprous patients with positive results, 40 showed results with titers 1:32 or less and 54 showed results with titers 1:64 to 1:1024. Of the 87 blood donors, only 5 showed positive results (3 with a titer of 1:4, one with a titer of 1:8, and one with a titer of 1:16). The 116 leprous patients included 11 of the undetermined form, 15 of the tuberculoid form, and 86 of the lepromatous form. Of the latter, 43 patients had active leprosy and 43 were in clinical regression, without bacilli in the nasal mucus and in the skin, and were scheduled to be transferred to the outpatient department. Among the patients with the lepromatous form in clinical regression, 14 showed negative results of the hemagglutination tests, and many of the others had very low positive agglutination titers. Three of them, however, had very high titers (1:256 and 1:1024). The authors explain these results as being caused by visceral leprosy, which probably did not show a regression similar to that of the tegumentary forms. In this connection, the authors' conclusion is that the Middlebrook-Dubos test is useful in supplying some indispensable information as to the course of the disease in patients with the lepromatous form and especially of candidates for transferral to the outpatient department.

Milestone in Campaign Against Yellow Fever.—A milestone has been reached in 1951 in the campaign against yellow fever in Brazil: the complete eradication of Aedes aegypti, the urban vector of the disease. A recheck is being made, however, in some parts of the eastern and northeastern regions, where the environmental conditions are very favorable to that species of mosquito, although the work done during the first five months of the current year in those areas has supposedly eradicated the mosquito. There still must be careful and continuous inspections in order to prevent the reimportation of the mosquito through the borders, and air and maritime ports, since A. aegypti still exists in 23 countries and possessions and colonies in the Americas.

The sylvan form of yellow fever is transmitted through other species of mosquitos. The last three cases of urban yellow fever were reported in 1937, and after this only the sylvan form of the disease was recorded in Brazil. During the three 5 year periods 1937-1941, 1942-1946, and 1947-1951 there were, respectively, 807, 141, and 45 cases of the disease. The campaign against the sylvan form of yellow fever includes mainly vaccination, viscerotomy, and laboratory examinations. About 900,000 persons were immunized against yellow fever in 1951, bringing the total number of vaccinations, since 1937, to more than 7 million. Immunization is mainly used to protect persons who have to work or live in forests. Viscerotomy is the best resource for the discovery of the silent foci of the disease in areas in which it frequently goes unrecognized even by trained physicians. There were, in 1951, a total of 1,324 localities with viscerotomy service, which sent 8,185 specimens of liver to be examined in the laboratory.

DENMARK

Civil Defense and Biologic Warfare.—In 1951 the governing body of civil defense requested the Danish public health authorities to compose a memorandum on civil defense problems connected with biologic warfare. In December of that year, the public health authorities appointed a committee of bacteriological, epidemiological, clinical, and administrative experts to pre-

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The items in these letters are contributed by regular correspondents in the various foreign countries.

pare such a memorandum. The completed memorandum is concerned with successfully combating the results of biologic warfare. The sources of information that the committee used are official publications and are available to all persons interested. The memorandum refers, of course, particularly to conditions that exist or may develop in Denmark. The authors conclude that biologic warfare is possible in theory and feasible in practice, although its effects cannot be calculated with certainty. They also conclude that though biologic weapons can be directed against military objectives, their employment undoubtedly greatly endangers civilians. Panic among civilians may be the only effect sought by an aggressor anxious to demonstrate his strength and to threaten the government of an enemy country. The possibilities for sabotage with biologic weapons are almost unlimited, and the fifth columnist can do his work with greater accuracy and with fewer weapons than a regular combatant. Many germs can be cultivated in great quantities under primitive conditions in secrecy in the country to be attacked. In Denmark an attack with infected insects is not likely to be successful owing to its climate and its indigenous insects, but yellow fever and rickettsia may be conveyed not only by insects but also by inhalation. The weight and volume of biologic weapons are so small that it may be impossible to prevent their entry into a country by smuggling. For this and various other reasons, attention must be focused on preventing the effects once such warfare has started. In Denmark there are not enough vaccines for vaccinating all persons in a community against all the diseases spread in biologic warfare, but it should be possible to produce a botulinus vaccine suitable for limited and much exposed sections of the community. The prophylactic employment of antibiotics presupposes enormous quantities of them, and they would have to be limited to sections of the community greatly exposed to infection. Penicillin and the sulfonamides are now produced in Denmark in sufficient quantities for an emergency of limited duration, but the cost of well-known antibiotics produced abroad is prohibitive.

Protests Against Wholesale Legal Abortions.—The Hippocratic attitude towards the induction of abortion has lately been defended by Prof. Erik Rydberg, in whose hospital abortions are induced only on strict and clear medical indications. Writing in the organ of the Danish Medical Association, *Ugeskrift for Laeger,* for May 22, he reports that the number of legally induced abortions has risen considerably since the adoption of the abortion law in 1937 and its practical application after 1939. Between 1940 and 1950 the number of legal abortions has risen from 522 to 3,969. It is calculated that in Copenhagen in 1951 the legal abortions were equivalent to 12% of all the confinements in this year. The annual reports of gynecologic hospitals suggest that most legal abortions are indicated by psychiatric considerations. For example, at the gynecologic department of the Frederiksberg Hospital in 1950 the 319 legal abortions included 267 induced on psychiatric grounds.

Declining Tuberculosis Rate in Childhood.—Several articles in the medical press have lately asked the reason for the tuberculosis morbidity declining more rapidly at the school age than at the preschool and postschool ages. They also wondered whether BCG vaccination offers any answer to this question. Dr. E. Groth-Petersen, who is in charge of the tuberculosis dispensary work of the County of Praestö, has studied the characteristics of tuberculosis in his district since a tuberculosis dispensary was started there in 1943. Between then and 1951 there was no great decline in the tuberculosis morbidity at the preschool age, at which BCG vaccination is comparatively rare. On the other hand, at the school age, from 7 to 14 years, the tuberculosis morbidity showed an abrupt fall, which has been maintained since 1947. Between the ages of 15 and 19 there was a more gradual fall. Practically every schoolchild found to be tuberculin-negative was vaccinated with BCG after 1943. After discussing the possibilities that the decline of the tuberculosis morbidity might be due to less exposure to infection or to changes in diagnostic standards or to other factors, Dr. Groth-Petersen concludes that the abrupt fall in the tuberculosis morbidity between the ages of 7 and 14 years can largely be explained as the outcome of wholesale BCG vaccination in school.

In another study, Dr. Jörgen Kringelbach compared the tuberculosis clientele of a children's hospital in the two years 1940 and 1950. This hospital, the only one of its kind in Denmark, dealt with tuberculous children from Copenhagen. Its 47 beds were, for many years, always in use for tuberculous patients, but of late most of them have been allocated to nontuberculous patients for want of enough tuberculous children. In the two years under review there was little difference in the number of preschool children, whereas there was a marked drop in the number of children of the school age, 7-14 years. The relative scarcity of the school age patients in 1950 is held by Kringelbach to be largely due to the extensive BCG vaccination of Copenhagen's tuberculin-negative school children in 1948 and 1949. There was a marked difference between the two years 1940 and 1950 with regard to the duration of hospitalization, which was 100 days on the average in 1940 as compared with only 62 days in 1950. The character of the disease was on the whole less serious in 1950 than in 1940 to judge by the radiological findings, the temperature, and the erythrocyte sedimentation rate. Thus in the brief span of 10 years a profound change had come over the clientele of this hospital.

PARIS

Iodochlorhydroxyquin in Experimental Tuberculosis.—P. E. David and F. Tisson reported to the National Academy of Medicine the first in vivo trials against tuberculosis of an iodochlorhydroxyquin suspension. Two groups, of 10 animals each, were inoculated with a human virulent strain of tuberculosis. At the end of three weeks one group was treated by daily subcutaneous administration of 3 cg. per kilogram of body weight of the drug while the other served as untreated controls. Eight weeks after the inoculation one guinea pig was killed in each group, the others being killed at intervals within the following 15 days. The results of the autopsy of these animals revealed that of the 10 animals who were given iodochlorhydroxyquin, 7 showed only lymph node involvement of the ganglions (stage 1), 1 an involvement of lymph nodes and spleen (stage 2), and 2 an involvement of lymph nodes, spleen, and liver (stage 3); none of these animals reached stage 4 (lymph nodes, spleen, liver, and lungs involved). On the other hand, of the 10 untreated control animals, 5 were in the disease stage 4, 1 in stage 3, and 4 in stage 2. None of the control animals remained in stage 1. Swabs of inguinal abscesses showed the presence of a larger number of bacilli in all control animals than in the treated group.

Microbial Substitution and Selection in Chronic Infections.—The failure of some therapy with antibiotics has been explained by the resistance acquired by bacteria in the course of treatment. It has also been found, in other cases, that the organism sets off a mechanism of the adaptation enzymes; for instance, penicillinase undergoes a morphological change, taking either the dwarf or the L form, according to Tulsane. J. Brissou of the French navy, on the basis of clinical and laboratory observations, noted that after a patient is treated with an antibiotic a relapse may occur after a transient improvement and the disease may become chronic. A further examination reveals new bacterial flora. There is a substitution of bacteria, and it is noted that, in this selection, resistant species are present. The author noted the substitution in urine of proteins or Klebsiella by Escherichia coli, of Proteus by enterococci and, in urethritis the substitution of streptococci by gonococci. In an unpublished work done in collaboration with Ardisson, the author show that, after massive doses of an antibiotic, the rich intestinal flor decreases and there is an upset of the bacterial equilibrium accompanied by new disorders, notwithstanding the eliminatio of Salmonella or Shigella. The author suggests a new idea the cause of chronic diseases by selection and substitution bacteria.

CORRESPONDENCE

PATIENTS WITH POLIOMYELITIS

To the Editor:—I have read with interest the article entitled "Home Care of Patients with Acute Poliomyelitis" by Philip M. Stimson, M.D., published in THE JOURNAL (June 21, 1952, page 719). There is no question but that the emotional attitudes of the family and patient are important. Hospitalization in many instances is unnecessary, and long periods of transportation are detrimental; however, I do feel it necessary to comment on some other considerations.

In the recognizable case without paralysis, it is well known that the severity of the early signs and symptoms bears no direct relationship to prognosis in regard to either life or developing weakness. Also, changes in the patient's condition can develop rapidly, and, if the patient is hospitalized, appropriate measures can be immediately instituted. However, if the patient is at home, the attendant may not notice the signs, the physician may not be located promptly, and the transportation to the hospital occur at a most inappropriate time from the point of view of the patient's welfare.

In some areas private duty nurses are scarce, and, if the patient remains at home, his care will be the responsibility of nonmedical attendants. If a diagnosis is made late in the disease, when the temperature is returning to normal and care is adequate, home care has advantages. However, we are preaching early diagnosis, and it would seem that the lack of relationship between early signs and symptoms and prognosis would indicate that hospitalization in early cases is preferable.

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EOSINOPENIC RESPONSE TO EPINEPHRINE

To the Editor:—It is unfortunate that so many investigators, such as Drs. Charles W. Dowden and James T. Bradbury (Eosinophil Response to Epinephrine and Corticotropin: Studies in Alcoholics and Nonalcoholics, *J. A. M. A.* **149**:725-728 [June 21] 1952) continue to use subnormal eosinopenic response to injection of epinephrine as a means of testing pituitary-adrenal function.

In 1950, on the basis of animal investigations, Thorn and his associates (Recant, Hume, Forsham, and Thorn: Studies on the Effect of Epinephrine on the Pituitary-Adrenocortical System, *J. Clin. Endocrinol.* **10**:187, 1950) postulated that the hypothalamus, pituitary, and adrenal cortex are successively stimulated to produce epinephrine eosinopenia. Thorn and Forsham wrote that ". . . a fall of 50% or more in circulating eosinophils (after injection of epinephrine) excludes the presence of serious pituitary A.C.T.H. or adrenal cortical insufficiency," (Thorn and Forsham: Clinical Tests for Adrenal Cortical Reserve, in Progress in Clinical Endocrinology, Soskin, S., editor, New York, Grune and Stratton, 1950, p. 213). However, a large number of physicians interpret the test erroneously. They believe that an inadequate response, i. e., of less than 50% fall, indicates disease or relative dysfunction of the hypothalamus, pituitary, or adrenal cortex.

My colleagues and I have observed greater than 50% drop of eosinophils following injection of epinephrine in patients with pituitary tumors and in patients who were subjected to adrenalectomy and who were receiving small doses of cortisone. Responses of less than 50% were seen in a large number of patients with unrelated pathological conditions (Best, Muehrcke, and Kark: Studies on Adrenocortical Eosinopenia, abstract, *J. Clin. Investigation* **30**:629, 1951; Best, Muehrcke, and Kark: Clinical and Statistical Evaluation of Four-Hour Eosinophil Response Tests, abstract, *J. Lab. & Clin. Med.* **38**:790, 1951; Muehrcke and others: Mechanisms of Adrenocortical Eosinopenia: Antagonisms Between Heparin and Cortical Hormones and the Action of Epinephrine Post-Adrenalectomy, *Proc. Inst. Med. Chicago* **19**:68, 1952. Kark and Muehrcke: Does Adrenaline Eosinopenia Reflect Pituitary Adrenal Function in Man? *Lancet* **1**:1189-1191, 1952. Best, Muehrcke, and Kark: Studies on Adrenocortical Eosinopenia: A Clinical and Statistical Evaluation of Four-Hour Eosinophil Response Tests. *J. Clin. Investigation,* to be published). These observations, together with clinical and statistical analysis of 702 four-hour response tests in 284 persons, indicate that the epinephrine test is of little value in the diagnosis of adrenal, hypothalamic, or pituitary disease; nor does it accurately assess the function of these three organs.

The verbal reports given two weeks ago at the annual meeting of the Endocrine Society indicate that injection of epinephrine into healthy persons does not produce a uniformly significant increase in urinary 17-ketosteroid excretion (Jeffries and Bochner: A Comparison of Effects of Single Injection of A.C.T.H. and Epinephrine upon Adrenal Cortical Activity) or in blood levels of 17-hydroxycortico-steroids (Nelson, Sandberg, Palmer, and Glenn: Levels of 17-Hydroxy-Corticosteroids Following Intravenous Infusion of Epinephrine into Normal Men). Our observations and these two reports indicate that the mechanisms of epinephrine-induced eosinopenia are yet obscure. Therefore, we believe the epinephrine response test should be abandoned in clinical practice, at least until the mode of action is clarified. Moreover, the test should not be used to test pituitary-adrenal activity in groups of patients. Proper assessment of functional endocrine changes in alcoholics, in schizophrenics, and in others must await assay of corticotropin and adrenal steroids in these patients.

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POSTPARTUM HEMORRHAGE

To the Editor:—In THE JOURNAL of June 21, 1952, there is an article by Dr. Ernest Myller titled "Control of Postpartum Hemorrhage" (page 757). In this article I am quoted as saying it is my "belief . . . that when the first uterine packing is not successful, the uterus is probably ruptured. If such is the case, a second packing is definitely contraindicated." This is not only a complete misquote, it is also contrary to my teaching of a number of years.

The correct quotation from the source used by Dr. Myller is ". . . severe postpartum hemorrhage which does not respond to massage and the rather efficient oxytocic agents, when given intravenously, particularly when it occurs in the multigravida, is quite likely to be due to a partial or a complete rupture of the uterus. If this be present, packing will not only do no good, but will actually be harmful, in that vitally necessary time will be lost."

Actually, in our clinics at the University Hospital, Baltimore, and the Baltimore City Hospitals, the uterine pack has not been used to control postpartum hemorrhage for more than six years, representing about 30,000 consecutive deliveries. It is my belief that the uterine pack to control postpartum hemorrhage has absolutely no place in present-day obstetrics.

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FUNGUS INFECTIONS

To the Editor:—In the editorial "Fungus Infections Complicating Antibiotic Therapy," which appeared in THE JOURNAL, June 21, 1952, page 762, mention is made that my co-workers and I reported six instances of pulmonary moniliasis. This notation is inserted in a paragraph in such a way that it would appear that these six instances followed the use of one of the antibiotics discussed in the article. I would like to correct this impression and note that all the patients studied had pulmonary moniliasis and received no antibiotic therapy and that each of these patients was studied in the era before aureomycin, chloramphenicol, and terramycin came into general use.

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BUREAU OF LEGAL MEDICINE AND LEGISLATION

MEDICOLEGAL ABSTRACTS

Malpractice: Drainage Tube Left in Patient Following Operation.—The plaintiff sued for damages alleged to have been caused by the malpractice of the defendant physician. The jury found in favor of the plaintiff, but the trial judge entered judgment for the defendant notwithstanding the verdict. The plaintiff therefore appealed to the Supreme Court of California.

The plaintiff consulted the defendant physician concerning a severe pain in his scrotum. The ailment was diagnosed as a varicocele, and an operation was recommended and performed by the defendant. The plaintiff told the defendant that he did not want to lose his left testicle, and the defendant said that the operation was a simple one and that if the testicle was healthy there would be no occasion for its removal. Shortly after the operation the defendant told the plaintiff's mother that the left testicle was smaller than the other one but that it appeared healthy and he had not removed it. The day following the operation the plaintiff's scrotum began to swell and become painful. The dressings had to be removed. The evidence showed that at that time the operative incision was tightly sewed and that no drainage tube protruded from it. Penicillin was administered, but the swelling continued, and about a week after the operation the defendant probed the wound with surgical forceps to establish drainage and directed the plaintiff to take sitz baths. After about two weeks he was taken home but was instructed to continue the baths and to walk as much as possible. His mother helped him with the baths and dressed the incision. About two months after the operation, while cleaning pus from the probe wound, the plaintiff and his mother noticed, deep in the wound, the edge of a substance that resembled "a muscle." When this substance was touched with the swab stick, about an inch of white rubber tube protruded. On the following morning, the rubber tube stuck to a dressing and came out. The plaintiff and his mother described it as being "very wet and full of pus." This was the first time either the plaintiff or his mother knew that a rubber tube had been inserted in his scrotum. The plaintiff immediately telephoned the defendant who said: "That is fine. I left it for a drain. I am glad you found it." The defendant then advised the plaintiff that a further operation would be necessary and that the left testicle would have to be removed. This operation was performed by the defendant assisted by Dr. Gummess, who had also assisted at the first operation. The left testicle was removed, and a drainage tube was inserted, its presence being indicated on the operative report. Several days later the drain was removed, and the plaintiff left the hospital. It was the plaintiff's contention that a rubber drainage tube had been negligently left in the incision after the first operation and that such negligence resulted in the necessity for the removal of his left testicle.

Concerning the first operation, the defendant testified that he had placed a rubber tube in the plaintiff's scrotum for drainage purposes. He described it as being attached to the wall of the scrotum by sutures, part of the tube protruding from a "stab wound" made in the lowest part of the scrotum. He admitted, however, that he told no one except Dr. Gummess of the tube. He did not tell the interns or nurses of its presence and "overlooked" mention of the tube or stab wound in making his operative report, although it called for pertinent information on drainage. When asked whether the tube might have slipped entirely inside the scrotum, the defendant replied that such was ". . . within the realm of possibility . . . ." Dr. Gummess substantiated the defendant's testimony as to the insertion of the tube but said that he did not see it at any time when he examined the patient after the operation. As a practicing specialist in urology, Dr. Gummess was asked whether ". . . in an operation for the removal of a varicocele, under proper medical and surgical practice here, would a physician and surgeon intentionally permit a wound to close, leaving a rubber tube within the scrotum, with no part of the rubber tube protruding?" He replied, "No, he would not." It was further established, by expert testimony, that drainage of scrotal wounds is a mandatory rule because "proper drainage will prevent infection." Two experts identified the pain and other symptoms of disease in the plaintiff's scrotum after the operation as "classical signs" and "indications" of infection, which indicates bacterial attack on living tissue. The defendant admitted that there was infection present in the incision that was draining and that the draining involved the testicle, the spermatic cord, and the scrotum. Such infection, he said, can cause tissue to become necrotic.

On this evidence the jury returned a verdict in favor of the plaintiff, but as previously stated, the defendant's motion for judgment notwithstanding the verdict was granted. A defendant's motion for judgment notwithstanding the verdict, said the supreme court, may properly be granted only when, disregarding conflicting evidence and indulging in every legitimate inference that may be drawn from the plaintiff's evidence, the result is a determination that there is no evidence sufficiently substantial to support the verdict. Relying on this rule, the plaintiff contended that the evidence, disregarding conflicts, and the legitimate inferences to be drawn from it furnished sufficient support for the jury's implied finding that the defendant was negligent in performing the varicocelectomy, which negligence proximately caused the subsequent removal of the testicle.

The record fully supports the plaintiff in this regard, said the Supreme Court. From the evidence presented by the respective parties, the jurors reasonably might have concluded that the rubber tube was completely enclosed within the scrotum at the time of the operation and the incision tightly sutured. If, however, they chose to believe that the defendant left the tube protruding, there is ample evidence tending to prove a failure to fasten it so as to prevent withdrawal into the scrotum, a possibility admitted by the defendant. On either view of the evidence, it shows a surgical procedure that permitted the operative incision to become tightly closed, leaving the rubber tube completely within the scrotum. One of the physician witnesses in the case testified that "It is a rule, it is a mandatory rule, that we always drain scrotal wounds . . . ." The evidence would justify a conclusion that the defendant violated this "mandatory rule" by failing to drain the scrotum during the period following surgery, either because he made no provision for drainage or because he permitted the drainage tube to slip into the scrotum and the operative incision to close completely.

There is no merit, the Supreme Court continued, in the contention that the evidence failed to establish a causal relation between the failure to properly provide for drainage and the subsequent necessity for removal of the testicle. "The law does not require that negligence of the defendant must be the sole cause of the injury complained of in order to entitle the plaintiff to damages therefor. All that is required . . . is that the negligence in question shall be a proximate cause of the injury . . . ." It was shown that at the time of the first operation the plaintiff's testicle was healthy and for that reason the defendant did not remove it. Yet, 42 days later, it was in such condition that removal was deemed necessary. Although the defendant argued to the contrary, the jury's implied finding that a causal relationship existed was not purely speculative. According to the medical testimony, the reason for the "mandatory rule" of drainage is the danger of infection. That testimony also characterized the plaintiff's symptoms after the operation as "classical signs" and "indications" of infection, and unquestionably it was a necrotic condition that necessitated the second operation.

Accordingly, the Supreme Court reversed the judgment of the trial court in favor of the defendant physician with directions to the trial court to enter judgment in favor of the plaintiff. *Champion v. Bennetts, 236 P. (2d) 155 (California 1951).*

MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A.M.A. Arch. Ophthalmology, Chicago

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Conservative Treatment of Fractures of Tibial Plateau. C. E. Badgley and S. J. O'Connor.—p. 506.
Surgical Emergencies Resulting from Corpus Luteum Cysts and Hematomas. T. Taniguchi, J. A. Klieger and M. J. Kuhn.—p. 516.

Cardiac Arrest.—Although there has been increasing interest in cardiac arrest, its pathogenesis and the possibilities of its prevention have not received sufficient attention. The "specific tissue" of the heart has no distinguishing anatomic features, but it has the capacity to originate rhythmical contractions autonomously. It is the hardiest tissue in the body, being able to function for long periods without oxygen or even blood supply. The many sources of reflexes that could influence the specific tissue are listed, and the role of anesthesia, anoxia, vitamins of the B group, and endocrine imbalance are discussed. In 35 cases in which a wide variety of anesthetic agents and stimuli were used, 19 patients showed evidence of functional derangements in the specific tissue. At the Lahey Clinic in the last seven years there have been 14 cases of cardiac arrest of such severity as to require surgical intervention, but there must have been many less serious cases that nevertheless represent examples of a vagovagal reflex. The specific tissue that has the capacity to originate stimuli is widely distributed throughout the auricles and ventricles, but there are no vagal fibers in the ventricles. In a normal healthy heart no stimulus from any source, including the carotid sinus, can cause a general collapse, because the specific tissue in the ventricles, being free of vagal influences, can form stimuli and function autonomously. Undesirable reflexes that interfere with the specific tissue may originate in the mucosa of the respiratory or gastrointestinal tracts and in the pleura; those from the pulmonary vessels act on the coronary vessels. Anesthesia depresses the capacity of the specific tissue to form stimuli; in other words, a heart under anesthesia is as vulnerable to vagal stimulation as a heart with widespread organic disease in the specific tissue. Anoxia per se will not cause or initiate these reflexes, but its presence enormously potentiates the action of the anesthetic agent, with a still further depression of the capacity of the specific tissue to form stimuli. Vitamin B complex is important for smooth cardiac function, and in deficient nutrition its adequate use preoperatively is valuable. During endocrine imbalance, such as in the climacteric, substitution therapy is a good preoperative measure. The authors stress that the introduction of various mechanical contrivances into the respiratory or gastrointestinal passages and the clamping or tying of structures containing vagal nerve endings are not without danger, and atropine should be given. In any case in which the surgeon restores cardiac activity after a cardiac arrest, with the possible exception of ventricular fibrillation, the standstill was due to a sudden depression in the automaticity of the specific tissue following a reflex stimulus and almost certainly was preventable.

Alabama State Medical Assn. Journal, Montgomery

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Essential Adjunct to Scientific Medicine: Personal Relationship in Private and Hospital Practice. A. S. Dix.—p. 281.
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American Heart Journal, St. Louis

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Interauricular Septal Defect: Correlation of Clinical, Radiologic, and Electrocardiographic Findings in 15 Cases, with Special Reference Given to Electrocardiogram. N. W. Smull and L. E. Lamb.—p. 481.
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Effect of Induced Ventricular Premature Systoles upon Precordial QRS Pattern in Case of Right Ventricular Hypertrophy. N. O. Fowler, R. N. Westcott, R. C. Scott and J. Taguchi.—p. 521.
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*Prognostic Significance of Anoxemia Test in Coronary Heart Disease: Follow-up Study of 254 Subjects. J. A. L. Mathers and R. L. Levy. —p. 546.
Importance of Patent Ductus Arteriosus in Infants. R. F. Ziegler. —p. 553.
Unusual Mechanism of Sudden Death in Congenital Heart Disease. F. F. Rosenbaum and F. C. Stiles.—p. 573.
Emetine Therapy and Heart Disease. W. A. Sodeman.—p. 582.
*Comparison of Effects of Veratrum Alkaloids and of Hexamethonium Bromide on Blood Pressure in Arterial Hypertension. F. H. Smirk and O. W. Chapman.—p. 586.
Pulmonary Heart Disease Due to Bilharzia: Bilharzial Cor Pulmonale. Clinical Study of 20 Cases. B. Girgis.—p. 606.

Electrocardiographic Finding in Single Ventricle Hearts.—It is generally agreed that the QRS complex of the electrocardiogram is due to depolarization of the ventricular muscle. The muscle bundles in the ventricle are those of the left ventricle, those of the right ventricle, and those of the interventricular septum. The configuration of the QRS curve depends on the relative size

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Titles marked with an asterisk (*) are abstracted.

of these muscle bundles and the direction of the wave of excitation with respect to the location of the exploring electrode. A heart with a single ventricle with either one or two atriums, can be studied electrocardiographically with respect to the ventricular complex. Freireich and Nicolson studied two patients with a single ventricle. One of these patients is still alive, and the diagnosis of single ventricle was confirmed at operation, in which a subclavian artery was anastomosed to the pulmonary artery. Necropsy in the other patient revealed true cor triloculare biatriatum, with two well-defined atriums, a single large ventricle, and a rudimentary chamber. In the fatal case, the left leg lead was the inverted mirror image of the right arm lead, while in the other case, the left leg lead showed a similar relationship with the left arm lead. This was probably due to the different electrical positions of the single ventricle in relation to the extremities. The ventricular complexes in all the leads consisted of only one or two deflections, a qR, QS, or R.

Anoxemia Test in Coronary Heart Disease.—Mathers and Levy emphasize three points with regard to the anoxemia test: 1. The test should be employed clinically only when the diagnosis is in doubt. 2. A positive result affords objective evidence of coronary insufficiency. 3. A negative result does not rule out the possible presence of coronary heart disease. In order to ascertain the prognostic value of the test, a follow-up study was made of 254 persons on whom it was performed during the decade 1937 to 1947. Included were 141 patients with manifest or suspected coronary heart disease, 10 with hypertension, and 103 with no evidence of a cardiovascular disorder. The average follow-up period was six years. Sixty-three persons died during the course of the study. In the 16 cases in which an autopsy was performed, the clinical diagnosis was confirmed. In an effort to find correlations between the result of the anoxemia test, the clinical status, and the prognosis, a statistical analysis was made, but the results were inconclusive. The cumulative mortality rates of the patients with coronary sclerosis and anginal pain and of the subjects without cardiac symptoms, grouped according to the result of the initial anoxemia test, showed distinct differences. In the patients with coronary disease whose tests were positive, the prognosis was significantly worse with respect to longevity than in those whose tests were negative. It should be emphasized, however, that, whereas this is true for the group as a whole, it does not necessarily apply to any individual patient. The anoxemia test furnishes an index of the coronary erve only at the time of its performance.

um Alkaloids versus Hexamethonium Bromide in Hyper- —In 36 patients with arterial hypertension attempts were le to reduce the blood pressure by oral administration of ixed Veratrum alkaloids. In 27 continued treatment with hexamethonium bromide was also attempted. The blood pressure was reduced substantially without important symptoms in 10 of 36 patients with mixed Veratrum alkaloid therapy and in all of 27 patients with hexamethonium bromide injected subcutaneously. Treatment with hexamethonium was continued for from 1 to 15 months. There seemed no way of predicting without individual trials which patient would respond to Veratrum alkaloids; if, however, the patient was responsive, the blood pressure level throughout the day could usually be well controlled by spacing the doses, though mild toxic symptoms were likely to occur periodically. Moreover, Veratrum alkaloids seemed to be safe in the proper doses, and their hypotensive action was not excessive. Administration is easy, and, if tolerance to the drug does develop, it is very slight. Mixed Veratrum alkaloids seem unsuitable for treatment of acute cardiac complications of hypertension and encephalopathic attac ratrum
alkaloids and hexamethonium bromide resemble e in
that the therapeutic range of dosage is narrow. Grad
can be obtained with less difficulty using hex
bromide. Continued administration leads to drug tol
hexamethonium bromide, but not with Veratrum alk
effective dose of hexamethonium bromide becomes s
some mo toleration does not interfere with
treatment. thonium bromide treatment bl
sure falls a the standing, intermediate in the
and least in e. After ingestion of Veratru
loids postural conspicuous. After ingesthexamethonium bromide the pulse is ordinarily accelerated, but following continued treatment it may be unchanged. Bradycardia occurs with Veratrum alkaloid therapy. The main toxic manifestation of hexamethonium bromide is faintness and lassitude associated with excessive fall of blood pressure; the symptoms are maximal when the blood pressure is at its lowest level. Toxic effects of the Veratrum alkaloids occur while the blood pressure is still falling, and may disappear before the blood pressure reaches its lowest level.

American Journal of Hygiene, Baltimore

55:155-322 (March) 1952. **Partial Index**

Experiments on Feeding Adult Volunteers with *Escherichia Coli* 3. Bi, Coliform Organism Associated with Infant Diarrhea. W. W. Ferguson and R. C. June.—p. 155.
Encephalitis and Pneumonia *Following Intranasal* Inoculation of Newcastle Disease Virus *in Different Strains of Mice.* C. Liu and F. B. Bang.—p. 182.
Q *Fever* Studies in Southern *California:* XIII. Vaccination of Dairy Cattle Against Q Fever. L. Luoto, J. F. Winn and R. J. Huebner. —p. 190.
Use of Triethylene Glycol Vapor for Control *of* Acute Respiratory Diseases in Navy Recruits. United States Naval Medical Research Unit No. 4.—p. 203.
Ornithosis (Psittacosis) in Chickens and *Poultry Workers.* A. Mandel and W. S. *Jordan Jr.*—p. 230.
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*Q Fever Studies: XIII: Effect of Pasteurization on Coxiella Burneti in Naturally Infected Milk. E. H. Lennette, W. H. Clark, M. M. Abinanti *and others.*—p. 246.
Id.: XIV. Observations on *Pathogenesis* of Experimental Infection Induced in Sheep by Intravenous *Route.* E. H. *Lennette,* M. A. Holmes and F. R. Abinanti.—p. 254.
Precision of Measurement of Lansing Virus Infectivity in Cotton Rats. C. E. *Schwerdt* and M. Merrell.—p. 268.
Studies on Plasmodium Gallinaceum*:* II. Factors in Blood of Vertebrate Host Influencing Mosquito *Infection.* D. E. Eyles.—p. 276.
Studies on Poliomyelitis Viruses in Cultures of Monkey Testicular Tissue. J. S. Youngner, E. N. Ward and J. E. Salk.—p. 291.

Effect of Pasteurization on Coxiella Burnetii.—The effect of pasteurization on the survival of Coxiella burnetii was investigated. Pasteurization was carried out in commercial processing plants under routine conditions. In 1 of 35 instances the organism survived pasteurization by the vat-holding method; in this instance, however, pasteurization, as judged by the phosphatase test, may not have been complete. In 2 instances of 42 the organisms were detected in milk pasteurized by the high temperature and short time method. In an addendum to this report the authors state that after this paper had been prepared for publication, there came to their attention the report of Marmion and his associates on "The Effect of Pasteurization on Milk Containing Rickettsia Burneti," (*Monthly Bull. Ministry of Health and the Pub. Health Lab. Service* 10:119-128, 1951). These workers found that treatment of naturally infected milk at 154 to 156 F for 15 seconds was sufficient to render the milk noninfective for guinea pigs. On the other hand, artificially infected milk containing large numbers of rickettsiae still possessed infectivity after treatment at 158 to 159 F for 15 seconds, but was rendered noninfective after treatment at 161 F for 15 seconds.

American Journal of Medicine, New York

12:379-498 (April) *1952*

Cerebral Circulation and Metabolism in Chronic Pulmonary Emphysema, With Observations on Effects of Inhalation of Oxygen. *J.* L. Patterson Jr., A. Heyman and T. W. Duke.—p. 382.
Pulmonary Function in Patients Convalescing from Acute Poliomyelitis with Respiratory Paralysis. D. S. Lukas and F. Plum.—p. 388.
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*Some Supravital Observations on "L. E." Phenomenon. R. J. Rohn and W. H. Bond.—p. 422.
Effectiveness of Various Drugs for Prophylaxis of Seasickness. H. I.
inn, S. W. Handford, T. E. Cone Jr. and P. K. Smith.—p. 433.
of Therapeutic Effects of Some Amebacidal Drugs. W. A. Sode-
d P. C. Beaver.—p. 440.

Observations on "L. E." Phenomenon.—After re-
nt literature on the "L. E." cell phenomenon,
present their method for obtaining, preparing,
staining peripheral blood and bone *marrow*

specimens. Using this technique they examined approximately 5,000 peripheral blood and 800 bone marrow specimens of patients referred to a hematology service during one year. They demonstrated L. E. cells in five patients. They found that the L. E. cell has two component parts. The L. E. cell inclusion body was always a mature, autolyzed polymorphonuclear neutrophil. The phagocytic cell was either a mature polymorphonuclear neutrophilic, eosinophilic, or basophilic leukocyte, with the neutrophilic leukocytes overwhelmingly predominating. Bone marrow examination proved the most reliable means of demonstrating L. E. cells by their technique, even though, on routine examination, they found L. E. cells in the peripheral blood in four of the five summarized cases. The authors regard this as a simple, effective, direct method for demonstrating L. E. cells without special procedures on peripheral blood or bone marrow.

Annals of Internal Medicine, Lancaster, Pa.

36:933-1156 (April) 1952

•Morphology and Pathogenesis of Cardiac Fibrosis of Liver. E. Moschcowitz.—p. 933.
Diverticula of Gastrointestinal Tract. M. A. Gold and J. G. Sawyer.—p. 956.
Provocative Serological Reaction in Late Syphilis: Its Relation to Technical Factors. R. W. Maxwell and V. Scott.—p. 979.
•Cortisone and Gold Therapy in Chronic Rheumatoid Arthritis. H. E. Thompson and H. J. Rowe.—p. 992.
Acute Severe Upper Gastrointestinal Hemorrhage: Review of 195 Cases. J. R. Gott Jr., E. L. Smith and D. D. Dornan.—p. 1001.
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Diagnostic Problems of Rheumatic Fever and Their Impact on Management of Rheumatic Fever Patient. F. J. Lewy.—p. 1042.
•Soft Tissue Calcification, with Special Reference to Its Occurrence in "Collagen Diseases." G. E. Wheeler, A. C. Curtis, E. P. Cawley and others.—p. 1050.
Oral Use of Combined Vitamin B_{12} and Folic Acid in Tropical Sprue. F. Diéz Rivas, F. Hernández Morales and L. M. Meyer.—p. 1076.
Effect of Penicillin on Renal Lesions of Subacute Bacterial Endocarditis. D. M. Spain and D. W. King.—p. 1086.

Hepatic Fibrosis in Cardiac Disease.—Moschcowitz prefers the term fibrosis to cirrhosis, and points out that there are widely differing opinions concerning the morphology and pathogenesis of hepatic lesions associated with cardiac disease. The earliest phase in the development of this condition is that characterized by what is usually referred to as "nutmeg" liver, in which there is dilatation of the central sinusoids with trabecular narrowing. Later, centrolobular necrosis ensues. Thereafter, the morphological changes depend on whether hypertension of the portal circulation is maintained long enough. This usually occurs in constrictive pericarditis, often in the terminal stages of tricuspid valve disease. That portal hypertension is maintained was shown by histological demonstration of "congestive splenomegaly." Under such circumstances, various forms of fibrosis occur: first, capillary sclerosis around the central veins; second, organization of the centrolobular necrotic areas with the development of new central veins in these fibrotic areas, either by autochthonous formation of capillaries or by direct sprouts from the terminal branches of the portal vein in the interlobular septa; and third, by sclerosis of the hepatic veins and the immediately adjacent parenchyma. The periportal fibrosis that has been described as occurring in "cardiac cirrhosis" bears no resemblance to that observed in true Laennec's cirrhosis. Cardiac fibrosis is not a replacement fibrosis but is an active and proliferative process. New capillary shunts or short circuits are developed simultaneously. There are four kinds of such shunts: first, between the terminations of the portal vein and the central vein in the interlobular septa; second, by newly formed capillaries that bud from the terminal branches of the portal veins within the portal septa to the central veins; third, by budding from the preexisting portal branches in the interlobular septa to the newly formed central veins; and fourth, by direct capillary communication between the terminations of the portal veins in the interlobular septa and the hepatic veins. These shunts are compensatory mechanisms to help equalize the pressures between the portal vein and the hepatic veins. Evidence is submitted that the morphological changes of cardiac fibrosis are the result of portal hypertension of central origin. Advanced hepatic cirrhosis may be suspected if there is constrictive pericarditis of long duration, persistent and recurring ascites, increased peripheral venous pressure, splenomegaly, and enlargement of right auricle and ventricle, and dilatation of the pulmonary artery.

Cortisone and Gold in Chronic Rheumatoid Arthritis.—A total of 92 patients with active chronic rheumatoid arthritis were divided into four series: first, eight patients who had been receiving gold with only moderate benefit were given cortisone in addition; second, 13 patients were given gold and cortisone concurrently; third, 42 patients were given gold alone; and fourth, 29 patients were given cortisone alone. For comparison, 273 patients who received neither gold nor cortisone were restudied. The dosage levels were as follows: Cortisone was given in doses of 100 mg. intramuscularly every eight hours for 24 hours, then every 12 hours for 24 hours, and then 100 mg. daily for 21 days. Then cortisone therapy was discontinued until a relapse occurred. It was then reinstituted at the previous level and, after 14 days, 75 to 100 mg. were given every other day parenterally, or 50 to 75 mg. per day orally. Administration of cortisone was then again discontinued. Gold was given in initial doses of 10 mg. either intramuscularly or intravenously, and then at weekly intervals in 50 mg. doses until a total of 1 gm. had been given. Administration of gold was then continued at 10, 14, 21, and 30 day intervals. The authors gained the impression that gold given to patients with active chronic rheumatoid arthritis with moderate benefit apparently had no additional effect when cortisone was added. Despite an excellent therapeutic response, no complete remissions were observed in this group of eight patients, and they returned to their precortisone state shortly after administration of the hormone was discontinued. Gold given concurrently with cortisone seemed to arrest rheumatoid arthritis in approximately the same percentage of patients as gold given alone, but it appeared superior to cortisone employed alone, and to therapy without gold or cortisone. Gold and cortisone administered concurrently gave an immediate increase in functional capacity, and in some patients it produced a complete remission. These effects continued in a number of patients when cortisone was discontinued. In addition, the concurrent administration of gold and cortisone has several therapeutic advantages: first, patients who have previously been intolerant to gold can be given gold with cortisone with less danger of a reaction (to gold). Second, with cortisone rehabilitative measures can be carried out before the gold levels are sufficiently high to be effective. Third, administration of cortisone may be discontinued in the patients who respond to gold.

Soft Tissue Calcification.—The term calcinosis circumscripta and calcinosis universalis imply the deposition of calcium salts in skin and subcutaneous tissue. Much of the confusion and mystery surrounding the use of the terms calcinosis circumscripta and calcinosis universalis stems from the practice of regarding calcification as a primary process, whereas careful study will always reveal a primary disease responsible for the calcification. This report is concerned with the 54 instances of calcinosis of the circumscript or universal varieties reported in the literature since 1938 and 12 cases of several types of collagen disease studied by Wheeler and associates at the University of Michigan Hospital. Detailed reports are given of two of the latter 12 patients and brief reports of the other 10. A table that lists the primary diagnoses of all the 66 patients with calcinosis of the circumscript or universal varieties shows that most patients had scleroderma, Raynaud's syndrome, or dermatomyositis. Thirty-four of the patients are in the scleroderma group and 32 in the dermatomyositis group. In general, serum calcium, phosphorus, and alkaline phosphatase determinations are reported as normal in collagen diseases associated with calcification, but it is suggested that possibly calcium is being removed from the blood and stored in injured tissue. Calcification may be secondary to a disturbance of factors regulating blood serum calcium and/or phosphorus levels. It may also occur in the so-called collagen diseases as a result of tissue injury. The following diseases should be considered when calcinosis is found: scleroderma, dermatomyositis, Raynaud's syndrome, rheumatoid arthritis, acrodermatitis chronica atrophicans, hyperparathyroidism, vitamin D intoxication, metastatic bone disease, multiple myeloma, Paget's disease of bone, renal failure, and pseudohypoparathyroidism. A table lists the results of laboratory tests and roentgenograms of the skeletal system to be expected in these conditions.

Annals of Surgery, Philadelphia

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Relation of Gastric Secretion to Events Following Pyloric Obstruction. J. S. Clarke, H. A. Oberhelman Jr., S. O. Evans and L. R. Dragstedt. —p. 433.
*Electrolyte Abnormalities in Pyloric Obstruction Resulting from Peptic Ulcer or Gastric Carcinoma. H. S. Lans, I. F. Stein Jr. and K. A. Meyer.—p. 441.
*Response of Dicumarol-Induced Hypoprothrombinemia to Vitamin K_1. A. Rehbein, A. Jaretzki III and D. V. Habif.—p. 454.
Intravascular Agglutination and Vascular Stasis in Peptic Ulceration. J. A. Key.—p. 470.
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Perforation of Sigmoid Colon During Barium Enema: Report of Case with Review of Literature, and Experimental Study of Effect of Barium Sulfate Injected Intraperitoneally. L. J. Kleinsasser and H. Warshaw. —p. 560.
Congenital Atresia of Esophagus with Tracheo-Esophageal Fistula, Duodenal Atresia, and Other Anomalies: Case Report. W. C. McGarity, O. A. Abbott and L. W. Grove.—p. 566.
Intraperitoneal Rupture of Iliac Aneurysm with Survival Following Surgical Treatment: Report of Case. P. A. Fernbach, L. Berman and B. Cohen.—p. 570.

Electrolyte Abnormalities in Pyloric Obstruction.—Prolonged untreated illness and protracted use of potassium-free intravenous fluids may result in electrolyte imbalances. The loss of sodium and chloride accompanying vomiting is well known, and recent studies have shown that a considerable amount of potassium may also be lost as a result of vomiting or prolonged gastric suction. Patients with gastric carcinoma or vagotomy may have alkalosis, hypochloremia, and hypopotassemia, because high concentrations of chloride and potassium ions may be present in the gastric contents in spite of the absence of titratable hydrochloric acid. Analysis of the gastric contents in patients with pyloric obstruction shows that their daily potassium loss may qual from 6 to 8 gm. of potassium chloride. The loss of chloride ceeds that of sodium when the obstruction is due to peptic lcer, because free hydrochloric acid is present in the gastric juice; this leads to hypochloremia and alkalosis. Gastric resection, on the contrary, usually results in a loss of sodium exceeding that of chloride, leading to acidosis. Hypochloremia and alkalosis resistant to isotonic sodium chloride solution may be corrected by intravenous potassium chloride therapy. The addition of 3 gm. of potassium chloride to a liter of isotonic sodium chloride solution, amino acids, or 5% dextrose solution provides potassium and chloride ions in excess of sodium, resulting in an increase of intracellular potassium and extracellular chloride. Such solutions should be administered at the rate of from 120 to 180 drops a minute; faster administration may lead to a high serum potassium level, which is toxic to the heart and may cause heart block and cardiac arrest. Potassium must be given cautiously to patients with renal insufficiency or dehydration. Parenteral potassium therapy is not needed after the patient is able to eat, because most foods are good sources of potassium. This treatment resulted in a striking improvement in the mental attitude and muscular strength of all patients; 14 were in critical condition when it was begun, but all responded to therapy and there were no deaths.

Response of Bishydroxycoumarin-Induced Hypoprothrombinemia to Vitamin K_1.—Vitamin K_1 in the form of an emulsion may safely be given intravenously. It has proved effective in reversing hypoprothrombinemia due to bishydroxycoumarin (dicumarol®) and other prothrombin-depressing drugs, returning the prothrombin time to normal or nearly normal levels in approximately six hours irrespective of the degree of hypoprothrombinemia and the amount of recently administered bishydroxycoumarin. The response to a dose of 50 mg. was as prompt and effective as it was to larger doses; when a 50 mg. dose was given, resistance to subsequent doses of bishydroxycoumarin did not develop. The fact that vitamin K_1 controls bleeding by correcting the coagulation defect more rapidly than any other agent makes the use of whole blood and plasma unnecessary except when blood volume replacement is required. Vitamin K_1 is also valuable in reversing hypoprothrombinemia immediately before operation, and in reducing excessive hypoprothrombinemia not accompanied by bleeding to a therapeutic level so that anticoagulant therapy may continue without interruption. For the latter purpose doses of from 0.5 to 2.5 mg. of vitamin K_1 given intravenously will reverse the prothrombin time rapidly, returning it in most instances to a safe but still therapeutic level in from three to six hours when average doses of bishydroxycoumarin have resulted in excessively high levels.

Diseases of Chest, Chicago

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Use of Trypsin in Chest Disease. L. C. Roettig, H. G. Reiser, W. Habeeb and L. Mark.—p. 245.
Primary Pulmonary Tuberculosis as Cause of Bronchiectasis in Children. L. B. Dickey.—p. 260.
Detection and Identification of Intracardiac Calcifications. J. E. Habbe. —p. 268.
Evaluation of Ioduron B in Bronchography: I. Preliminary Study on Animal Tissues. W. Hentel, M. B. Cohen and L. C. Brandenstein. —p. 280.
Id.: II. Preliminary Study in Man. M. B. Cohen, W. Hentel and L. C. Brandenstein.—p. 286.
Lung Immobilization Chamber Therapy in Chronic Pulmonary Tuberculosis (with Case Reports). W. Grosfeld and J. C. Nash.—p. 296.
Pathogenesis of So-Called Idiopathic Spontaneous Pneumothorax and Related Conditions. A. Sattler.—p. 315.
Bronchial Onset of Reinfection Tuberculosis in the Adult. J. J. Castillo. —p. 320.
Pneumonia Associated with Ingestion of Petroleum Products by Children. M. L. Podolsky.—p. 327.
Epiphrenic Diverticulum of Esophagus: Review of Its Surgical Treatment and Report of Case. H. W. Kausel and G. E. Lindskog.—p. 334.
Pulmonary Histoplasmosis: Report of Case Long Considered Tuberculous Improved by Acti-Dione. H. M. Wilson and A. W. Duryea.—p. 346.

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On Lasting Protective Effect of Hydrazine Derivatives of Isonicotinic Acid in Experimental Tuberculosis Infection of Mice. E. Grunberg, B. Leiwant, I.-L. D'Ascensio and R. J. Schnitzer.—p. 369.
Tolerance of Macacus Rhesus for Isonicotinylhydrazines: Preliminary Report. R. A. Lewis and I. Zieper.—p. 378.
*Tuberculosis Chemotherapy with Hydrazine Derivatives of Isonicotinic Acid. I. J. Selikoff and E. H. Robitzek.—p. 385.
Determination of Blood Plasma Levels of Hydrazine Derivatives of Isonicotinic Acid. S. H. Rubin, L. Drekter, J. Scheiner and E. De Ritter. —p. 439.
Chloramphenicol in Control of Bronchopulmonary Suppuration. M. S. Harris, L. Gornell, C. Shore and H. Lovitz.—p. 450.
Chronic Friedlander's Pulmonary Infection Treated with Chloromycetin: Case Report. H. Steinberg and M. N. Towbin.—p. 455.
Pulmonary or Bronchial Lavage and Simultaneous Tomographies. M. de Abreu.—p. 460.
Tuberculous Empyema Treated with Streptokinase-Streptodornase: Report of Case with Cure. A. Goldman.—p. 478.

Hydrazine Derivatives of Isonicotinic Acid in Tuberculosis.—The authors report results of treatment with the hydrazide of isonicotinic acid ("rimifon") and its isopropyl derivative ("marsilid") in 92 patients with moderately or far-advanced pulmonary tuberculosis. All patients had positive sputum and active disease before institution of therapy; 45 were toxic and febrile. Patients, divided into groups, were treated with 2, 4, and 10 mg. per kilogram of "marsilid" and 4 mg. of "rimifon" for from 4 to 15 weeks. Fever and toxicity disappeared within two to three weeks in all cases. Improvement in appetite and in sense of well-being was associated with weight gains, which were as great as 64 lb. (29 kg.) and averaged 18 lb. (8.2 kg.) in nine weeks of treatment. The sputum became negative in 25% of patients and in another 28% sputum that had been consistently positive became "occasionally positive." Cough and expectoration disappeared completely or almost so in from two to three weeks. Cavity closure occurred in 2 cases, marked reduction in cavity size was noted in 33 cases, and clearing of exudate occurred 17 times. In four cases the exudate became more extensive, and in one case a new cavity seemed to form where exudate had been present before treatment. Cure or great improvement was observed in patients with tuberculous laryngitis, otitis, or glossitis. Toxic effects of marsilid and rimifon therapy included hyperreflexia, leg-twitching, constipation, vertigo, and reflex disturbance of micturition. No fatality resulted from use of these drugs, and every patient was benefited.

Endocrinology, Springfield, Ill.

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Acute Effect of Adrenal Cortical Extract, Adrenocorticotropic Hormone, and Desoxycorticosterone Acetate on Thiocyanate, Bromide, and Mannitol Spaces; Plasma Volume and Total Body Water in Normal Dogs. J. M. B. Bloodworth Jr.—p. 174.
Enhancement of Adrenocorticotropic Hormone Activity by Alum in Normal 21-Day Old Male Rats. T. Hayashida and Choh Hao Li.—p. 187.
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Journal of Bone and Joint Surgery, Boston

34A:249-504 (April) 1952. Partial Index

The Surgical Experiment. J. S. Barr.—p. 249.

SYMPOSIUM: STREPTOMYCIN IN TREATMENT OF BONE AND JOINT TUBERCULOSIS

Streptomycin in Bone and Joint Tuberculosis. D. M. Bosworth and H. A. Wright.—p. 255.
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*Evaluation of Streptomycin Therapy in Controlled Series of Ninety Cases of Skeletal Tuberculosis. Public Health Service Cooperative Investigation.—p. 288.

*Treatment of Tuberculous Bone Disease by Surgical Drainage Combined with Streptomycin. M. S. DeRoy and H. Fisher.—p. 299.
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Osteogenetic Potency and New-Bone Formation by Induction in Transplants to Anterior Chamber of Eye. M. R. Urist and F. C. McLean.—p. 443.
Sensory Nerves of Hip Joint. L. G. Wertheimer.—p. 477.
Surgical Approach for Neuroma of Plantar Digital Nerve (Morton's Metatarsalgia). D. C. McKeever.—p. 490.
Incidence of Separate Neural Arch and Coincident Bone Variations. M. B. Roche and G. G. Rowe.—p. 491.

Streptomycin in Skeletal Tuberculosis.—Of 90 patients with skeletal tuberculosis observed for 15 months, 50 were given at least one course of streptomycin with daily intramuscular doses of 20 mg. per kilogram of body weight for 91 days, while 40 were not treated with streptomycin. The small number of patients in the streptomycin and control groups limits the conclusions that may be drawn from the authors' observations, which are as follows: 1. On the basis of x-ray evidence alone, the condition of the patients in the streptomycin-treated and in the control groups was essentially the same; more than four-fifths of the patients in each group showed improvement roentgenographically at the end of 15 months. 2. On the basis of the patients' condition as judged by x-ray and clinical data combined, there was more improvement in the streptomycin-treated patients. At 15 months only 28 of the 40 patients in the control group showed improvement, whereas improvement was noted in 38 of the 50 streptomycin-treated patients as early as three months, and in 41 patients of this group at 15 months. 3. Streptomycin appears to produce a generalized effect on the well-being of the patient with skeletal tuberculosis and on the secondary manifestations of the tuberculous process, as illustrated by cessation of sinus drainage, reduction in the sedimentation rate, return towards a normal hemoglobin level, and evidence of greater improvement in functional ability. The data appear to substantiate the hypothesis that because of the relative avascularity of bone lesions and because a tuberculous focus is usually isolated by fibrous tissue walls, little streptomycin reaches the skeletal lesion. Hence the roentgenographic evidence of progress is quite similar for the streptomycin and control groups. However, streptomycin may ameliorate the toxic effects of the tubercle bacillus, and, in contiguous areas where the vascular supply is better, may decrease the spread of the disease process and bring about improvement in the general condition. Clinically patients treated with streptomycin were in a better condition than the control patients.

Surgical Drainage Combined with Streptomycin in Tuberculous Bone Disease.—Treatment with streptomycin in doses of 1 gm. per day intramuscularly in conjunction with the surgical incision of abscesses, débridement of lesions, and their conversion to freely draining sinuses by the use of gauze packing was practiced between October, 1946, and April, 1950, in 52 patients between the ages of 1 and 72 years who had 63 closed tuberculous bone lesions. Excellent results were obtained in 37 of the 52 patients, with cessation of bone and joint destruction, closure of all sinuses, absence of pain, retention of 75% or more of active peripheral joint motion or solid surgical fusion when performed, no recurrence of drainage, and improvement in general health. Thirteen patients obtained good results, with cessation of bone and joint destruction, closure of all sinuses, relief of pain, retention of 25 to 75% of active peripheral joint motion, recurrence of slight drainage (additional streptomycin being required for closure), and improvement in general condition. Two patients died, one 18 months after operation, of tuberculosis of the kidney and lungs and amyloid disease, although the bone lesions were healed. The other died of amyloid disease and destruction of the kidneys 37 months after operation. Although the series is small and the average follow-up period of three and a half years is short, the following preliminary conclusions may be drawn: Following adequate surgical drainage and treatment with streptomycin, destruction from bone and joint tuberculosis ceased, and roentgenograms showed evidence of recalcification and apparent regeneration of bone. There was no tendency toward reactivation of the lesion, clinically or symptomatically. In the surgically produced sinuses, subsidence of drainage and closure in from 3 to 16 weeks occurred. Although the surgically treated lesions will heal clinically, new lesions in other parts of the body may develop and progress, despite administration of streptomycin. Bone tends to regenerate in children, with reestablishment of an apparently normal epiphyseal plate and no disturbance in the rate of growth. A good functional range of motion was the usual end result, even when there was roentgenographic evidence of extensive destruction of the joint. The factor of secondary infection was apparently of no consequence in the arrest and healing of these lesions.

J. Bowman Gray School Med., Winston-Salem, N. C.

10:27-56 (April) 1952

- *Potassium Deficiency States and Their Clinical Management. H. H. Cook Jr.—p. 27.
- Effects of Hepatic Factors on Peripheral Vascular System. C. McMillan. —p. 38.

Potassium Deficiency.—Reviewing the present status of knowledge on potassium deficiency, Cook stresses that it is a common disorder and should be suspected any time oral feeding is diminished sharply or discontinued with continued urinary excretion or there is persistent loss of gastrointestinal secretions in any form, and in diabetic acidosis and coma. Potassium deficiency also occurs in digitalis intoxication, with adrenocortical substitution therapy (including administration of corticotropin), in familial periodic paralysis, and in bulbar poliomyelitis, and is of particular importance in the latter where there is usually a marked decrease in respiratory reserve. The symptoms associated with hypopotassemia are nonspecific but usually include weakness, drowsiness, langour, hypotonia of the skeletal muscles, dyspnea with gasping respiration, cyanosis, nausea, vomiting and occasionally mild sensory disturbances in the form of mild paresthesias. The diagnosis depends not so much on the symptomatology as on recognition of situations in which potassium deficiency is likely to occur. The electrocardiogram is the most practical adjunct to the clinical impression. Typical electrocardiographic changes are prolongation of the Q-T interval. The T wave is decreased in amplitude and ultimately inverted. The S-T segment sags, and the S-T takeoff is depressed. These signs are exactly opposite to those characteristic of potassium intoxication. In the treatment of potassium deficiency the oral route is preferred, and potassium chloride is probably the best drug. There is no danger from oral potassium therapy in patients with good renal function, but adequate laboratory facilities should be available when potassium is administered intravenously.

Journal Clin. Endocrin. & Metab., Springfield, Ill.

12:257-370 (March) 1952

- *Further Studies on Treatment of Congenital Adrenal Hyperplasia with Cortisone: I. Comparison of Oral and Intramuscular Administration of Cortisone, with Note on Suppressive Action of Compound F and B on Adrenal. L. Wilkins, L. I. Gardner, J. F. Crigler Jr. and others. —p. 257.
- *Id.: II. Effects of Cortisone on Sexual and Somatic Development, with Hypothesis Concerning Mechanism of Feminization. L. Wilkins, J. F. Crigler Jr., S. H. Silverman and others.—p. 277.
- Adrenal Cortical Function in Hypertension. B. S. Hetzel and D. C. Hine. —p. 296.
- Action of Estrogen and Progesterone on Gonadotropin Content of Pituitary of Monkey (Macaca Mulatta). H. A. Salhanick, F. L. Hisaw and M. X. Zarrow.—p. 310.
- Effects of Chorionic Gonadotropin and Prolactin on Maintenance of Corpus Luteum Function. P. H. Fried and A. E. Rakoff.—p. 321.
- Acid Phosphatase Concentration of Prostatic Exprimate During Normal Puberty. J. E. Kirk, A. Eisenstein and C. M. MacBryde.—p. 338.
- Effect of Cortisone on Tolerance to Ascorbic Acid and Level of Blood Cholesterol in Man. W. M. Booker, J. B. Johnson, R. L. Hayes and others.—p. 346.
- Studies on Mechanism of Spermatic Release of Male North American Frog (Rana Pipiens): Reaction to Various Hormones. S. L. Robbins and F. Parker Jr.—p. 354.

Cortisone in Congenital Adrenal Hyperplasia.—Eleven patients with congenital adrenal hyperplasia were treated continuously with cortisone for from five to 17 months. Six were female pseudohermaphrodites between the ages of 8½ and 18½ years, three were female pseudohermaphrodites, between 11 and 19 weeks of age when treatment was started, with defects of electrolyte regulation and two were boys with macrogenitosomia praecox, aged 4½ and 7 years. As a result of their experience with these patients, the authors make the following tentative suggestions: To obtain rapid suppression of the abnormal androgenic activity of the patient's adrenals, initial treatment with relatively large doses of intramuscular cortisone may be given. For patients eight years of age or over, 50 mg. of cortisone intramuscularly per day are adequate; in infants, 25 mg. per day. It seems preferable to begin treatment with cortisone intramuscularly rather than orally, because the results are more predictable. The maximum suppression of urinary ketosteroids should be attained within 5 to 10 days, after which the maintenance dose should be determined. For patients over 8 years of age the maintenance dose of cortisone given intramuscularly is usually 25 mg. per day. Doses of 75 mg. of cortisone every third day or 100 mg. every fourth day are just as effective. When cortisone is given orally doses of 50 or 75 mg. per day are required. These amounts should be given in equally divided doses, two, three, or four times a day. For young infants the maintenance dose of cortisone is smaller. In the authors' patients, intramuscular injections of 5 mg. daily and of 25 mg. every fourth day and oral doses of 5 mg. and 12.5 mg. per day seemed to maintain suppression, but larger amounts may be required in some cases. For children between the ages of 1 and 8 years, the maintenance dose is probably somewhere between these amounts and 25 mg. per day, when the drug is given intramuscularly. It should be determined individually for each patient. The effects of intramuscular medication seem to be more predictable, probably because of the more uniform rate of absorption. In young infants who have the sodium-losing type of congenital adrenal hyperplasia, it is important that treatment include a high sodium intake and/or desoxycorticosterone in addition to cortisone. In the one patient who was given compound F (17-hydroxycorticosterone) in doses of 25 mg. per day, it seemed to have approximately the same effect as cortisone on the suppression of adrenal activity. In an infant with the salt-losing type of adrenal hyperplasia, compound B (corticosterone) seemed to have somewhat less suppressive action than cortisone, but a definite sodium-retaining effect was exerted by doses of 12.5 mg. per day. The ultimate effects on the adrenal of the amounts of cortisone administered are not known. Although cortisone seemed to suppress the response of the adrenal to 24 hour tests with corticotropin (ACTH), the activity of the adrenals as measured by the 17-ketosteroid excretion returned promptly when cortisone was discontinued after 227 days of continuous therapy.

Effects of Cortisone on Sexual and Somatic Development.—The effects of cortisone on sexual and somatic development were studied in the 11 patients with congenital adrenal hyperplasia who were treated continuously with this drug for 5 to 17 months as reported in the preceding paper. In all six girls between the ages of 8½ and 18½ years the breasts promptly developed and the vaginal smears showed estrinization. Three of these patients have menstruated regularly. There has been an unquestionable decrease of hirsutism in all patients, and in two the decrease was quite pronounced. To obtain the most satisfactory results, it is essential to give the minimum amount of cortisone necessary to maintain the excretion of 17-ketosteroids below 8 mg. per day in the older patients, as recurrence of androgenic manifestations was observed when larger amounts of ketosteroids were excreted. In one infant, 25 mg. of intramuscular cortisone daily inhibited growth and osseous development, while 5 mg. suppressed excessive androgen secretion without inhibiting normal growth. During childhood it is advisable to follow both the urinary 17-ketosteroids and the rates of growth and osseous development as guides to treatment. The minimal effective dose of cortisone and the level at which the urinary 17-ketosteroids should be maintained must be determined individually in each case. In one of the two boys with macrogenitosomia praecox, cortisone led to adolescent development and growth of the patient's testes, suggesting that normal maturation was occurring. The rapid female sexual development in three pseudohermaphrodites between 8½ and 10½ years old and the testicular development in a boy aged 7 may be due to the fact that these patients had attained a somatic and osseous development of from 13 to 15 years, owing to the accelerating effects of excessive androgen. Apparently the pituitary-gonadal mechanism had matured enough to be capable of functioning promptly as soon as the inhibiting effects of adrenal hormones were suppressed.

Journal of Clinical Investigation, Cincinnati

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Responses to Painful Stimuli of Patients with Severe Chronic Painful Conditions. M. A. Kennard.—p. 245.

Diurnal Variation in Urinary Excretion of Neutral Lipid-Soluble Reducing Steroids in Congestive Cardiac Failure and Cirrhosis of Liver with Ascites. R. Goldman and S. H. Bassett.—p. 253.

Effects of Potent Analgesics upon Vestibular Function. L. B. Gutner, W. J. Gould and R. C. Batterman.—p. 259.

Effect of Theophylline with Ethylenediamine (Aminophylline) on Cerebral Hemodynamics in Presence of Cardiac Failure With and Without Cheyne-Stokes Respiration. J. H. Moyer, S. I. Miller, A. B. Tashnek and R. Bowman.—p. 267.

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Volume of Distribution of Sodium Thiosulfate as Measure of Extracellular Fluid Space. R. H. Cardozo and *I. S. Edelman.*—p. 280.

Electrolyte Content of Skeletal Muscle in Congestive Heart Failure: Comparison of Results with Inulin and Chloride as Reference Standards for Extracellular Water. R. Mokotoff, G. Ross and F. Leiter. —p. 291.

Disturbances of Ionic Equilibrium of Intracellular and Extracellular Electrolytes in *Patients with Tuberculous Meningitis.* H. E. Harrison, L. Finberg and E. Fleishman.—p. 300.

Validity of Endogenous Creatinine Clearance as Measeure of Glomerular Filtration Rate in Diseased Human Kidney. B. F. Miller, A. Leaf, A. R. Mamby and Z. Miller.—p. 309.

Comparison of Allantoin and Inulin Clearances for Measurement of *Filtration Rate in Diseased Human Kidney.* B. F. Miller, A. Leaf, A. R. Mamby and Z. Miller.—p. 314.

Some Aspects of Human Pharmacology of Tetraethylthiuramdisulphide (Antabus)-Alcohol Reactions. C. H. Hine, T. N. Burbridge, E. A. Macklin and others.—p. 317.

Reversal of Diurnal Variation in Renal Function in Cases of Cirrhosis *with Ascites.* R. A. Jones, G. O. McDonald and J. H. Last.—p. 326.

Studies in Glycine-2-C^{14} Metabolism in Man: II. Tissue Distribution. N. I. Berlin, B. M. Tolbert and C. Lotz.—p. 335.

Basal Secretion of Pepsin by Human Stomach. H. D. Janowitz and F. Hollander.—p 338.

Journal Lab. and Clinical Medicine, St. Louis

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Anemia of Renal Insufficiency as Induced by Bilateral Nephrectomy of Rabbit: With Emphasis on Its Hemolytic Nature. E. E. Muirhead, *F. Jones and A. Grollman.*—p. 505.

Diuresis Produced by Concentrated Dialyzed Human and Pregnant Mare Urine. Assay Procedure in Dog for Diuretic Substances. J. M. Little. —p. 518.

Effect of Repeated Phlebotomies in Hemochromatosis: Report of Three Cases. W. D. Davis Jr. and W. R. Arrowsmith.—p. 526.

Flocculation Tests; Contribution to Study of Their Mechanism. R. Armas-Cruz, G. Lobo-Parga and M. Madrid.—p. 533.

Serum Cholinesterase Activity in Liver Disease. J. D. Mann, W. I. Mandel, P. L. Eichman ond others.—p. 543.

Serum Phospholipid-Cholesterol Ratio as Test for Coronary Atherosclerosis. L. M. Morrison.—p. 550.

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Effect of Cortisone and ACTH in Leukemia. W. S. Adams, W. N. Valentine, S. H. Bassett and J. S. Lawrence.—p. 570.

Determination of Packed Cell Volume. F. S. Schlenker and J. Noll. —p. 582.

Determination of Trapped Plasma in Centrifuged Erythrocyte Volume of Normal Human Blood with Radioiodinated (I^{131}) Human Serum Albumin and Radiosodium (Na^{24}). O. N. Vazquez, K. Newerly, R. S. Yalow and S. A. Berson.—p. 595.

*Effect of ACTH, Cortisone and Operative Stress upon Blood Cholesterol Levels. L. H. Kyle, W. C. Hess and W. P. Walsh.—p. 605.

Studies in Serum Proteins: VI. Extension of Standard Biuret Method to Estimation of Total Protein in Urine. P. W. Foster, J. J. Rick and W. Q. Wolfson.—p. 618.

Drainage of Radioactive Silver Colloids by Lymphatics Following Intrapulmonary Administration in Dogs. P. F. Hahn, G. Rouser, H. Brummitt and others.—p. 624.

Effects of Mixed Exchange Resins on Anionic and Cationic Metabolism. E. W. McChesney.—p. 629.

Cross-Resistance to Antibiotics: Effect of Repeated Exposures of Bacteria to Streptomycin or Neomycin on Resistance to Both of These and to Six Other Antibiotics. T. H. Haight, C. Wilcox and M. Finland. —p. 637.

Histochemistry of Lipid Carbonyl Compounds. G. Gomori.—p. 649.

Effect of Corticotropin, Cortisone, and Operative Stress on Blood Cholesterol.—Kyle and associates followed the blood cholesterol levels of patients during administration of corticotropin and cortisone and following the stress of surgical intervention, and compared the observed changes with variations seen in normal persons. They found that the determination of plasma cholesterol and cholesterol esters by chromatographic separation is reproducible within 2% on repeated determinations on the same sample. The variation in plasma cholesterol content on the same patient within two to eight hours is likewise within 2%, and for periods of from two to eight days the variation is approximately 5%. The administration of corticotropin in nine patients caused an average reduction of total cholesterol of 24%, and the greater decrease in cholesterol was well correlated with greater physiological stimulation by corticotropin. In two patients who obtained little stimulation from the corticotropin there was no significant depression of cholesterol. Intravenous administration of corticotropin to one patient for 20 days and to another for 14 days produced a marked decrease in plasma cholesterol. The effect of the administration of cortisone to nine patients was less consistent than that of corticotropin. The average change was a decrease of 7%, but a more marked decrease was observed in two patients who were given larger doses of cortisone. Compound F was given to one patient for 20 days, producing only a slight decrease in plasma cholesterol, although a marked decrease in the eosinophil count occurred. In 9 of 10 cases there was a decrease in plasma cholesterol following surgery, the decrease occurring later than the reduction in eosinophil count. It is suggested that the decrease in blood cholesterol that accompanies administration of corticotropin is caused by direct action of the adrenal hormone, either by inducing temporary impairment of hepatic function, or, more probably, by suppressing cholesterol synthesis.

Journal Nat. Cancer Inst., Washington, D. C.

12:701-936 (Feb.) 1952. **Partial Index**

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Spontaneous Pulmonary Tumor in Rat: Report of Lesion. H. A. Horn and H. L. Stewart.—p. 743.

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Mortality of Mice After Total-Body Irradiation as Influenced by Alterations in Total Dose, Fractionation, and Periodicity of Treatment. H. S. Kaplan and M. B. Brown.—p. 765.

Acute Necrotizing Renal Papillitis Experimentally Produced in Rats Fed Mono-N-Methylaniline. J. White and P. Mori-Chavez.—p. 777.

Increase in Incidence of Leukemia in Hybrid Mice Bearing Thymic Transplants from High Leukemic Strain. L. W. Law.—p. 789.

Effect of Guanazolo on Antibody Formation. R. A. Malmgren, B. E. Bennison and T. W. McKinley.—p. 807.

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Effect of Arterial Occlusion on Epidermal Carcinogenesis in Rabbit. A. C. Ritchie.—p. 847.

Influence of Cortisone Acetate upon Growth of Transplanted Rhabdomyosarcoma in C3H Mice. G. M. Higgins and W. A. Bennett.—p. 851.

Ovarian Tumorigenesis Following Intrasplenic Transplantation of Ovaries from Weanling, Young Adult, and Senile Mice. M. Klein.—p. 877.

Proliferative Lesions Resembling Chordoma Following Puncture of Nucleus Pulposus in Rabbits. C. C. Congdon.—p. 893.

Acid and Alkaline Phosphatase of Serum and Hepatic Tissue of Rats Fed Carcinogens. A. S. Mulay and H. I. Firminger.—p. 917.

Metabolism of L-Histidine-2-C^{14} in Normal and Hepatoma-Bearing Rat. J. C. Reid, M. O. Landefeld and J. L. Simpson.—p. 929.

Journal of Nutrition, Philadelphia

46:141-270 (Feb.) 1952. **Partial Index**

Studies on Effects of Atabrine on Vitamin A Metabolism. K. Guggenheim. —p. 141.

Rancid Lard Effect on Rats Fed Complete and Riboflavin-Deficient Diets. H. Kaunitz, R. E. Johnson and C. A. Slanetz.—p. 151.

Study of Minimum Calcium Requirements of Adult Men. D. M. Hegsted, I. Moscoso and C. Collazos Ch.—p. 181.

Studies on Interrelationship Between Methionine and Vitamin B_6. H. J. DeBey, E. E. Snell and C. A. Baumann.—p. 203.

Retention of Crystalline Vitamin B_{12} by Healthy Male Individuals Following Intramuscular Injection. C. Lang, R. A. Harte, C. L. Conley and B. F. Chow.—p. 215.

Excretion of N^1-Methylnicotinamide and Creatinine as Influenced by Vitamin B_{12} and Various Methyl Donors. I. E. Liener and M. O. Schultze.—p. 223.

Military Surgeon, Washington, D. C.

110:249-322 (April) 1952

*Clinical Aspects of Cold Injury. F. H. Mowrey and P. J. Farago.—p. 249.
Intramedullary Fixation of Fractures in Combat Casualties. E. A. Brav.—p. 254.
Modern Conception of Veterinary Public Health. J. H. Steele.—p. 258.
Military Government and Public Health. J. A. Tobey.—p. 260.
Public Health Aspects of Leptospirosis. W. S. Gochenour Jr.—p. 265.
Management in Army Hospitals. R. O. Pennepacker.—p. 269.
Treatment of Dislocated Condylar Fractures. E. G. Friedrich.—p. 271.
Procedures and Technique in Registrar Division of Veterans Administration Hospitals. J. M. DiPietro.—p. 273.
Epidemic Hemorrhagic Fever of Far East, or Endemic Hemorrhagic Nephroso-Nephritis: Short Outline of Disease, with Supplemental Data on Results of Experimental Inoculation of Human Volunteers. C. F. Mayer.—p. 276.

Clinical Aspects of Cold Injury.—Cold injury is trauma produced by exposure to cold, and includes such entities as chilblains, immersion foot, trench foot, and frostbite. Of 593 patients at the average age of 22, who with the troops in Korea had been exposed to temperatures of −20 to −30 F and were evacuated to the United States for frostbite treatment, 474 (80%) had either superficial or deep gangrene. Eighty per cent of the patients had involvement of the feet only, 16.8% had involvement of both feet and hands, and 3.2% had involvement of the hands only. Clinical experience with these patients showed that it is almost impossible to determine accurately the amount of tissue damage or whether amputation will be required without prolonged observation, up to three months in some cases. Conservative therapy is indicated to insure maximum preservation of function. The necrotic eschar on digits should be bivalved early to prevent contraction and further tissue damage. Ointments, particularly white petrolatum U. S. P. (vaseline®) gauze, are contraindicated as they result in ulceration and infection. Hexamethonium was the most effective vasodilator, as shown by high and prolonged elevation of skin temperature. This drug is just as effective as lumbar blocks and much simpler. Priscoline® (2-benzyl-4,5-imidazoline) hydrochloride administered orally was the next most effective agent. The use of vasodilators made no clinically evident difference in the rate of healing, but may have lessened the incidence of "post frostbite syndrome," such as cold feet with dependent rubor and/or cyanosis and hyperhidrosis. Administration of corticotropin (ACTH) and cortisone resulted in mild euphoria, lessening of pain, decrease in purulent drainage and odor, and apparently earlier demarcation of the necrotic area. Neither drug lessened the amount of tissue loss nor resulted in more rapid healing of the lesions. Soaks in warm, sterile, isotonic sodium chloride solution softened the eschar and in some cases apparently promoted more rapid healing.

New England Journal of Medicine, Boston

246:523-560 (April 3) 1952

*Use of Vaginal Smear as Screening Test. H. L. Lombard, M. Middleton, S. Warren and O. Gates.—p. 523.
Deep Venous Thrombosis in Leg Following Effort or Strain. C. Crane.—p. 529.
Allergy to Viral and Rickettsial Vaccines: III. Influence of Repeated Inoculations on Acquisition of Egg Allergy. B. Ratner, S. Untracht and F. Hertzmark.—p. 533.
Successful Treatment of Neuroblastoma: Report of Two Cases with Apparent Cures. H. W. Hudson Jr., J. W. Chamberlain and F. B. Becker.—p. 536.
Some Practical Aspects of Cancer Prevention. E. L. Wynder.—p. 538.

The Vaginal Smear as a Screening Test.—A single vaginal smear was obtained from each woman who attended the state-aided cancer clinics of Massachusetts between Jan. 1, 1945, and June 30, 1947. Three years after the original smear a follow-up history and, whenever possible, a pelvic examination and a repeat smear were secured. The total number of records was 3,581. Of the 210 women in whom cancer was diagnosed at the time of the original smear, 62.4% showed a positive test, 23.8% a questionable test, and 13.8% a negative test. Thus, the tests did not indicate that cancer was present in nearly one-seventh of those who had cancer of the uterus. Of 1,888 persons who had no apparent cancer of the uterus at the time of the smear or three years later, 1.9% originally showed a positive smear, 5.9% a questionable smear, and 93.1% a negative one. It is true that a few unsuspected cases of cancer have been discovered through the use of the vaginal smear, but the cost per discovered case was approximately $1,700. The large error found among the group most likely to have cancer (those who bleed) impairs the diagnostic value of this test for screening purposes. It does not seem feasible for a state health department to offer this test on an extensive scale for women without gynecologic symptoms, as the cost would be prohibitive and relatively few cancers would be found. The authors reiterate that they are not concerned with the value of the vaginal smear as a means of diagnosis per se, but rather with its use as a method of screening large populations. Its usefulness as a screening procedure is obviously less than in the study of an individual case.

New Jersey Medical Society Journal, Trenton

49:87-124 (March) 1952

The General Practitioner Treats the Uncomplicated Diabetic. E. O. Bauman.—p. 91.
Fate of Casein Hydrolysate Amino Acids Administered Intravenously to Human Subjects. R. N. Cagan, R. L. Klein and L. Loewe.—p. 99.
Hypoplastic Uterus in Infertility. M. Morgenstern.—p. 102.
Clinical Results with Prantal Methylsulfate: New Parasympathetic Blocking Agent: Preliminary Report. W. F. Vogel.—p. 105.

49:125-166 (April) 1952

The Cancer Program in New Jersey, Part VI: Administration of Radio-Active Isotope Program in New Jersey. W. O. Wuester, W. E. Bray, J. S. Gallo and others.—p. 129.
Functional Disease of Colon: Roentgen Study Based on 200 Cases. R. J. Gross, O. H. Cohen and C. R. Weinberg.—p. 131.
Teen-Age Narcotic Problem in New Jersey. E. P. Ivey and J. L. Evans Jr.—p. 136.
Trichlorethylene in Obstetrical Anesthesia. M. Fenster and H. Levy.—p. 138.
Clinical Diagnosis and Case Management. G. V. Stoddard.—p. 139.
Acute Methemoglobinemia Due to Aniline: Report of Case. A. D. Casciano.—p. 141.
Rehabilitation of Aged Chronic Invalid. J. O. Smigel.—p. 143.
Brain Anoxia: Report of Case. F. Frisch.—p. 146.
Method of Psychotherapy for General Practitioner. J. Chernus.—p. 148.
Scabies Epidemic in an Institution. H. C. Goldberg.—p. 152.

Western J. Surg., Obst. & Gynecology, Portland, Ore.

60:93-140 (March) 1952

Simplified Putti-Platt Reconstruction for Recurrent Shoulder Dislocation: Preliminary Report. E. A. Brav and V. H. Jeffress.—p. 93.
Diabetes and Pregnancy. J. M. Whitely and T. W. Adams.—p. 98.
Neurilemoma Involving Main Peripheral Nerve Trunk with Complete Excision and Preservation of Nerve Continuity and Function: Report of Three Cases. J. P. Trommald.—p. 108.
Some Considerations Regarding Use of Vascular Grafts. L. R. Sauvage and H. N. Harkins.—p. 113.
Vaginismus as Cause of Dyspareunia: Report of Cases and Method of Treatment. S. P. Hall.—p. 117.
Anal Ileostomy in Chronic Ulcerative Colitis. W. B. Hutchinson.—p. 121
*The Pancreas and the Steering Wheel. M. Joseph.—p. 129.
Carcinoma of Cervix Complicating Pregnancy. A. P. Hudgins.—p. 132

Injuries to the Pancreas by Steering Wheels.—Having observe
three cases of subcutaneous nonpenetrating trauma to the pa
creas within a period of a few months, the author suggests th
pancreatic trauma is not as rare as is generally believed, a
therefore the notion that the pancreas is invulnerable because
its cushion protection by the stomach and transverse co
should be discarded. When a ruptured viscus is suspected af
severe abdominal trauma, and no injury of the liver, spleen,
the hollow viscera is found, the pancreas should always be ca
fully examined. Serum amylase and lipase should be determi
in all cases of vague epigastric symptoms in which results
careful routine tests, including x-ray examination, are negat
Surgery should be directed toward the control of hemorrha
relief of excessive tension in the capsule of the pancreas,
removal of septic material and fluid. The value of drainag
uncertain. All three patients had had automobile accident
which the steering wheel had been forced against the abdo
and chest. The possibility of a pancreatic trauma should be
sidered whenever a blow to the upper abdomen has been
tained. Early exploration and repair of damage are impor

FOREIGN

Acta Paediatrica, Stockholm

41:101-206 (March) 1952. **Partial Index**

*Simultaneous Visualization of Posterior and Anterior Mediastinum After Insufflation in Peridural Space: Tridimensional Tomographic Study. G. Sansone and A. De Maestri.—p. 101.
Weight of Thyroid Gland in Infants up to 2 Years of Age. H. Tähkä. —p. 108.
Airborne Infections: VI. Continued Investigations into Control of Dust-Borne Streptococcal Infections in Infants' Wards. G. Laurell.—p. 124.
Iron Metabolism in Infants: I. Intake of Iron in Breast Feeding and Artificial Feeding (Milk and Milk Foods). Y. M. Feuillen and M. Plumier.—p. 138.
ACTH in Treatment of Juvenile Rheumatoid Arthritis. J. Vesterdal, O. Remvig and M. Sprechler.—p. 145.
Report of Two Cases of Acrodynia Appearing After Calomel Therapy but Without Excretion of Mercury in Urine and Cured with Dimercaprol (BAL). R. Houet.—p. 158.
Lipochondrodystrophy with Cutaneous Deposits. B. Andersson and O. Tandberg.—p. 162.
Sexual Precocity in Male Infant Due to Estrogenic Ointment. L. Hesselvik.—p. 177.

Visualization of Mediastinum.—Study of the posterior mediastinum is difficult, particularly in children, in whom gas insufflation following transtracheal puncture has been considered inadvisable. The authors found the intraspinal, extradural approach, that is, injection of a gas contrast medium into the peridural space, ideal for use in children. This technique permits simultaneous, sharp visualization of both the anterior and posterior mediastinum. It is easy, safe, and well tolerated by children. Used with tomography (in anteroposterior, lateral, and axial transversal projections) this method gives interesting tridimensional pictures.

British Journal of Dermatology, London

64:129-168 (April) 1952

Bloch-Sulzberger Syndrome (Incontinentia Pigmenti). H. Haber.—p. 129.
Pathogenesis of Incontinentia Pigmenti. With Observations on Associated Eye Disturbance Resembling Retrolental Fibroplasia. G. H. Findlay. —p. 141.
Vascular Naevi and Their Treatment. R. T. Brain and C. D. Calnan. —p. 147.

British Medical Journal, London

1:831-880 (April 19) 1952

Fifth and Seventh Cranial Nerves in Relation to Nervous Mechanism of Taste Sensation: New Approach. W. Harris.—p. 831.
*Mass B.C.G. Vaccination in Malta: Report on B.C.G. Vaccination Campaign Held by I.T.C. in Malta in 1950. V. Zammit-Tabona. —p. 837.
Gall-Stones: Clinical Survey. T. R. Littler and G. R. Ellis.—p. 842.
Pulmonary Tuberculosis and Pregnancy. J. R. Edge.—p. 845.
Value of Pneumoencephalography in Diagnosis of Fits. J. Marshall and C. W. M. Whitty.—p. 847.
Isolated Neuritis of Anterior Interosseous Nerve. L. G. Kiloh and S. Nevin.—p. 850.
Verbal Usage of the CDE Notation for Rh Blood Groups. S. Haberman and J. M. Hill.—p. 851.
Response of Megaloblastic Anaemias of Pregnancy to Animal Protein Factor: Preliminary Report. H. Foy, A. Kondi and A. Hargreaves. —p. 852.

Mass BCG Vaccination in Malta.—Between March and June, 1950, 54,328 persons in Malta were tuberculin-tested, and 38,681 were vaccinated with BCG. Of the latter 36,320 were between the ages of 1 and 18 years and 2,361 were over 18. This campaign was held because destruction of habitable dwellings during World War II and the ensuing housing shortage had resulted in severe overcrowding, favoring spread of tuberculosis. The tuberculin tests indicated that between the ages of 16 and 20 more boys than girls were infected with tubercle bacilli. This is in accord with the finding that in this age group more males developed pulmonary tuberculosis, in whom, however, it was less likely to be fatal than in females. The incidence of tuberculosis infection and of pulmonary tuberculosis were lower in the rural areas, while in the semirural, suburban, and urban areas they were approximately the same. BCG vaccination was carried out by means of Wallgren's intradermal injection technique, using 0.1 ml. of a vaccine containing 0.025 mg. of BCG or about 1 million "BCG bacilli" per 0.1 ml. An accelerated reaction at the site of inoculation was observed in 11 persons. Between the 30th and 53d days after vaccination 23 patients had discharging ulcers, which tended to heal rapidly. Of 54 persons with lymph node enlargement in the axilla following BCG vaccination, 2 had a secondarily infected BCG ulcer, and 2 complained of discomfort on movement, while in 21 children between the ages of 1 and 6 years the condition progressed to suppuration. Elevation of temperature following the vaccination was rare, one person had erythema nodosum, and 17 had phlyctenular conjunctivitis. To check the efficacy of both the vaccine and the technique of its administration, postvaccination tuberculin tests were given to 2,421 unselected persons according to the epinephrine-Pirquet technique. Positive reactions occurred in 98.95% of these persons. Up to 12 to 15 months after the campaign no cases of tuberculosis had been reported among persons vaccinated with BCG.

Edinburgh Medical Journal

59:161-212 (April) 1952

*BCG-Vaccination as Public Health Measure. A. Wallgren.—p. 161.
Determination of Bacterial Sensitivity to Antibiotics. J. C. Gould and J. H. Bowie.—p. 178.
Staph. Aureus Air Infection in Maternity Hospital. A. T. Wallace and J. P. Duguid.—p. 200.

BCG Vaccination as Public Health Measure.—Experience with BCG vaccination in about 17,000,000 persons in the Scandinavian countries, in the war-devastated countries in Europe, and in undeveloped countries outside Europe suggests that adequately performed BCG vaccination produces specific immunity strong enough to provide protection to a great extent against severe tuberculosis, miliary tuberculosis, and tuberculosis meningitis, and to a certain extent against pleurisy and postprimary pulmonary tuberculosis. The morbidity and mortality rates of tuberculosis have been reported to be lower in groups of adequately vaccinated persons than in nonvaccinated groups of tuberculin-negative persons. Most observers believe that BCG vaccination should be accepted as a public health measure against tuberculosis. BCG vaccination in itself will not abolish tuberculosis in a community. Other measures are more important, such as those aimed at preventing tuberculous infection, at increasing the natural resistance of the population by a high standard of living, at early detection of tuberculous disease, and at giving every person in need of it adequate treatment. The object of BCG vaccination is to produce artificial specific immunity in persons who still are uninfected, and the earlier artificial immunity is established the better. In a country with high incidence of tuberculosis vaccination of children as young as possible is advisable as a public health measure. If most babies are born in maternity hospitals, the BCG vaccination is most conveniently performed on newborn infants, without any preliminary tuberculin testing. By tuberculin control and revaccination if necessary, the virulent infection, when and if it occurs, is generally prevented from producing clinical primary tuberculosis and early postprimary disease. In persons of all age groups exposed to sources of infection, such as children in homes with tuberculous patients and hospital personnel, BCG vaccination should as a public health measure be performed in those still tuberculin-negative.

J. of Canadian Association of Radiologists, Montreal

3:1-30 (March) 1952

Physical Characteristics of Radiation in Cobalt^{60} Beam Therapy. H. E. Johns.—p. 2.
Clinical Possibilities of Cobalt^{60} Beam Unit. T. A. Watson.—p. 7.
Room Protection Measurements for Cobalt^{60} Teletherapy Units. W. R. Dixon, C. Garrett and A. Morrison.—p. 11.
Cobalt^{60} Beam Therapy: Some Influences and Advantages. I. H. Smith. —p. 16.
Considerations in Design of Cobalt^{60} Beam Therapy Equipmnt. D. T. Green and R. F. Errington.—p. 20.
*Betatron in Cancer Therapy: Part II. T. A. Watson and C. C. Burkell. —p. 25.

Betatron in Cancer Therapy.—Observations on 58 patients with advanced cancer treated by means of a 22 *mev* betatron are reviewed. A 22 mev betatron has limited usefulness in treatment of deep-seated lesions. It does not appear to have specific biological advantage over conventional x-rays, but does have certain physical attributes that can be used to advantage. Constitutional effects are minimal. Lack of variation of depth dose with field size makes for simplicity. The low skin dose compared with a very high depth dose is of great value. The immediate results, in advanced cases of cancer of the cervix and bladder, were promising, but the doses given apparently caused disappearance of detectable signs of the tumor rather than eradication. It has been impossible to achieve a dose lethal for the tumor in most cases. The limiting factor in treatment of cancer of the cervix is the effect on the bowel and bladder, but even irradiation to the pelvis is possible. A dose of 6,000 r to the whole pelvis delivered in three weeks was considered the maximum possible. Considerable palliation was achieved in advanced tumors of the lung and brain. The response of cancer of the rectum was disappointing.

Lancet, London

1:775-830 (April 19) 1952

Medical Research Council National Tuberculin Survey 1949-50: Background and Methods of Survey. R. T. James, E. C. H. Huddy, J. E. Geddes and others.—p. 775.
•*Thymectomy in Treatment of Myasthenia Gravis.* R. T. Ross.—p. 785.
Review of 200 Chronic Alcoholics. L. Williams.—p. 787.
Assay of A.C.T.H. on Thymus of Nestling Rat. H. M. Bruce, A. S. Parkes and W. L. M. Perry.—p. 790.
•*Prevention of Acute Dinitro-Ortho-Cresol (D.N.O.C.) Poisoning.* P. L. Bidstrup, J. A. L. Bonnell and D. G. Harvey.—p. 794.
Estimation of Dinitro-Ortho-Cresol in Blood. D. G. Harvey.—p. 796.
Diabetic Acidosis Without Ketonuria. J. Henneman.—p. 797.
Extensive Resection of Small Bowel. J. H. Johnston.—p. 799.

Thymectomy in Myasthenia Gravis.—A benign hyperplastic thymus gland was removed from 100 consecutive patients with myasthenia gravis. Only one of the seven patients who died postoperatively died of myasthenia gravis. Of the survivors, 41 patients have no symptoms and require no treatment; 26 have slight and inconstant symptoms and are able to work with no limitations due to myasthenia gravis, with irregular and much less treatment than before the operation; 20 patients have fewer or less severe signs and symptoms, and respond to a significantly smaller dose of neostigmine as well or better than before the operation; and 6 patients are unchanged or worse. The benefit was greatest in the patients who had had myasthenia for the shortest time before operation. The age of onset did not seem to be significant. Comparison with three series of patients treated by medical means alone reveals that among these the proportion of cured or greatly improved is smaller and the proportion of deaths due to myasthenia gravis much higher than in the group presented here.

Prevention of Acute Dinitro-Ortho-Cresol Poisoning.—In Great Britain eight fatal cases of poisoning have occurred since 1945 in men engaged in the manufacture of dinitro-ortho-cresol, and in spray operators using dinitro-ortho-cresol for control of weeds in cereal crops. No cases of poisoning have been recorded among men who use the substance as a late winter wash on fruit trees. Experiments on volunteers showed that, when the dinitro-ortho-cresol level in the blood was about 20 μg per gram a further dose of about 1 mg. per kilogram of body weight led to a sharp increase in the concentration of this compound in the blood, and when this was more than 40 μg per gram all the volunteers had headache, lassitude, and general malaise. When the level of this substance in the blood was about 20 μg, all the volunteers had an exaggerated feeling of well-being. Single estimations were made on blood collected from 23 workers engaged in manufacture of dinitro-ortho-cresol, 39 men using dinitro-ortho-cresol as a late winter wash on fruit trees, and 133 spray operators applying dinitro-ortho-cresol to cereal crops. The dinitro-ortho-cresol levels in four cases of acute poisoning show the correlation between the blood level and the symptoms and signs of poisoning. A man should be removed from further contact with this compound for a minimum of six weeks if the dinitro-ortho-cresol level eight hours after the last exposure is 20 μg per gram or higher. Since the main route of absorption is by inhalation, respirators should be worn, especially by spray operators. The authors consider that estimation of dinitro-ortho-cresol levels in the blood might prove a valuable measure in the prevention of acute poisoning, but that determination of dinitro-ortho-cresol in the urine is unlikely to prove a reliable test.

Medical Journal of Australia, Sydney

1:385-424 (March 22) 1952

*Surgical Relief of Mitral Stenosis: Preliminary Report. F. H. Mills, J. K. Maddox, R. B. Blacket and A. J. Palmer.—p. 385.
Toxoplasmosis: Review of Literature and Report of Case. I. B. Jack. —p. 391.
Laryngeal Ventricle and Its Appendix (Laryngeal Saccule) and Their Associated Pathological Lesions, with Report of Three Cases of Laryngocele. G. Swinburne.—p. 396.
Secretion of 3,3'-Ethylidene Bis (4-Hydroxycoumarin) in Milk. P. Fantl and J. F. Nelson.—p. 404.
Renal Trauma. A. Gee.—p. 405.
The Q-T Interval of Electrocardiogram in Elderly Subjects. P. Hagen. —p. 407.
Flexible Plastic Tube Adapter for High-Pressure Injections. D. G. Renton.—p. 408.

Surgical Relief of Mitral Stenosis.—Commissurotomy according to the techniques of Bailey and Brock was performed by a posterolateral approach through the bed of the left fifth rib in 23 selected patients with mitral stenosis. The first patient was treated in March, 1951. Separation of the valve cusps at the commissures with the finger was attempted. If this was not possible, the finger was withdrawn and a small commissurotomy knife fashioned to fit the curve of the finger was inserted. With the knife it was usually easy to slit the valve and then carry out digital dilatation to the muscular wall of the heart. One patient with severe stenosis died during the induction of anesthesia, although adequately prepared for surgical intervention, and a second patient with auricular fibrillation of several years' standing died of embolism in the cerebral and iliac vessels on the third postoperative day. Patients with a very large heart, with established right-sided failure, with free mitral regurgitation, with significant aortic valve disease, or with active rheumatic carditis should not be operated on. In patients between 30 and 45 years of age who are not in a state of congestive failure, who have not had severe congestion, but who have had recurrent "bronchitis," pulmonary edema or hemoptysis and are seriously incapacitated, the operative mortality should be negligible and the results should be good. Two of the 21 living patients were not yet well enough for their improvement to be assessed, but 14 showed great improvement, 2 were only slightly improved, and 3, in whom for technical reasons dilatation was unsuccessful, were unimproved. The only serious postoperative complication was auricular fibrillation, which was controlled with digitalis and quinidine. The severity of the lesion was demonstrated preoperatively by cardiac catheterization in four of the first six patients operated on; the authors now carry out this procedure as a routine measure before considering surgical treatment. The authors' results and those of other workers suggest that sooner or later every patient with mitral stenosis should be treated surgically.

Nederlandsch Tijdschrift v. Geneeskunde, Amsterdam

96:661-722 (March 22) 1952. **Partial Index**

Periarteritis Nodosa Cured with Corticotropin. A. J. M. Lohman. —p. 671.
Combat of Bovine Tuberculosis and Change in Epidemiologic Picture of Tuberculosis. A. C. Ruys.—p. 676.
New Methods for Concentration of Ameba Cysts in Feces. J. J. Laarman. —p. 681.
*Persistent Excretion of Tubercle Bacilli by Patients with Clinically Inactive Pulmonary Lesions. C. Dijkstra.—p. 690.

Excretion of Tubercle Bacilli by Patients with Inactive Pulmonary Lesions.—Frequent control tests were carried out over long periods on the sputum and gastric contents of 445 patients who had had an open form of pulmonary tuberculosis but had been clinically cured. It was found that 18% continued to eliminate tubercle bacilli intermittently. In about 15% of the patients the last 10 specimens examined for tubercle bacilli were negative, but since prolonged intermission in excretion of bacilli of from four to six months had occurred before, it is not certain whether the lesions in these patients are definitely closed.

Presse Médicale, Paris

60:489-508 (April 5) 1952. **Partial Index**

Importance of Auscultation of Thyroid in Diagnosis of Basedow's Disease. C. Lian.—p. 489.
*Anti-Edematous Therapy Combined with Palliative Chemotherapy of Epithelial Cancers. A. Morel and A. Josserand.—p. 490.
Pathology and Treatment of Abortions of Endocrine Origin. M.-F. Jayle. —p. 492.

Anti-Edema Therapy in Epithelial Cancers.—The frequent early, severe relapses following incomplete cytocidal therapy in cancer may result from perifocal swellings caused by an allergic reaction to the products of partial tumor lysis. Neoplastic cells proliferate in the edematous fluid and the exudates, carried by greatly dilated lymphatic vessels, cause distant, so-called inflammatory, adenopathies; these in turn are colonized by the cancer cells. Excellent palliative results with apparent stabilization of the tumors have been obtained by treatment in three stages: a cytocidal attack; brief, intense therapy to reduce edema; and a fairly long period of antihistaminic therapy designed to prevent reinfiltration of the perifocal area. Treatment begins with the intravenous administration of sodium oxyferriscorbone in 2 or 3 cc. of 30% hypertonic glucose solution morning and evening for five days to prepare the perifocal area for the cytocidal attack. No treatment is given on the sixth day, but on the seventh colchicine is given intravenously followed by from four to six additional injections of sodium oxyferriscorbone. Twenty days of antihistaminic therapy follow, including intradermal injection of an antihistamine solution every other day, to prevent the reappearance of edema. The complete series is then repeated as often as necessary. The treatment is generally well tolerated and may be individualized by such means as altering the dose or using other cytocidal preparations alternately with colchicine. The pain of epithelial cancer, which is usually caused by pressure of the fine nerve fibers owing to perineoplastic cellulitis, diminishes or disappears entirely after reduction of edema, and the effect of the cancerocidal substances is prolonged and accentuated by the decongestive treatment.

Riforma Medica, Naples

66:197-224 (Feb. 23) 1952

*Q Fever in Umbria Region: Clinical Observations in 51 Cases. R. Frascarelli.—p. 197.
Anti-Allergy Treatment in Severe Shock Caused by Burns. A. Loizzi. —p. 202.
Danger and Harm of Treating Ureteral Lithiasis with Oligo-Mineral Waters. M. Sorrentino.—p. 205.
Use of Bogomoletz Serum. G. Carnera.—p. 207.

Q Fever in Umbria Region.—The occurrence of Q fever is reported in 51 persons from the Umbria region. The incubation periods ranged from 14 days to 26 days. The symptoms were elevation of temperature, severe headaches, profuse sweating, asthenia, and pulmonary involvement. Laboratory tests revealed an increased erythrocyte sedimentation rate during the acute stage of the disease, slight but frequent albuminuria, and in seven patients decreased reaction to tuberculin. The blood picture and the differential blood count remained unchanged. The liver was enlarged in four patients only, of whom two were alcoholics and one both alcoholic and diabetic. Since the urine urobilin level was normal, there was no bilirubin in the blood, and the Takata-Ara test was normal, the author concludes that usually Rickettsia burneti cause slight liver damage. Repeated tests demonstrated that the complement fixation was always positive and that the complement-fixing antibodies had not been influenced by antibiotic treatment and were still in circulation several months later. All these patients, except one, had a bronchopneumonic process, but in none was Q fever associated with another disease. According to the author, Q fever occurs both with and without pulmonary involvement. Diagnosis based on clinical findings is inadequate and laboratory tests for Rickettsia burneti are the decisive factor in differential diagnosis from primary atypical pneumonia and other febrile processes. Treatment of Q fever is not difficult, since the disease may regress spontaneously. Fifteen patients were treated with aureomycin, 13 with terramycin, 7 with chloramphenicol (chloromycetin®), and 1 with paraaminobenzoic acid. Aureomycin and terramycin were equally effective, while chloramphenicol failed in two patients. In all the other patients the disease disappeared spontaneously, suggesting that the effects of antibiotics on Q fever should not be overemphasized.

Schweizerische medizinische Wochenschrift, Basal

82:372-440 (April 12) 1952. **Partial Index**

Incidence of Sensitization by Mercury as Cause of Infantile Acrodynia. T. Baumann.—p. 375.
Accidental Death in Childhood, Particularly in Motorized Street Traffic. J. Dettling.—p. 377.
*Etiology of Retrolental Fibroplasia. H. Goldmann and W. Tobler. —p. 381.
Role of Infection in Development of Atrophic Conditions in Infants. P. Kiss.—p. 387.
Localization of Lobar Pneumonia in Childhood. S. Engel.—p. 403.
Examination of Functional Capacity of Adrenal Cortex in Early Childhood. E. Rominger.—p. 411.
*Treatment of Celiac Disease with Corticotropin and Cortisone. J. Ugland, L. Wennevold and L. Salomonsen.—p. 420.

Etiology of Retrolental Fibroplasia.—A single case of retrolental fibroplasia was discovered among 570 prematurely born infants who weighed less than 2,000 gm. at birth. A search for etiological factors revealed that the recent introduction of a new type of incubator and the use of high concentrations of oxygen probably played a part in development of retrolental fibroplasia. They suggest two possible causes: (1) the unphysiological administration of oxygen may cause the eye lesions or (2) although the prematurely born infant may become adapted to the oversupply of oxygen, damage occurs when the baby is removed from the high oxygen atmosphere. The authors feel that oxygen must be used cautiously in prematurely born infants.

Corticotropin and Cortisone in Celiac Disease.—The four children with celiac disease whose cases are reported in this paper were boys between 15 months and 5½ years of age. They had had symptoms of celiac disease for from six months to three years. The response to dietetic treatment had been unsatisfactory. Treatment with corticotropin and cortisone was continued for from four to seven weeks, during which a modified dietetic treatment was continued. Corticotropin was given for two days in large doses, then for several weeks in an adjusted maintenance dose and finally in slowly decreasing doses. Patients who were treated with cortisone were given some corticotropin at the end of the course of cortisone. Treatment with corticotropin or cortisone effected improvement in the general condition, mood, appetite, appearance and fat content of stools, sideropenia, anemia, hypoproteinemia, and the blood sugar tolerance tests, but no effect was noted on osteoporosis, on the retarded development of the epiphyseal nucleus, or on the roentgenologically demonstrable changes in the small intestine.

Semaine des Hôpitaux de Paris

28:1085-1118 (April 6) 1952

Cortisone in Cirrhosis of Liver in Alcoholics. R. Cachera and F. Darnis. —p. 1085.
*Biochemical Diagnosis of Renal Lithiasis. J. Cottet.—p. 1096.

Biochemical Diagnosis of Renal Lithiasis.—Since dietetic and medicinal treatment of renal lithiasis varies according to the chemical nature of the lithiasis, biochemical diagnosis of the lithiasis was attempted by the author on the basis of a test in which crystalluria is induced. The crystalline sediment and the pH were studied systematically in the urine specimens collected in the course of one day while the specific gravity of these specimens varied physiologically. For the induced crystalluria test to be of any value, the specific gravity must exceed 1.015 in at least two or three specimens of the urine. The test was performed on 24 persons who had no signs of renal lithiasis, on 18 patients with renal colic but without evidence of calculi, and on 56 patients who had urinary calculi. Results of the tests suggested that it may be possible to infer the chemical nature of the lithiasis from the constitution of the urinary crystalline sediment. This concept was confirmed by a comparative study of the results of the induced crystalluria tests, which were done in 28 patients with one or several urinary calculi, the chemical constitution of which was known. The presence of a urate sediment indicates the formation of calculi of uric acid or oxalic acid. The presence of an oxalate sediment does not provide precise indications but is suggestive of the formation of calcium oxalate or phosphate calculi. The presence of uric acid in a mixed sediment rules out phosphate lithiasis, and the presence of phosphates in a mixed sediment rules out uric acid lithiasis.

Strahlentherapie, Munich

86:489-640 (No. 4) 1952. **Partial Index**

Results of Irradiation Therapy of Carcinoma of Cervix at the University Women's Clinic in Munich. H. Eymer and J. Ries.—p. 535.
Treatment of Malignant Melanoma. W. Hellriegel.—p. 548.
Myasthenia Gravis Pseudoparalytica as Disturbance of the Sympathetic System and Its Modification with the Aid of Functional Irradiation Therapy. J. Trautmann and J. G. Frey.—p. 571.
New Experimental Results with Thorium X on Human Tissues: Diagnosis by Means of Radioactive Isotopes. H. Moser and E. Schoen.—p. 539.
*Spontaneous Fractures After Roentgen Irradiation. W. Greve.—p. 617.

Spontaneous Fractures After Roentgen Irradiation.—Most reports of spontaneous fractures following roentgen irradiation concerned the neck of the femur. This paper reports one case of bilateral fracture of the neck of the femur in a man, aged 79, in whom 5,500 r had been applied to a tumor of the penis and prophylactic doses of 1,800 r to each inguinal field. The fractures of the neck of the femur were accompanied by bone atrophy, but there were no signs of bone metastases. Whereas the bone tolerance dose is usually estimated at between 3,800 and 4,300 r, in this patient it was estimated that the neck of the femur had received a focal dose of only about 1,500 r. In the three other cases reported in this paper, rib fractures followed intensive postoperative irradiation after mastectomy for cancer. There was no callus formation in these cases; on the contrary, the irregular fracture cleft became enlarged, in some cases up to 3 cm., owing to partial decomposition of the bone. Simultaneously, fibrosis of the corresponding lung field developed in these cases. In these patients with spontaneous rib fractures the bone tolerance dose had been exceeded.

Tubercle London

33:97-130 (April) 1952. **Partial Index**

Home Treatment of Respiratory Tuberculosis. H. Stevens.—p. 107.
Some Aspects of Composite Area Surveys in Mass Radiography. E. Psoner.—p. 112.
Effect of Penicillin on Mycobacteria in Vitro and in Vivo. M. A. Soltys. —p. 120.

West African Medical Journal, Nigeria

1:1-42 (Jan.-Feb.-March) 1952. **Partial Index**

Recent Advances in Tropical Medicine. Blackwater Fever in West Africa. B. Maegraith.—p. 4.
Clinical Picture and Treatment of Whipworm Infection. R. C. Jung and D. B. Jelliffe.—p. 11.
Oedematous Beri-Beri in Gambian Palm Wine Tappers. J. H. Walter and D. A. Smith.—p. 21.
*Cerebral Thrombosis in Sickle Cell Anaemia. D. B. Jelliffe.—p. 38.

Cerebral Thrombosis in Sickle Cell Anemia.—Sickle cell anemia is characterized by a chronic or recurrent hemolytic process and a tendency toward formation of intravascular thromboses. American authors who reported the occurrence of cerebral thrombosis in sickle cell anemia concluded that almost any neurological syndrome may be produced, including coma, hemiplegia, aphasia, and cranial nerve lesions, and they emphasized the importance of examining the blood for sickling in all cases of neurological disease in Negro patients. In this paper Jelliffe describes a girl, aged 4, who had shown signs of general ill health and had become drowsy the day before hospitalization. Despite intramuscular treatment with quinine and procaine penicillin, the child rapidly became more comatose and died. A diagnosis of sickle cell anemia was made, in view of the blood count, slight icterus, rapid development of in vitro sickling, and the presence of in vivo sickling. An associated intracerebral thrombosis suggested by the patient's condition was confirmed at autopsy. Clubbing of the fingers and toes and parietal bossing of the skull have been described as occurring in sickle cell anemia. The radiological appearance of the bosses reveals a characteristic perpendicular spiculated arrangement. The sickle cells seen in this case tended to have a long filamentous shape. This is in agreement with the findings of Foy, Kondi and Brass, who feel that this type of sickle cell occurs only in cases of sickle cell anemia, in contrast to the more compact, regular, half-moon cells seen in carriers of the sickle cell trait. This observation, which requires confirmation, would provide a rapid method of differentiating the two manifestations of sickle cell disease.

Ztschr. f. Hygiene & Infektionskrankh., Heidelberg

134:123-218 (No. 2) 1952. **Partial Index**

Further Tests on Sterility and Stability of Vaccine Lymph Prepared with Streptomycin. A. Herrlich and M. Bednara.—p. 123.
*Pylorospasm: Prophylaxis, Pathogenesis, and Therapy. G. Jordan.—p. 129.
Antibiotics of Animal Origin. M. Pavan.—p. 136.
Action of Cobalt on Antibody Titer of Blood: Experiments on Rabbits. G. Gillissen.—p. 173.
Coxsackie Viruses: With Consideration of Their Relationship to Virus of Poliomyelitis. F. H. Top.—p. 183.
New Neurotropic Viruses Found in Africa. K. C. Smithburn.—p. 212.

Pylorospasm.—The literature on pylorospasm reveals that it occurs almost exclusively in northern latitudes, 10 to 15 times more frequently in boys than in girls, and oftener in breast-fed infants. These and other aspects suggest a spastic genesis produced by insufficient calcium ions, which results in a tendency for tetany to develop. It has been observed that shortly after delivery the calcium content of the serum of many infants is decreased, presumably as the result of deficiency of vitamin D in mother and child. The sex ratio of pylorospasm is similar to that of rickets. Inter-relations between vitamin D and hypophyseal, gonadal, and adrenal hormones also point to disturbances in the calcium metabolism: the parathyroids are larger in females than in males, and the sex hormones influence calcium metabolism. Pylorospasm occurs most frequently during the third week of life when the serum calcium content is the lowest. Pylorospasm is thought to be the result of latent tetany in which vitamin D deficiency, nutritional and endocrine factors are involved. This concept of pylorospasm implies that calcium and vitamin D should be given prophylactically to the expectant mother, particularly near delivery. During the first four weeks the infant should be nursed often, but the amounts given at each feeding should be small. Blood for transfusions should contain heparin but should never be citrated. Infusion of Tyrode's solution and calcium intravenously is recommended. Acid therapy in the form of ammonium chloride and calcium chloride should be accompanied by control of the alkali reserve. If medical therapy fails, surgical treatment in the form of Ramstedt's operation should be resorted to.

Zeitschrift für Urologie, Leipzig

45:193-256 (No. 4) 1952. **Partial Index**

Embryonal Mixed Tumors of Kidney in Adults. H. G. Gleichmann. —p. 193.
Stones in Diverticuli of Bladder. L. Pfau.—p. 206.
Problem of Primary Cortical Focus in Renal Tuberculosis. H. Eufinger. —p. 213.
Recanalization of Mechanically Closed Ureters. W. Dornes, H. Lurz and B. Sachsse.—p. 229.
*Rare Metastases of Prostatic Carcinoma in Testis, Epididymis and Spermatic Cord. S. Rummelhardt.—p. 231.

Metastases of Carcinoma of Prostate in Testes, Epididymis, and Spermatic Cord.—Reports of metastases of the prostatic carcinoma to the testes have been rare. Four such cases have been reported in the American literature, and Eufinger describes an additional case. In all of these cases the metastasis was discovered during microscopic study of the surgical specimen. Later, in the course of 14 orchidectomies a change indicative of chronic inflammation was observed in one epididymis of one patient and the testis was removed on this side. Microscopic examination of the surgical specimen revealed cancerous infiltration in the spermatic cord and in the epididymis. Such mestatization from the prostate to spermatic cord, epididymis, and testis may take place by the blood or lymph streams or by canaliculi. When orchiectomy is done, the epididymis and spermatic cord should be carefully examined, so that easily removed metastases will not be left behind. Since the testicular metastases were observed neither clinically nor during operation, microscopic examination of the testicular parenchyma should not be neglected.

BOOK REVIEWS

The Prevention of Rheumatic Fever. By Lowell A. Rantz, M.D., Associate Professor of Medicine, Stanford University School of Medicine, San Francisco. Publication number 125, American Lecture Series, monograph in American Lectures in Circulation, edited by Irvine H. Page, M.D., and A. C. Corcoran, M.D. Cloth. $2.25. Pp. 66, with 1 illustration. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto 2B, 1952.

This brief monograph attempts to bring together the information that has accumulated in recent years regarding the role of Streptococcus in rheumatic fever. The author is primarily concerned with presenting evidence indicating the importance of prevention and early treatment of infection by the Streptococcus in the prophylaxis of rheumatic fever. On the basis of such evidence, specific recommendations for the prevention of rheumatic fever are discussed.

This monograph should be of great value to the pediatrician and internist interested in the problems of rheumatic fever. The brevity and simplicity of presentation should increase its usefulness for the student and house officer. With the exception of the small number of persons who still question the role of Streptococcus in rheumatic fever, there is little in the main theme of the subject matter that is controversial. Questions might be raised concerning the specific prophylactic procedures recommended, although most of them seem reasonable; however, the validity of the repeated injunctions against the use of sulfonamides in active rheumatic fever may be doubted. There seems to be no objective data to support this view, although it has been widely accepted by clinicians. This book may not be regarded as a definitive work in the general field of rheumatic fever, and its conclusions may require revision with increasing knowledge; nevertheless, it should serve as a valuable progress report on the present state of knowledge with respect to the control of rheumatic fever.

Case Histories in Psychosomatic Medicine. By Staff of Psychiatric Service, Massachusetts General Hospital, Boston. Edited by Henry H. W. Miles, M.D., Stanley Cobb, M.D. and Harley C. Shands, M.D. Cloth. $4.50. Pp. 306, with 4 illustrations. W. W. Norton & Company, Inc., 101 Fifth Ave., New York 3, 1952.

Selected cases from the clinical conferences conducted by the Psychiatric Service of the Massachusetts General Hospital are discussed from the point of view of this group's experience and approach to the understanding and handling of psychosomatic problems. Emphasis is on expanding psychiatric education, and the necessity of "team work" of psychiatrists, internists, surgeons, social workers, and psychologists is discussed. The book is planned with two chapters of preparatory material. The first chapter, which is concerned with principles and applications of psychosomatic medicine, gives a short review of the anatomic basis of emotional behavior and discusses three mechanisms of emotion, the evidence supporting emotions as a cause of disorders of function, the evidence that emotions may be one of the causes of lesions, selectivity of reactions, and psychophysiological sequences. The second chapter discusses the physician-patient relationship and its role in therapy. Following this are 21 case reports and conference presentations that cover a wide range of psychosomatic problems.

It is questionable whether the introductory chapters would satisfy the psychiatrist, the student, the general practitioner, or a specialist in any field. The chapter on the physician-patient relationship contains several ideas that seem to be used in an unusual way; for example, on page 38, the authors discuss constitutional factors from the physician's side of the physician-patient relationship and base their discussion on the physician's sex, age, and racial or national background. Although these factors are important, it is questionable whether they are constitutional factors.

Some of the conference discussions are disappointing. It is frequently impossible to detect the goals of the physicians taking part in the discussions. It seems as if one discussant is interested in the psychodynamics of the case, another in using the case material to teach, and a third in contributing generally. In addition, there is so much difference in the psychiatric approaches that one becomes confused.

Although the therapy discussed in the book is designated in the foreword as being psychoanalytically oriented, there is little evidence that this is generally so, in spite of the fact that several psychoanalytic formulations are offered. In many instances, the psychoanalytic approach is tied in by simply explaining that the psychoanalyst would use the word "castration" for something that other approaches would call "mutilation." The following quotation appears on page 201: "Some of the concepts developed by psychoanalytic investigations have appeared 'far-fetched' and even ridiculous to some physicians, in many instances because one cannot see explicit confirmation in the material obtained in a few face-to-face interviews." It would seem pertinent to question whether the approach to psychiatric problems by various disciplines is promoted for the student or the nonpsychiatrically trained physician by these demonstrations of how they work together.

Several patients mentioned in the case histories were presented to the staff. On the basis of the records, the presentations seem to have contributed little to the discussions; however, other psychiatrists have found that the presentation of the patient to the group can be beneficial to both patient and physicians.

In many of the case histories, the therapeutic approach seems to be highlighted by a warm, friendly, accepting physician-patient relationship. The role of therapy for patients whose cases are reported as failures therapeutically but who show remarkably beneficial results in the follow-up will be difficult for the beginner to understand. Although the purpose of this book, the need for discussion of the psychosomatic approach, is laudable, this effort does not, in general, achieve its aim.

The Cost of Sickness and the Price of Health. By C.-E. A. Winslow, Dr.P.H., Consultant in Public Health Administration, World Health Organization, Geneva. World Health Organization Monograph Series, no. 7. Paper. $1.50; 7/6; 6 Sw. francs. Pp. 106. World Health Organization, Palais des nations, Geneva; [Columbia University Press, 2960 Broadway, New York 27], 1951.

As part of a program for improved world health, Dr. Winslow, professor emeritus of public health at Yale University, has prepared this monograph as a basis for discussions on the economic value of preventive medicine. His thesis is that mankind suffers from many grave preventable diseases that not only involve human suffering but also impose a heavy burden on the economic resources of the regions involved. Cited studies indicate that "prevention is not only better than cure, it is also cheaper than cure." A recent study by the United States Committee on the Costs of Medical Care covering 9,000 families over a one year period showed that seven days a year were lost on account of illness, exclusive of institutional cases. It has recently been reported that in 1949 the United States had a total national income of $217,000,000,000 of which $10,600,000,000 (5%) was spent on medical and institutional care of the diseased. Any attempt to translate the statistics of life expectancy and disease prevention into terms of financial value to the community is difficult because of demographic differences of various countries.

There seems to be a high correlation between poverty and disease, or as Dr. Winslow states, "Poverty causes disease and disease creates more poverty in a vicious circle." But the prob-

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The reviews here published have been prepared by competent authorities and do not represent the opinions of any official bodies unless specifically stated.

lem does not end with the elimination of poverty or the creation of a sound financial system. It requires the integration of all the resources of agriculture, industry, economics, and education, with the combined effort aimed at better, more healthful living.

As a consultant to the World Health Organization, the author feels this organization should guide programs and studies in which the more fortunate areas may cooperate with those of less advanced development for the common goal of a healthful, prosperous, and peaceful world. Achievements that have been made in environmental sanitation, control of diseases spread from man to man, promotion of mental health, and promotion of positive health such as maternal and child care and nutrition, are listed and evaluated in areas where such achievement can be more easily measured. Plans, programs, studies, blueprints, outlines, and volumes of good suggestions are, unfortunately, worth very little unless they are financially and socially possible and until they are set in motion, since success is measured by the actual delivery of essential services.

You and Your Aging Parents. By Edith M. Stern and Mable Ross, M.D., Director, Prince Georges County Mental Health Clinic in Maryland. Cloth. $2.75. Pp. 212. A. A. Wyn, Publisher, 23 W. 47th St., New York 19, 1952.

One of the most important dividends of disease control by medical science is the prolongation of lives. Numerically, older citizens are increasing. Accordingly, many problems are confronting the medical profession. Not the least of these is the care of elderly parents. Questions concerning their relationship to their children, living quarters, physical care, and routine living are often the responsibility of their children.

This book discusses, in readable style, over 100 real-life histories that describe situations likely to appear. The book is written particularly for the children. It should be kept in mind that many elderly parents are capable of managing their own affairs: they cannot be classified as problem oldsters; and, as far as possible, they should guide their own destinies.

All too frequently, time brings about diminished resources. These situations are discussed in five sections: (1) the challenge of aging parents, (2) their living apart from their children, (3) their doubling up with other members of the family, (4) bereavements of aging parents, and (5) old age expectancies, safety, and comfort. A number of personal items such as illness and diet are discussed.

In this attractive volume, many families will find helpful suggestions that will enlighten them in their search for adequate answers to vexing problems that arise as their parents grow older. This book gives much detailed information that thousands of families with elderly parents will find of inestimable value.

Anatomy and Physiology for Nurses. By W. Gordon Sears, M.D., M.R.C.P., Physician Superintendent, Mile End Hospital, London. Second edition. Cloth. $2.50. Pp. 395, with 204 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2; Edward Arnold & Co., 41-43 Maddox St., London, W.1, 1951.

The author states that in the preparation of the second edition of this text he has aimed to keep the work as simple as possible without sacrificing the scientific and technical approach to the subject. Then he has attempted to include all facts that are essential for examination purposes and for meeting the requirements of the syllabus laid down by the General Nursing Council of England and Wales for the preliminary state examinations. Thirdly, his aim has been to present sufficient facts to make the subject understandable and interesting. And lastly, facts are included that have a subsequent practical application and serve as basic information for the study of other subjects in the curriculum.

The data on anatomy, designed to meet the requirements of students in nursing schools in Britain, may not, in all instances, be sufficiently detailed to serve as an anatomy text for nursing schools in the United States. The material on physiology is not explained and integrated to the degree that is essential if the subject is to be understood and applied in actual nursing practice. While the author states as his aim the promotion of understanding, interest, and ability to apply, his material seems actually to be directed essentially towards presenting facts with which average examination questions can be answered. This is not an uncommon shortcoming of anatomy and physiology textbooks for nurses. While the nursing curriculum does not allow for an extensive study of physiology, there is an urgent need for intensive study of a few basic physiologic principles that are essential in nursing knowledge and practice. While classroom instruction will contribute considerably to greater understanding and integration, a textbook is not fulfilling its purpose if its material does not at least approximate that which will be covered in the classroom.

This book by an English physician includes 15 chapters, each devoted to a system of the body. The style of presentation is almost in outline form. Under some circumstances, this book may be useful as a text on anatomy. It is doubtful if it can be effective as a text or reference on physiology for student nurses.

Untoward Reactions of Cortisone and ACTH. By Vincent J. Derbes, M.D., F.A.C.P., Associate Professor of Medicine, Tulane University of Louisiana School of Medicine, New Orleans, and Thomas E. Weiss, M.D., Instructor in Medicine, Tulane University of Louisiana School of Medicine. Publication number 131, American Lecture Series, monograph in American Lectures in Internal Medicine. Edited by Roscoe L. Pullen, M.D., Director, Division of Graduate Medicine, Tulane University of Louisiana. Cloth. $2.25. Pp. 77. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto, 2B, 1951.

The authors have been able to crowd a surprising amount of information into this short monograph, which is succinctly worded and excellently documented. The treatise first considers the physiological effects of corticotropin and cortisone and then illustrates how their physiological effects may be exaggerated into untoward reactions by treatment. The authors emphasize that treatment dosage, except in deficiency states, constitutes an unphysiological quantity of hormone, and outline the effects that may occur in various anatomic systems. The salient factors of interest are given in outline form at the head of each chapter; they are then more fully developed in the text. This handling of textual material is a definite aid to study and review. With the exception of the physiological considerations, the ideas concur with present day thought. This book is recommended as an excellent teaching and review text for medical students and physicians.

A Practical Handbook of Midwifery and Gynæcology for Students and Practitioners. By W. F. T. Haultain, O.B.E., M.C., B.A., Hon. Consulting Obstetrician and Gynæcologist, Royal Infirmary, Edinburgh, and Clifford Kennedy, M.B., Ch.B., F.R.C.S.Ed., Assistant Obstetrician and Gynæcologist, Royal Infirmary, Edinburgh. Including Section on The Management of the Infant and Neo-Natal Conditions. By J. L. Henderson, M.D., F.R.C.P.Ed., Professor of Child Health, St. Andrews' University, Dundee. Fourth edition. Cloth. $6. Pp. 412, with 48 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2; E. & S. Livingstone, 16 and 17 Teviot Place, Edinburgh 1, 1952.

This edition of this practical handbook of obstetrics and gynecology has been revised extensively, and new material has been added. The chapter on newborn and infant feeding has been brought up-to-date by Dr. J. L. Henderson, professor of child health at St. Andrews' University. A new section on the Rh factor has been written, and the section on venereal diseases has been revised.

The authors discuss modern teaching in this specialty. All the subjects are treated in a concise manner, many in outline form, devoid of nonessential details. Thus, the student or practitioner, for whom this book is intended, can easily obtain essential clinical data and ideas on appropriate therapy. A few carefully chosen illustrations amplify the text.

Although one finds reference to some practices that have become obsolete in the United States, such as uterine douching for hemorrhage, the obstetric and gynecologic teaching is sound and in the best Edinburgh tradition. This handy synopsis can be heartily recommended to students and general practitioners.

Human Biochemistry. By Israel S. Kleiner, Ph.D., Professor of Biochemistry and Director of Department of Biochemistry, New York Medical College, New York. Third edition. Cloth. $7. Pp. 695, with 88 illustrations. C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1951.

In this edition, many chapters have been revised considerably, particularly those sections concerned with mineral metabolism and water balance, respiration and acid-base balance, blood coagulation, and vitamin A function. New material has been added, but, because of restrictions on the size of the book, it was necessary to give only limited space to the discussions.

The topics covered include carbohydrates, lipids, proteins and their metabolism, tissues, milk, blood, urine, enzymes, digestion, vitamins, foods, physiological oxidations, mineral metabolism and water balance, chemistry of respiration and acid-base balance, energy metabolism, hormones, chemical structure in relation to biologic phenomena, and recent clinical applications. The last-mentioned chapter includes brief sections on liver function tests, kidney function tests, dental caries, biochemistry of inflammation, biochemistry of tumors, acid phosphatase and the prostate gland, antibiotics and sulfa drugs, radioactive isotopes, mucolytic enzymes, cortisone and corticotropin (ACTH), and pyrogens.

Each chapter has general and special references that include some of the literature through 1950. The appendix includes a table of standard buffer solutions, one for the composition of foods and another for the sodium and potassium content of foods.

Living in Balance. By Frank S. Caprio, M.D. Cloth. $3.75. Pp. 246. Arundel Press, Inc., P. O. Box 2606, Washington 13, D. C., 1951.

Dr. Caprio, in the preface to his book, states that "one of its purposes is to show how nervous breakdowns can be prevented . . . to teach the reader to achieve better living through better thinking." In a good measure he has succeeded. His style and arrangement is particularly designed to capture the interest of a large group of persons who, knowing little or nothing of the workings of the human mind, wish to learn how mental and nervous illness begins and what can be done about it. Some of Dr. Caprio's concepts and formulations are open to argument, but, generally, he has lucidly and simply discussed, in everyday language, the basic principles that reflect the attitude of present-day psychiatry. The chapter on self-analysis through dream interpretation might well have been omitted.

The chapters on marital situations, sex, fear, alcoholism, and neurotic reactions are written in a manner that will stimulate interest in persons who happen to need advice and guidance. The busy medical practitioner could well recommend this work to a large group of patients who come to him with perplexing problems of everyday living.

Textbook of Biochemistry. By Edward Staunton West, Ph.D., Professor of Biochemistry, University of Oregon Medical School, Portland, and Wilbert R. Todd, Ph.D., Associate Professor of Biochemistry, University of Oregon Medical School. Cloth. $12. Pp. 1345, with 80 illustrations. The Macmillan Company, 60 Fifth Ave., New York 11, 1951.

This book includes an excellent discussion of the principles of physical and organic chemistry, a feature that is often inadequately discussed in biochemistry texts. These principles are introduced in chapters on gases and solutions, electrolytic dissociation and the mass law, acids, bases, and buffers, osmotic pressure, the colloidal state and membrane phenomena, lipids, carbohydrates, monosaccharides, compound sugars, proteins, nucleoproteins, and nucleic acids. These sections comprise about 30% of the book. Correlation between facts and the underlying principles are repeatedly stressed throughout the book for pedagogic reasons.

The major portion of the text is comprehensive and detailed, with chapters on composition of tissues, enzymes, digestion, absorption, detoxication, bloods and body fluids, respiration, acid-base balance, electrolyte and water balance, energy metabolism, vitamins, biologic oxidation-reduction, intermediary metabolism, organic phosphates in metabolism, chemical energetics, lipid, carbohydrate, and protein metabolism, nutritional aspects of proteins, amino acids, lipids, and carbohydrates, metabolism of inorganic elements, foods, urine formation and composition, hormones, and antimetabolic agents.

There are many formulas, charts, and tables. The numerous tables of composition and values of biologic data compiled in one volume enhances its value as a ready reference work.

The material is clearly discussed, and both general and special references are included. This book is highly recommended to the physician to bring him up-to-date on the status of biochemistry in medicine and to the student who will be able to better appreciate the relationship between apparently isolated biochemical phenomena and general chemical and physical principles.

The Child in Health and Disease: A Textbook for Students and Practitioners of Medicine. By Clifford G. Grulee, M.D., Rush Professor of Pediatrics, University of Illinois, Chicago, and R. Cannon Eley, M.D., Assistant Clinical Professor of Pediatrics, Children's Hospital, Children's Medical Center, Harvard University Medical School, Boston. Second edition. Cloth. $15. Pp. 1255, with illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1952.

This text on pediatrics will be useful to students and general practitioners of medicine. In the preface, the authors give two reasons for sponsoring the second edition: first, medicine is moving so rapidly that the print is scarcely dry before there is something new on the subject; second, new subjects have been incorporated that were not included in the first edition. These subjects, rare in textbooks on pediatrics, include medical supervision in summer camps, cardiovascular surgery, viral hepatitis, erythroblastosis fetalis, and pancreatic fibrosis. The material is printed in two columns on each page. The book is easy to read, and each chapter contains a bibliography. Dr. Grulee's experience as a teacher of pediatrics, as editor of the *American Journal of Diseases of Children* for many years, and his official position with the American Academy of Pediatrics make him particularly well qualified to select the proper authors for the various chapters. A book of this type, which is so up-to-date, should perhaps be made into a loosely bound volume, so that newer aspects and more recent chapters can be added from time to time. This text is an excellent addition to the library of the pediatrician, general practitioner, or student of pediatrics.

Shock Treatments, Psychosurgery and Other Somatic Treatments in Psychiatry. By Lothar B. Kalinowsky, M.D., Research Associate in Psychiatry, College of Physicians and Surgeons, Columbia University, New York, and Paul H. Hoch, M.D., Principal Research Scientist (Psychiatry), New York State Psychiatric Institute and Hospital, New York. Foreword by Nolan D. C. Lewis, M.D., Professor of Psychiatry, College of Physicians and Surgeons, Columbia University, New York. Second edition. Cloth. $8.75. Pp. 396. Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1952.

This is an enlarged edition of the only authentic textbook on the place of the physical therapy in psychiatry. A large section on psychosurgery is included in the edition as well as a chapter on theoretical considerations. The extensive research into sources of data, which the authors have critically integrated with their personal experiences, is indicated by the 52 pages of bibliography. The book is well written and the subject matter demarcated to enable easy reference to various aspects of the subject. For psychiatrists employing physical therapy, this work should be a great help.

A Bio-Bibliography of Edward Jenner, 1749-1823. By W. R. LeFanu. Limited numbered edition. Cloth. $16. Pp. 176, with 29 plates. J. B. Lippincott Company, 227-231 S. Sixth St., Philadelphia 5; Aldine House, 10-13 Bedford St., London, W.C.2; 2083 Guy St., Montreal, 1951.

This volume consists of a catalogue of the writings of Edward Jenner, the discoverer of vaccination, together with a brief bibliographic sketch and numerous excellent photographic reproductions of original manuscripts and title pages. The book will be of interest largely to book collectors and librarians; however, the general reader will find that it contains much of interest, especially if he is unaware of the wide variety of investigations undertaken by this 18th century scientist, many in collaboration with his teacher and friend John Hunter.

QUERIES AND MINOR NOTES

LABORATORY FACILITIES IN RURAL AREA

To the Editor:—*My clinic in a rural area is 38 miles from the nearest clinical laboratory. We make our own electrocardiograms, x-ray examinations, basal metabolic rate studies, routine urinalyses, blood cell counts, and sedimentation rate studies. It is impossible to obtain a registered laboratory technician; therefore, to obtain chemical analyses, we are obliged either to mail the blood or to send it by bus to the laboratory mentioned, and often the blood is unsuitable for examination by the time it reaches its destination. Furthermore, the accuracy of tests on mailed blood specimens can be challenged. Are there simple, accurate methods of chemical analysis that can be performed by untrained personnel? Is a photoelectric colorimeter simple enough for use by untrained personnel? In mailing the specimens, what preservative should be used? How long will blood keep under mailing conditions? Kindly discuss this in relation to such determinations as nonprotein nitrogen, urea nitrogen, blood sugar, chloride, sodium, cholesterol, and protein levels, and liver function tests. I believe this to be an acute problem in rural practice, especially since we younger men are attempting to establish the same high standards of medical care that prevail in larger cities.*

J. J. Kirschenfeld, M.D., Fort Deposit, Ala.

Answer.—Whether it is logical to establish a routine blood chemistry laboratory in a rural clinic when it is impossible to obtain a registered laboratory technician is not a simple question; however, if an adequate clinical laboratory is available within 38 miles, it would appear that it would not be logical. There are many advantages that rural areas do not have, including brain surgeons, laboratories for cardiac catheterization, and general clinical laboratories. There are no simple, accurate, and foolproof methods for chemical analyses, and chemical analyses cannot be performed by untrained office personnel. Photoelectric colorimeters are highly technical instruments and require constant attention by someone skilled in their use, and highly trained personnel are needed to calibrate them. If the clinical laboratory referred to is within 38 miles of the rural clinic, it would appear that a satisfactory arrangement could be made with the laboratory for examining and reporting on specimens. This presupposes that the laboratory is competent. There are anticoagulants that have been described as being satisfactory for shipping specimens by mail. They vary with the test to be performed and the method used. Sodium fluoride and thymol will preserve specimens for sugar determinations and many other tests but cannot be used for urea. Sodium oxalate can be used when urea is to be tested.

STIMULATION OF GROWTH

To the Editor:—*A 15-year-old boy is only 5 ft. 1 in. (154.9 cm.) tall. His physical development is entirely normal, except for his height, which is partly a family characteristic. Would hormone therapy help his growth?*

N. B. Livingston Jr., M.D., Mohall, N. D.

Answer.—The patient should be carefully studied from a general medical and endocrinological point of view. Roentgenograms should be taken of the bones of the elbow, hands, and knees to see whether epiphyseal closure has occurred and to estimate the growth potential remaining in the skeleton. If the epiphyses are closed, future growth is impossible. If they are open, the degree of potential growth can be estimated by careful study. If the patient has hypothyroidism, the administration of desiccated thyroid will stimulate growth. If his basal metabolism is within normal limits and he is maturing sexually at a satisfactory rate, no form of therapy will be effective. If the patient is sexually immature, growth of the skeleton can be stimulated by the administration of chorionic gonadotropin or androgen. Androgen is not a substitute for the thyroid hormone or for the growth hormone of the pituitary, without which the skeleton cannot attain normal length. Roentgenograms of the sella turcica should be made to exclude the presence of a pituitary tumor. The growth hormone of the pituitary is not very effective in stimulating growth in man.

LOSS OF HAND FUNCTION

To the Editor:—*Please give an opinion on the percentage loss of function of a hand in which there remains a good functioning thumb, two-thirds of the middle finger, slightly over one-half the ring finger, and a little over one-half the small finger. The patient closes his hand with some difficulty because of residual swelling in the palm and in the amputation stumps.*

M.D., New York.

Answer.—Since there is no uniform system of rating disability in all states in this country, it is difficult to give an opinion on the percentage loss of function of this hand. Although it may be possible to ascribe a certain percentage of disability based on the loss of part or all of certain digits, a major factor in considering the percentage loss of function should be the particular loss of function that has resulted in this case. Before establishing a permanent disability rating, it should be determined whether further improvement in the usefulness of the hand can be obtained by the use of rehabilitative procedures, including physical therapy and occupational therapy. The patient's occupation should also be considered in determining the amount of final disability. Assistance in determining the percentage loss of function may need to be obtained by reference to tables of disability acceptable to the various parties having an interest in this particular case. The complexity of the problem of determining the percentage loss of function of a hand is apparent in an excellent discussion of this subject that appears in the book "Accidental Injuries" by H. H. Kessler (ed. 2, Philadelphia, Lea and Febiger, 1941).

ELECTRIC SHOCK TREATMENTS

To the Editor:—*Some of my patients with melancholia were given electric shock treatments years ago, but I was discouraged by the violent reactions produced and the uncertain results. Do the later type machines produce the violent convulsions produced by the older types, and do they produce better results?*

W. B. Chapman, M.D., Joplin, Mo.

Answer.—Electric shock therapy involves the application of electric currents to the head. These currents usually vary from 300 to 1,200 ma., produced by voltages of from 70 to 150 volts, and current passage times of 0.1 to 0.5 seconds are used. The purpose of the application of these currents is to produce a convulsive seizure. The dose required for production of the convulsive seizure varies from patient to patient and must be determined by the person applying the treatment. The use of an electrical device in shock therapy is advantageous in that application is simple and it is possible to induce either minor or major seizures. Modifications in the older type machines have been made, but the machines still produce a convulsive seizure.

Some use has been made of tubocurarine chloride to reduce the contractile power of the skeletal muscle in conjunction with electric shock therapy. For shock treatment as used in psychiatry, New and Nonofficial Remedies (Chicago, American Medical Association, 1952, p. 431) gives the usual dose of tubocurarine chloride as 3 mg. (20 units) for each 40 lb. (18.1 kg.) of body weight. Usually, for greater safety, a smaller dose is used initially.

The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

ASYMPTOMATIC NEUROSYPHILIS

To the Editor:—*A woman, aged 45, is symptom-free from latent syphilis. Her weight has not changed in 10 years. She gives a history of latent acquired syphilis of 22 years duration. In 1950, her husband developed cerebral spinal syphilis with paresis. At that time she sought treatment and was given 600,000 units of penicillin daily for 15 days. On Jan. 17, 1952, a blood serum test was strongly positive (4+) for syphilis. The results of a spinal fluid examination were as follows: quantitative reaction to Wassermann test, 444; 1 white cell per cubic millimeter; the globulin level was normal, the total protein content being 19 mg. per 100 cc.; and the colloidal gold test reaction was 1122100000. What treatment is indicated for this patient? Should treatment with a saturated solution of potassium iodide be continued?*

M.D., Pennsylvania.

Answer.—If the patient described has not yet been examined by a skilled neuropsychiatrist, such a consultation should be promptly requested. If the results of his examination are normal, the patient may be considered as having asymptomatic neurosyphilis. If the latter proves to be the correct diagnosis, the patient is not in need of further treatment. The spinal fluid apparently was not examined before the patient received treatment. On examination 8 months after the administration of 9 million units of penicillin, the spinal fluid is "inactive" in the sense that the cell count and protein content are normal. The normality of these two tests indicates that whatever inflammatory component of the asymptomatic neurosyphilis may have been present when treatment was started has now been adequately dealt with. There is a 90 to 95% certainty that this result will be permanent. Nothing is gained by the administration of potassium iodide, arsenic or bismuth, or, for the time being at any rate, additional penicillin therapy or fever therapy.

The patient should be followed from this point with repeated physical (especially neuropsychiatric) examinations and with recheck examinations of the blood serum and the spinal fluid at intervals of every 6 months for the next 3 years, at yearly intervals thereafter for another 3 years, and at somewhat longer intervals after that. Indications for retreatment will be the appearance of clinical signs or symptoms of neurosyphilis of any type or the reappearance in the spinal fluid of an increased cell count and/or protein content. The persistence of a positive complement fixation test and/or flocculation test in the blood and spinal fluid may be ignored as long as the patient's physical condition and the cell count and protein content of the spinal fluid remain normal.

UNDESCENDED TESTES IN YOUNG BOY

To the Editor:—*What is the consensus regarding treatment of a 3-year-old boy with bilateral undescended testicles? The boy is otherwise well developed, but the testicles cannot be palpated either in the scrotum or in the canals. The penis is extremely small and was noted as being very small at birth. The pregnancy and delivery were uneventful. Other siblings are normal, as are the parents.*

M. E. Friedman, M.D., New Buffalo, Mich.

Answer.—The following point of view is the one most generally accepted concerning the treatment of undescended testes: 1. Accurate diagnosis is important. The patient should be examined at least twice in both the upright and recumbent positions to ascertain the exact location of the testis. 2. The earlier the testis is brought into the scrotum the better its function in later life. The testis cannot function normally except in the environment of the scrotum. The longer it remains at some level above the scrotum, the greater the likelihood of permanent damage. 3. Glandular treatment should be instituted as soon as it is practical to carry out operative procedures so that these may be used if glandular therapy is not effective. This means that treatment should be started by about the age of 3 years. 4. The material of choice is chorionic gonadotropin. It should be given in a dose of 500 I. U. by intramuscular injection 3 times a week. In patients with bilateral undescended testes, chorionic gonadotropin may be ineffective. Under these circumstances, testosterone propionate should be given in a dose of 25 mg. 3 times a week by intramuscular injection. 5. If glandular therapy is effective, descent will usually occur within eight weeks. If the treatment is ineffective, operative procedures should be carried out at once by some one specially skilled in this field. Great care must be taken to preserve the blood supply of the testis. Glandular therapy should be continued until the time of operation. 6. Only a small amount of genital growth should be induced. This means that chorionic gonadotropin and testosterone propionate should not be continued for a long period of time in a young boy.

FERTILITY IN MEN

To the Editor:—*What effect, if any, have the following on fertility in men: (a) inhalation of carbon tetrachloride, (b) high altitude flying, (c) inhalation of fumes of the galvanizing process, (d) exposure to radar waves, as experienced by naval personnel who are in fairly constant contact with them, and (e) infectious hepatitis?*

Norman L. Wilson, M.D., Quincy, Mass.

Answer.—(*a*) Inhalation of carbon tetrachloride does not cause male sterility. The liver detoxicates this chemical. (*b*) High altitude flying over long periods of time has not been proved to cause male sterility. Modern airplane cabins have both pressure and oxygen control for human safety. (*c*) In the galvanizing process, iron is coated with tin and is then immersed in a zinc bath. Physiological damage from exposure to this process would be the same as occurs in lead or zinc poisoning. Progressive anemia may occur and may result in temporary or permanent sterility. (*d*) Exposure to radar waves does not cause sterility. Radar waves are high frequency electromagnetic radiation and may have the same frequency as short-wave or ultrashort-wave radio waves. They do not produce ionization within tissues and cannot cause sterility. The alpha, beta, and gamma rays generated by radioactive substances, such as radium, or by x-ray apparatus are the three types of rays usually considered in the problem of sterility. The gamma rays are those that produce sterility; the beta rays, it is believed, probably do not produce sterility; the alpha rays cannot produce sterility. (*e*) In infectious hepatitis as a primary infection of the liver, secondary sites of infection may develop as a result of invasion by the infecting organism, after its transmission through the blood stream. Orchitis may result, with unilateral or bilateral sterility, depending on the severity of the attack and the extent of involvement (one or both testicles). Mechanical blocking of the vas deferens by invading organisms, such as filariae and amebas will prevent egress of spermatozoa from the vas deferens, causing male sterility. High body temperatures sustained over long periods of time can destroy the ability of the testes to manufacture spermatozoa.

DIATHERMY IN REGION OF METALLIC IMPLANTS

To the Editor:—*A patient with several silver wires in her fractured patella needs short wave diathermy for her arthritic knee. Is it dangerous to apply diathermy to bones with silver wires in them? Is there a type of diathermy (for instance, diathermy by Microtherm®) that can be applied with safety?*

M.D., New York.

Answer.—Both short wave and microwave diathermy, which are effective in heating human tissues, are even more effective in the heating of metals. For this reason, it is generally felt that in any condition in which metallic implants are present near the area or in the area to be treated, short wave and microwave diathermy are contraindicated. Metallic substances, such as bullets, screws, bands, and wires in tissues may be excessively heated if diathermy is applied. It is known that, in many instances, damage has not occurred, but there is a potential danger, both actual and theoretical, which makes it advisable to heat these areas by means other than medical diathermy.

REFERENCE

Lion, K. S.: The Effect of the Presence of Metals in Tissues Subjected to Diathermy Treatment. Arch. Phys. Med. 28: 345-347 (June) 1947.

PENICILLIN INJECTIONS

To the Editor:—*In* The Journal, *June 28, 1952 (page 906), the answer to the query about penicillin injections contains a serious error. It suggests the use of "two injections a day of 300,000 to 600,000 units of crystalline penicillin G or K." A number of years ago it was conclusively shown that penicillin K was relatively ineffective in almost all infectious diseases. It is not obtainable commercially, and all penicillin manufacturers make every effort to remove penicillin K from their products. The only penicillins now commercially available are penicillin G and penicillin O in the form of one or another of their salts. The answer is further at fault in that it makes no distinction between repository preparations of penicillin G and aqueous solutions of crystalline penicillin.*

J. E. Moore, M.D.
803-807 Medical Arts Bldg.
Baltimore 1.

FLUORIDE BY MOUTH DURING PREGNANCY

To the Editor:—*In answering the question of whether fluoride given to the mother by mouth during pregnancy will cause the baby to have better teeth in life, it is stated that there is no evidence that fluoride taken by the mother during pregnancy will cause a baby to have better permanent teeth* (J. A. M. A. *March 1, 1952).*

In the April, 1951, Journal of Dental Medicine, *Dr. Reuben Feltman of Passaic, N. J., writes "Since 1948, 309 children have been born to mothers who have ingested tablets containing 2 to 4 mgms. of Calcium fluoride daily. Some of the children who were born from these mothers have been examined. The results to date are most heartening,—the numbers are too small and the ages too young to be of importance at this stage of the investigation.*

"This much can be reported, the caries rate in three year olds in this area is two decayed teeth. Caries has been found in only one child. The enamel has the appearance of the inside of an oyster shell highly polished and pearly."

The following quotation is from "Industrial Fluorosis: A Study of the Hazard to Man and Animals Near Fort William, Scotland" (Medical Research Council, Memorandum no. 22, London, His Majesty's Stationery Office):

"Evans Phillips and Hart (1938) suggested that fluorine is transferred through the placenta of the cow to the foetus. Velu (1936) held that fluorine in young animals born of mothers exposed to fluorine must have passed to the foetus. Knouff (1936) confirmed the passage of fluorine through the placenta of bitches."

Ample evidence has been compiled from the literature to lend feasibility to the theory that fluorine ingested during pregnancy and the formative years of tooth development will cause a reduction in the incidence of dental caries. There is a maternal transference of fluorine, which makes both the deciduous and permanent teeth less subject to caries if the child continues to get the optimum daily dose regularly.

David W. Bronson, D.D.S.
200 W. 40th St.
New York 18.

o the Editor:—*A question concerning the effect of fluoride given by mouth during pregnancy was answered in your column in* The Journal *of March 1, 1952, with the statement that "the tooth buds of the permanent teeth are not even formed at the time of birth [so] it is difficult to see how fluoride can affect the baby's future teeth in utero."*

According to J. A. Salzmann (Principles of Orthodontics, Philadelphia, J. B. Lippincott Company, 1943, p. 182) epithelial buds are present for the permanent incisors, cuspids, bicuspids, and first molars at the time of birth, and differentation of the tooth germ has so far advanced that calcified tissue is found in the germ of the maxillary and mandibular first permanent molars. The permanent mandibular first incisor also shows calcification. The tooth germ of the permanent maxillary first incisor and the permanent mandibular second incisor have reached a high degree of development. The precursors of the other teeth listed above are described as small undifferentiated buds at this stage.

It would seem, therefore, that the failure of ingested fluoride to affect the baby's teeth in utero must depend upon some mechanism other than the lack of permanent tooth buds in the fetus. Such failure, it is not inconceivable, may even be apparent rather than real and due to difficulties in assembling and evaluating clinical material.

John H. Rasse, D.M.D.
Lafayette and Court
Marshall, Mo.

PAINFUL FEET

To the Editor:—*I would like to add the following aids that I have found valuable in the treatment of metatarsalgia to that described in* The Journal *March 8, 1952 (p. 877):*

1. The use of carpeting with foam rubber underpads to provide a soft walking surface in the home or office. 2. The addition of an extra leather sole to the shoe. This helps tremendously to give a cushioned effect to the sole of the shoe. 3. Application of metatarsal bars to the shoe. I have found it best to make and apply these myself.

H. C. Goldberg, M.D.
7 Watchung Ave., Plainfield, N. J.

PROGRESSIVE MYOPIA IN A CHILD

To the Editor:—*In the query concerning progressive myopia in a child in* The Journal *March 29, 1952 (page 1165), a statement was made that "The amount of correction of myopia has no effect on the progress of the refractive error." I agree that in some young patients who abuse their eyes, myopia cannot be checked even with the strongest glasses; however, in most cases the progress of myopia can be decreased by the use of full correction eyeglasses. This favorable outcome is significant socially, particularly for girls. If the myopia can be checked at the strength of three diopters, attendance at formal functions later in life will be possible without eyeglasses.*

I disagree with the philosophy that "There need be no insistence on the wearing of the glass unless clear vision is preferred by the patient." This erroneous concept is contrary to the concepts of modern preventive ophthalmology and may explain why some physicians do not refer young myopic patients for refraction until complaints of poor vision are recorded by the school authorities. The progress of myopia is accelerated when the indifference suggested in the reply is practiced. Normal or 20/20 vision must be demanded by all practitioners and not left to the whims of children or their parents.

Teachers in the field of eye muscle dysfunctions know that in young myopic patients with accommodative exophoria who fail to wear eyeglasses constantly the muscle imbalance may be aggravated and divergent strabismus may develop. In a series of cases previously reported (Smith, J. W.: The Problem of Strabismus in Childhood, with Especial Reference to Convergent Strabismus in Infancy and Early Childhood, Am. J. Ophth. **31:**85 *[Jan.] 1948), 10% of patients in early stages of divergence were restored to orthophoria by continually wearing eyeglasses to correct their myopia.*

It is regrettable that authorities are not in agreement regarding the treatment of myopia. In my experience, extending over thirty years, early and full correction and insistence on the constant wearing of the glasses during the school years is the regimen that pays dividends in good eye health. This plan insures the minimum of progression and in many cases actually arrests the myopia.

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DISEASES OF THE AGED

ANALYSIS OF PATHOLOGICAL OBSERVATIONS IN 1,251 AUTOPSY PROTOCOLS IN OLD PERSONS

Leon S. Medalia, M.D.
and
Paul D. White, M.D., Boston

For the first time in history, the average expectation of the duration of human life at birth has passed the Biblical mark of three score and 10 years. This applies to a girl born in the United States today; a boy has an expectation of about five years less. This sex difference in longevity is a characteristic found throughout the animal kingdom and may prove to be an insurmountable law of nature. It is gratifying to know, however, that the duration of human life has steadily and rapidly increased during the past two generations for both sexes. Coincident with our gaining control primarily over the infectious diseases in the majority of persons, both young and old, a heavy responsibility has devolved on us to study and to lessen, as far as we are able, the progress and effects of the chronic, so-called degenerative diseases, mainly those involving the heart and blood vessels, in the earlier old age decades as well as to combat malignant neoplastic disease. If we can eventually make possible the maintenance of reasonably good health in our fellow citizens up to the age of perhaps 90 and can utilize their accumulated experience, knowledge, and wisdom, the world will be a better place in which to live. In order to make a start in this worth-while, indeed essential, effort, we must establish a baseline of knowledge of the incidence of diseases in the aged, for adequate information in this area is still very scanty. This paper is an initial effort to record more accurate data on the incidence of the diseases of older persons than have been hitherto available.

At the time this study was finished and was ready for publication (January, 1950), we had found only one reference in the literature that was in any way related to our work—an article published in 1949 by Trevor Howell.[1] He reported the pathological observations in 240 patients who were 80 years old and over made in a large general hospital in London. No comparisons were made by him between data for this group and those of other age decades. As an illustration of the paucity of such studies in the literature he quotes Sherlock Holmes: "I have no data yet. It is a capital mistake to theorize before one has data. Insensibly one begins to twist facts to suit theories, instead of theories to suit facts." It was this lack of data in the literature that held up our publication of this article. We believed our inability to find any references in the literature pertaining to our work was due to the lack of a proper search, so we continued searching.

In a recent article entitled "Morbid Anatomy of Old Age," Trevor Howell begins by saying, "There is as yet little knowledge concerning the morbid anatomy found in aged patients."[2] In this article he reports the pathological observations in 25 patients aged 90 or over and in 90 patients between the ages of 80 and 89. In both series he deals with the description of the pathological conditions. He makes no comparisons between the patients of different decades nor between men and women; however, it is a worth-while study, and the cumulative reports of such studies, we believe, will be of great value in the future.

Rosenthal and Feigin [3] give no statistical data on their observations in patients of different age groups. On the other hand, they quote Philo, who, in the same hospital, studied cancer of the prostate in 1,081 patients on whom autopsies were done. He reported his observations according to the patients' ages by decades from 40 years old and over, thus providing material for comparison. Monroe [4] recently published a survey of the records of the patients 61 years old and over who were treated at the Peter Bent Brigham Hospital from its opening in 1913 to 1943. Although he states that some of the information given in the records was verified by autopsy, his report is

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Read before the Section on Biology and Medicine at the Second International Gerontological Congress, St. Louis, September 11, 1951.

[1] Howell, T.: Old Age, Geriatrics 4:281, 1949.

[2] Howell, T. H., and Piggot, A. P.: Morbid Anatomy of Old Age, Geriatrics 6:85, 1951.

[3] Rosenthal, J., and Feigin, I.: Some Aspects of More Common Pathological Findings in Hospital for Chronic Diseases, Geriatrics 3:1, 1948.

[4] Monroe, R. T.: Diseases in Old Age, Cambridge, Mass., Harvard University Press, 1951.

Myocardial infarcts were found in only 2.5% (6 men and 4 women) of the 406 patients with normal heart weights, although 47 others (28 men and 19 women) showed myocardial scarring; in contrast, 13% (70 men and 42 women) of the 845 persons with abnormal heart weights had myocardial infarcts, and 197 more (114 men and 83 women) showed myocardial scarring. Myocardial infarcts and scarring occurred with little difference in frequency in patients in all four decades (fig. 2).

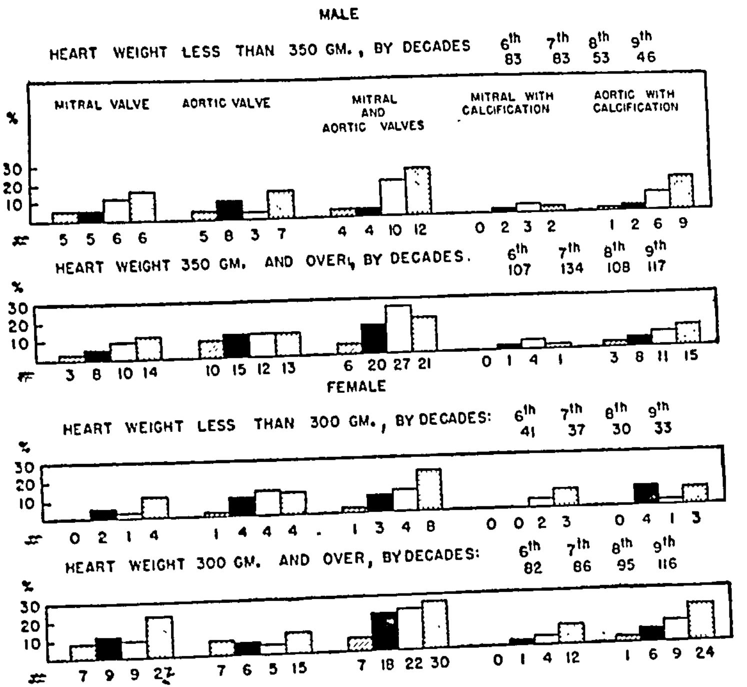

Fig. 5.—Incidence of valvular sclerosis in the 1,251 patients whose autopsy protocols were analyzed (arranged as in figure 2).

Aortic atherosclerosis without calcification (fig. 4) dedecreased in frequency of occurrence in successive decades in both men and women with hearts of normal weight; for example, it occurred in about 65% of the men in their sixth decade compared with 40% in their ninth decade and in about 60% of the women in their sixth decade compared with 28% in their ninth decade. Aortic atherosclerosis with calcification, however, increased in occurrence in successive decades in both men and women with normal heart weights, increasing from 12% of the men in their sixth decade to 55% in their ninth and from 10% of the women in the sixth decade to 75% in their ninth. There was much less difference in the incidence of aortic atherosclerosis either among the decades or between the sexes in patients with increased heart weights.

Valvular sclerosis with or without calcification, especially basal, (fig. 5 and 6) was seen occasionally, occurring increasingly with advancing age. It occurred in either the mitral or the aortic valve or, more frequently, in both, whether or not the heart weight was increased. Recognizable rheumatic heart disease was rare, being diagnosed in only 16 (5%) of 313 patients in their sixth decade, in 17 (5%) of 340 patients in their seventh decade, in 10 (3.5%) of 286 patients in their eighth decade, and in 8 (only 2.5%) of 312 patients in their ninth decade.

Hypertension in slight degree was not a rare condition, especially in the case of persons with hearts that were heavier than normal, but it did not vary much in occurrence either in the decades or between the sexes. There were 165 (13.2%) patients with slight hypertension and abnormally heavy hearts and only 33 (2.5%) with slight hypertension and hearts of normal weight. Fewer patients had moderate hypertension, which again was much commoner among those with heavier hearts, 116 (9%) patients being affected, than among those with hearts of normal weight, 18 (1.5%) patients being affected. Marked hypertension was rare in these older age patients, confirming the clinical impression that malignant hypertension is a disease primarily of youth and middle age; it occurred in only 35 patients (2.8%) altogether, of whom all except 2 had abnormally heavy hearts and only 23 were over the age of 60.

Cerebral vascular accidents occurred oftenest in patients under age 70, affecting 67 (5.4%) patients altogether, only 21 of whom were over 70. Only 2 of the latter 21 patients had hearts of normal weight, but 24, or slightly over one-half, of those under age 70 had hearts of normal weight. The cases were about evenly divided between the sexes, occurring in 35 men and 32 women. Acute pericarditis was seen in 100 (8%) patients in all, of whom 66 were men and 34 were women. All decades were represented, from 22 patients in the sixth to 23 in the ninth, but there was a considerable minority of patients (only 19 of the 100) whose hearts were not overweight. Cardiovascular syphilis (fig. 4) was surprisingly rare, being found in only 51 (4%) patients altogether. Forty of the patients were men, and only 20 patients had hearts of normal weight. Only 3% (23 men and 15 women) of the total number of patients had aortic aneurysms.

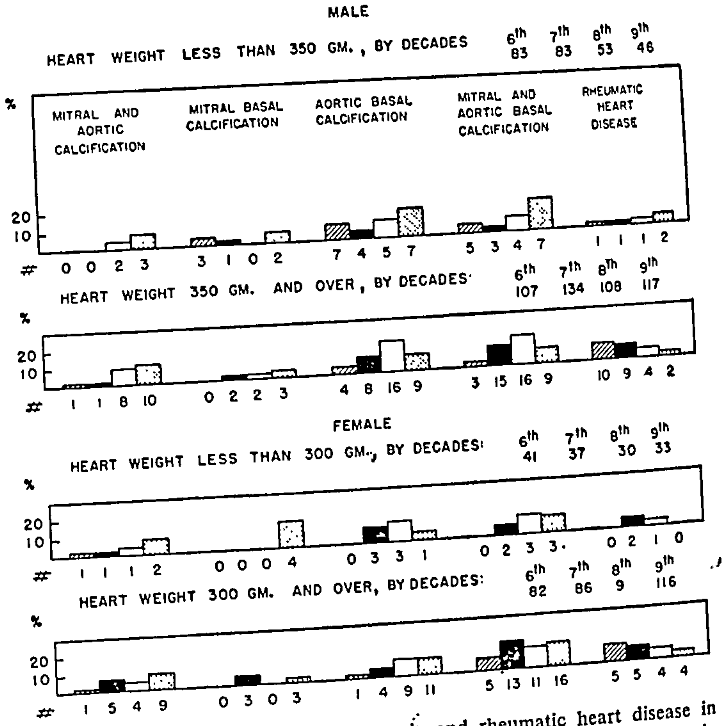

Fig. 6.—Incidence of valvular sclerosis and rheumatic heart disease in the 1,251 patients whose autopsy protocols were analyzed (arranged as in figure 2).

Nephrosclerosis was common, being diagnosed in 460 (37%) patients. Its prevalence increased with increasing age, occurring in 71 patients in their sixth decade and in 171 in their ninth. There was no appreciable difference in its occurrence in the sexes (remembering that men outnumbered women in the total series 7 to 5), 249 men and 211 women being affected. There were 121 (about 25%) of these patients whose hearts were not overweight. In contrast to arteriosclerotic nephritis (nephrosclerosis), other kidney diseases were relatively rare. There were only 16 patients who had nephritis as such and 105 who had pyelonephritis, of whom 68 were men and 37 were women. Cystitis occurred in 134 (11%) patients, and was seen more often in the men (86) than in the women (48).

Noncardiovascular Diseases.—Of noncardiovascular diseases, bronchopneumonia (fig. 1) occurred oftenest, affecting a total of 449 (36%) patients, the number affected increasing with increasing age from 81 in their sixth decade to 147 in their ninth. There was an uneven sex distribution, with 270 men and 179 women affected. Lobar pneumonia was relatively rare; there were only *51 (4%)* cases altogether, 38 in men and only 13 in women. Next in prevalence was cancer (fig. 1), with 303 (24%) patients affected, 68 in their sixth decade, 83 in their seventh, 85 in their eighth, and 67 in their ninth. The distribution of cases by sex was even for the series, with 167 in men and 136 in women. The majority of these patients (160) had hearts of normal weight or below. Active tuberculosis (fig. 1) was rare, occurring in only 117 patients altogether, with 39 in their sixth decade, 30 in their seventh, 34 in their eighth, and only 14 in their ninth. It was commoner in the men (78) than in the women (39) and occurred oftener in persons with normal heart weight (71) than in those with abnormal heart weight (46).

Blood diseases, chiefly anemia, were rare, affecting only 35 patients altogether, 22 women and 13 men. Cirrhosis of the liver was rare, occurring in 107 patients (64 men and 43 women). It was commonest in patients in their sixth decade (35) and showed no particular relation to heart weight. The occurrence of cholecystitis increased with increasing age, affecting 17 patients in their sixth decade, 37 in their seventh, 43 in their eighth, and 61 in their ninth. Women were affected oftener than men, numbering 100 and 68, respectively. Peptic ulcers (fig. 1) were rare. Only 43 patients (29 men and 14 women) had duodenal ulcers, and only 39 (22 men and 17 women) had gastric ulcers. The total of 82 patients made up only 6.5% of the entire group. .

CONCLUSION

This survey of 1,251 autopsies of aged persons has brought to light some well known and some rather surprising indications of disease incidence in old persons. This study indicates the need for further analyses of this sort to establish a firmer foundation for the appraisal of measures for the medical care and the prevention of disease in the increasing number of old persons. Social, economic, and educational as well as medical advances are to a certain degree dependent on this increasing knowledge.

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DUODENAL ULCER

RESULTS OF TREATMENT WITH SUBDIAPHRAGMATIC VAGUS RESECTION AND GASTROENTEROSTOMY; INTERIM REPORT

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The physiological basis for the use of vagotomy in the treatment of duodenal ulcer rests on two findings: (1) patients with duodenal ulcer have increased nocturnal gastric secretion of hydrochloric acid and (2) complete vagotomy abolishes the increased secretion.[1] Excellent clinical results from the use of subdiaphragmatic vagotomy with gastroenterostomy in duodenal ulcer have been reported by Dragstedt, Crile, and others.[2] An unpublished report of a nation-wide survey conducted by the vagotomy committee of the American Gastroenterological Association in 1950 cited excellent results from the operation in nearly 98% of the patients. Other reports have indicated an unsatisfactory experience with the operation, and the 1951 report of the committee mentioned above was less impressive than that of the preceding year, although there were still about 94% of patients satisfied with the results of the operation.[3]

Because of these conflicting views, further studies on the long-term results of the operation are desirable to form a basis for critical comparison with results from other procedures, such as gastric resection. In this connection, a report of the present condition of 147 patients who have been followed from two to four years after vagus resection with gastroenterostomy for duodenal ulcer is presented.

From the Cleveland Clinic Foundation and Bunts Educational Institute.

[1] Levin, E.; Kirsner, J. B.; Palmer, W. L., and Butler, C.: A Comparison of the Nocturnal Gastric Secretion in Patients with Duodenal Ulcer and in Normal Individuals, Gastroenterology **10**: 952 (June) 1948. Woodward, E. R.; Harper, P. V., Jr.; Tovee, E. B., and Dragstedt, L. R.: Effect of Vagotomy on Gastric Secretion in Man and Experimental Animals, Arch. Surg. **59**: 1191 (Dec.) 1949.

[2] Dragstedt, L. R., and Camp, E. H.: Follow-up of Gastric Vagotomy Alone in the Treatment of Peptic Ulcer, Gastroenterology **11**: 460 (Oct.) 1948. Crile, G., Jr.: Surgical Treatment of Duodenal Ulcer: A Comparison of Results of Treatment with and without Vagotomy, Rev. Gastroenterol. **17**: 781 (Sept.) 1950. Orr, I. M., and Johnson, H. D.: Vagotomy for Peptic Ulcer: Indications and Results, Brit. M. J. **2**: 1316 (Dec. 10) 1949.

[3] Jordan, S. M.: Present Status of Vagotomy, in Sandweiss, D. J.: *Peptic Ulcer: Clinical Aspects, Diagnosis, and Management*, Philadelphia, W. B. Saunders Company, 1951, p. 539. Walters, W., and Fahey, M. M.: Vagotomy: Immediate Results in 28 Cases and Later Results in 68 Cases, Proc. Staff Meet., Mayo Clinic **24**: 501 (Sept. 28) 1949. Healy, M. J., Jr., and Sauer, P. K.: Some Limitations of Vagotomy in Treatment of Peptic Ulcer: Critical Follow-up Analysis of 50 Cases, Ann. Surg. **130**: 985 (Dec.) 1949.

MATERIAL

The study is based on the hospital records and the answers to questionnaires received from a group of 147 patients who were operated on between September, 1946, and March, 1949, at the Cleveland Clinic Hospital. Since seven additional patients operated on during this period could not be traced, the follow-up is 95% complete. Two years had elapsed since operation for 78 patients, three years for 53, and four years for 16. These patients had physical evidence of a duodenal ulcer at the time of laparotomy in nearly every instance; in the few cases in which it was not demonstrated, there was roentgenographic evidence together with a consistent clinical history. The questions submitted to the patients were as follows:

1. Have you had any recurrence of ulcer pain since your operation?
2. Have you had any other abdominal distress? If so, describe.
3. Have you had any diarrhea? If so, how many stools a day on the average?
4. To your knowledge, has hemorrhage from the stomach occurred since operation?
5. What is your present weight?
6. How is your appetite (good, fair, poor)?
7. Are you satisfied with your operation?
8. Are you able to work or perform your usual duties?

Table 1.—*Age Distribution of Patients*

Age, Yr.	No. of Patients
20-29	6
30-39	25
40-49	48
50-59	53
60-69	14
70 plus	1
Total	147

Table 2.—*Duration of Symptoms*

Duration, Yr.	No. of Patients
1 or less	3
2-4	17
5-9	36
10-14	28
15-19	21
20 plus	42
Total	147

Table 1 gives the age distribution. It will be noted that approximately half the patients were less than 50 years of age. Some believe that in the older patients a gastroenterostomy alone will give a good clinical result but that in the younger patients a high incidence of marginal ulceration is to be expected if a vagotomy is not performed at the same time. (The experience of the Cleveland Clinic is that age is not an important factor in the development of marginal ulceration.) Table 2 shows the duration of symptoms. It may be seen that ulcer symptoms had existed for many years in the great majority of patients and that these patients, by inference, had had years in which to try the effects of a medical regimen.

INDICATIONS FOR OPERATION

A review of the records of the 147 patients shows that the indications for surgery were strict. The percentage of patients who are operated on for duodenal ulcer and its complications at the Cleveland Clinic, in comparison with the total number treated for this disease, has remained remarkably constant in recent years, varying from 10% to 15%. During the period studied, approximately 13.5% of the patients with duodenal ulcer were treated surgically. The policy at the Cleveland Clinic has been to regard duodenal ulcer as fundamentally a "medical" disorder and to recommend surgery only for those patients who have had one or more of the major complications or who have had repeated and incapacitating recurrences of activity of the ulcer.

Table 3 lists the primary indication for surgery, as well as previous complications and weight loss in each main group. It is seen that in the group of 75 patients operated on primarily for "intractability" there were 11 who gave a history of hemorrhage from the ulcer; eight had a history of a previous perforation, and two had had obstruction. When these 21 patients are added to the 56 who were operated on for obstruction and 14 who were operated on for hemorrhage, the number of patients who had had one or more of the major complications of ulcer totals 91 (63%). The 14 patients who were operated on because of hemorrhage had surgery during a quiescent interval. (Vagus resection with gastroenterostomy is not advisable during active bleeding; if emergency surgery must be done, the procedure of choice is a direct ligation of the bleeding vessels in the ulcer bed and a subtotal gastric resection.) The two patients in whom operation was undertaken for suspected neoplasm had roentgenographic evidence suggestive of gastric carcinoma; the duodenal ulcer in these two was disclosed at operation.

Table 3.—*Indications for Operation*

Principal Reason for Surgery	No. of Patients	Other Complications, No. of Patients			
		Weight Loss of 10 Lb. (4.5 Kg.)	Previous History of		
			Hemorrhage	Obstruction	Perforation
Intractability	75	6	11	2	8
Obstruction	56	22	15	..	5
Hemorrhage	14	2	..	1	0
Neoplasm (?)	2	1	0	0	0

OPERATIVE PROCEDURE

The operation carried out on these patients was a subdiaphragmatic division of the vagus nerves (segments always being sent to the pathologist for subsequent microscopic verification), together with any accessory fibers that could be found, and a short loop posterior gastroenterostomy. During the period covered by this report other operative procedures were performed for duodenal ulcer, but these are not included in this analysis. The largest group comprised some 50 patients who had vagotomy and a pyloroplasty instead of a gastroenterostomy. There were a few who had a gastric resection with the vagotomy, several who had only a vagotomy without an accompanying drainage operation, and three instances of unilateral vagotomy among the earliest cases in which a posterior branch of the vagus was not found.

In a series of 397 patients undergoing the procedure through Oct. 1, 1951, there were two operative deaths, a mortality rate of 0.5%. There have been no deaths in the last 159 patients. It would seem that operative mortality should not exceed 1%.

POSTOPERATIVE DIARRHEA

It is difficult to ascertain the significance of diarrhea from answers to the questionnaires (table 4). In only four patients was the diarrhea a troublesome daily event, and in at least one of these patients diarrhea had existed before the operation.

RESULTS

It is worth while noting that the routine management of these patients after operation did not include antacids or antispasmodics. After six weeks on a bland diet, the patient is told he may eat when he pleases and what he pleases, and smoking or drinking in moderation is permitted. This is in sharp contrast to the rigid ulcer schedules employed by some surgeons after a gastric resection for duodenal ulcer.

Very strict criteria were employed in the appraisal (see figure). An "excellent" result means that the patient has no gastrointestinal complaints, takes no medicaments, follows no dietary restrictions, pursues his regular occupation, and himself regards the operation as an unqualified success. In this classification there were 101 patients (69%). Nine of these patients recorded a very occasional, mild diarrhea, which required no treatment.

TABLE 4.—*Incidence of Diarrhea After Operation*

Degree Diarrhea		No. of Patients
Not troublesome		16
Infrequent and mild	11	
Daily and mild	5	
Troublesome		14
Infrequent	10	
Daily	4	
Transient (in hospital only)		3
Total		33 (22% of 147)

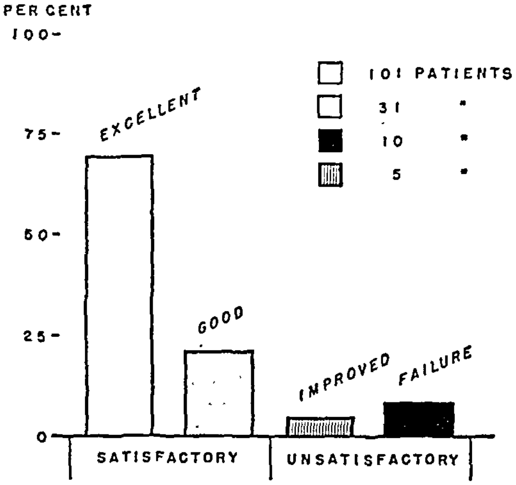
A "good" result was one in which the patient had occasional vague digestive complaints, such as gas, bloating, or upper abdominal distress. Ulcer pain was not present. Regular medication was not needed, and the usual occupation could be followed. Some of these patients undoubtedly suffer from the irritable colon syndrome, and some are mild neurotics who would never be in excellent health under any circumstances. All these patients regarded themselves as improved by the operation, and all but a few were completely satisfied. When "good" results are added to the "excellent" results, there are 132 patients, or 90%, in whom the operation may be termed successful.

Five patients continue to have partially incapacitating gastrointestinal disturbances, but so far as we could determine there was no clinical evidence of recurrent ulceration. These were classified as "improved." Psychic factors seemed to play a large role in the unsatisfactory result.

There are 10 patients in whom treatment failed. Four of these patients had subsequent operations, resulting in the relief of symptoms in two; the condition in four of the remaining six is satisfactorily controlled with a medical regimen. There are thus four whose present condition as it relates to the ulcer is unsatisfactory. Failure was felt to be due to a mechanically imperfect stoma in three, to an incomplete vagotomy in one, to the dumping syndrome in one, and to severe psychoneurosis in one; in four instances, the reason was not clear. In 6 of these 10 patients, the presence of a marginal ulcer was proved roentgenographically, was seen at a secondary operation, or could be inferred from the clinical course.

COMMENT

Although gastric resection has proved of value in the treatment of duodenal ulcer, the continued search by surgeons for alternative procedures is evidence that it is not a perfect solution. The principal objections to gastric resection are two: 1. It is an intrinsically hazardous procedure, which in the hands of master surgeons still carries a mortality rate of 2% to 4%. The mortality in the hands of the average surgeon can only be conjectured, but it is certainly much higher. 2. Many patients who have had two-thirds or more of the stomach removed cannot be considered "normal" from a digestive standpoint. While the patients are free from ulcer symptoms and the complications of ulcer, the majority of careful investigators find that, after gastrectomy, a substantial proportion of patients fail to regain their normal weight and often suffer in some degree from "stomach consciousness," anemia, malnutrition, or the dump syndrome, or must follow a restricted diet. The fact that these discomforts usually are insignificant in comparison with the symptoms for which the patient had his operation may lead both surgeon and patient to treat them lightly. The surgeon performing a gastric resection for duodenal ulcer still faces a delicate problem: the less stomach he removes, the more likely is recurrence of ulceration; while the more stomach he removes, the more likely is the occurrence of other sequelae.



Summary of results of surgical treatment of duodenal ulcer in 147 patients.

The initial appeal of vagus resection was tremendous, since it appeared to answer both the objections to gastric resection, in offering a substantially lower mortality rate and in leaving intact the entire digestive tract. As so often occurs with new procedures, time was necessary to establish certain shortcomings. It is now clear that transthoracic vagotomy has at best a very limited usefulness, since it prevents an accurate operative diagnosis of gastroduodenal pathological changes and also results in a high incidence of persistent gastric retention. It has likewise become clear that vagus resection has no place in the surgical treatment of gastric ulcer (in which gastric resection is almost invariably indicated) or in the treatment of gastritis or gastric neuroses. It may also be regarded as established that, in the treatment of duodenal ulcer, a vagus resection should not be performed without an accompanying drainage operation. Yet, failures resulting from the practices noted above are still cited as evidence that vagotomy has no place in the treatment of duodenal ulcer. Some authors lump together gastric ulcer, duodenal ulcer, and marginal ulcer as if they were the same disease and may even confuse the issue further by failing to separate the procedures done with the vagotomy, such as gastroenterostomy, pyloroplasty, or gastric resection. When the data are broken down to cases of a specific lesion (i. e., duodenal ulcer) for which a specific operation was performed (i. e., vagotomy with gastroenterostomy) the number of failures often becomes too small to be of significance.

We feel that some of the surgeons currently condemning vagotomy with gastroenterostomy on the basis of personal experience may have used the operation in a selective way without realizing the consequences of such selection. For example, a surgeon who finds little or no evidence of an ulcer at operation is usually unwilling to perform a gastric resection but may decide to perform a vagotomy with gastroenterostomy rather than do nothing. Results in such borderline cases are almost always poor, since the symptoms are usually due to some other condition. If the surgeon habitually performs a gastric resection for a real pathological condition, and vagotomy for a questionable pathological condition, he is bound to be disappointed with his results from vagotomy.

Many experienced surgeons who regard gastric resection as the procedure of choice contented themselves in the prevagotomy days with performing a simple gastroenterostomy for duodenal ulcer in patients in whom old age, obesity, serious coexistent disease, or technical difficulties rendered gastrectomy unduly hazardous. Under similar circumstances some now add vagotomy to the gastroenterostomy. This is undoubtedly a sound and conservative surgical practice and should yield good clinical results, but it may produce misleading statistics and a deceptively low mortality rate for gastric resection. More studies of consecutive series of patients surgically treated for duodenal ulcers are needed for proper evaluation of the two procedures.

Finally, it is certain that many surgeons have been discouraged by a high incidence of persistent gastric retention following vagotomy with gastroenterostomy. We feel that certain technical details in the placement and execution of the gastroenterostomy have great bearing on this point, as emphasized by one of us (G. C.).[4] These details have received insufficient emphasis in the literature. We shall give this matter further attention in a subsequent publication. In our hands, the supplementary gastroenterostomy has proved superior to a pyloroplasty.

Bearing in mind the possibility that a longer period of observation may reveal flaws in the operation not now apparent, we believe that our experience of 90% satisfactory results justifies the policy of continuing to perform vagus resection with gastroenterostomy as the operation of choice for duodenal ulcer. We feel that the operation has no place in the treatment of gastric ulcer or in patients who have no demonstrable pathological condition at the time of surgery. We recognize that failures will occur and that some patients will require a subsequent gastric resection. It is possible that careful study of the failures will reveal some as yet unrecognized clinical features, enabling prediction of failure from vagus resection and permitting selection of the small group of patients in whom a gastric resection should be done as the primary procedure.

SUMMARY

The results in 147 patients treated for duodenal ulcer by subdiaphragmatic vagus resection with gastroenterostomy is reported after a follow-up of two to four years. Seven patients were not traced; thus the follow-up is 95% complete. In 90% of the patients, the operation has produced a satisfactory clinical result. In 10%, the results have been unsatisfactory, including failure in 6.8%. During the period studied, approximately 13.5% of patients treated for duodenal ulcer in the Cleveland Clinic Hospital underwent operation. In approximately half the patients included in this report, surgery was performed for one or more of the major complications of duodenal ulcer.

The operation is not advocated for gastric ulcer (for which gastric resection is the procedure of choice) nor for patients who do not show clear-cut evidence of ulcer at the time of operation. It should not be performed as an emergency procedure for active hemorrhage. In view of the good clinical results and a low operative mortality (less than 1%), further employment of the procedure seems warranted.

2020 E. 93rd St.

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[4] Crile, G., Jr.: Technique of Vagotomy and Gastroenterostomy in the Treatment of Duodenal Ulcer, Surg., Gynec. & Obst. 92:309 (March) 1951.

Diagnostic Significance of 17-Ketosteroids.—The urinary 17-ketosteroids may be determined simply and accurately in the laboratory. They reflect the secretory activity of the testes and the adrenal cortex. The values obtained appear to be of little diagnostic significance in testicular hypofunction, but may be of considerable assistance in ascertaining the state of the adrenal cortex. However, an abnormally low value for these steroids is of no diagnostic significance in itself, since there is a lowered 17-ketosteroid excretion in many nonspecific chronic diseases. In those conditions with an intact adrenal cortex, the administration of ACTH over a period of time will cause an elevation in these values.

In the adrenogenital syndrome this laboratory determination is of greatest diagnostic importance. The 17-ketosteroids will not only assist in making the diagnosis of adrenal virilism, but may be of help in determining the nature of the underlying adrenal pathology, whether it be hyperplasia, adenoma or carcinoma. If a neoplasm is present, surgical extirpation is indicated. If hyperplasia is present, the administration of cortisone appears to be the treatment of choice at the present time. The success or failure of therapy can be easily gauged by following the urinary 17-ketosteroid values.—Joseph W. Jailer, M.D., Diagnostic Significance of the 17-Ketosteroids, *The Medical Clinics of North America*, May, 1952.

COORDINATED MEDICAL PROGRAM FOR DOMICILIARY PATIENTS IN A VETERANS HOSPITAL

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and
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The Veterans Administration is at present furnishing medical care to approximately 17,000 chronically ill veterans in 15 domiciliary units in the United States. The largest of these domiciliary units has a capacity of 3,074 veterans and comprises part of the Los Angeles Veterans Administration Center. A 1,400 bed general medical and surgical hospital and a 2,000 bed neuropsychiatric hospital complete the medical facilities of the center. Both hospitals function under the Veterans Administration Deans' Committee Program.

MEDICAL PROGRAM

During the past two years, a coordinated medical program has been established in the domiciliary unit under the supervision of the chief of professional services of the general medical and surgical hospital. The purpose of this program has been to improve the medical care given the 3,074 chronically ill, aging veterans residing in the domiciliary unit by (*a*) critically evaluating their medical, psychological, and sociologic problems, (*b*) returning as many as possible to a more useful and relatively more normal life in the outside community, (*c*) providing those remaining in the domiciliary unit with an integrated therapeutic regimen and medical follow-up, (*d*) training physicians in clinical geriatrics, and (*e*) providing opportunities for investigative studies of a well-controlled aging population.

The backbone of this program has been a general practitioner system, in which each of the eight 400-bed buildings operates as a modified hospital wing and is manned by a team consisting of a full-time physician, a part-time physician, a registered nurse, and a medical secretary. The ultimate responsibility for the patient's complete medical care rests with the full-time general practitioner and his part-time associate. The nurse's duties combine those of a public health nurse with those of an office nurse. Specialistic examinations are obtained from a domiciliary consultation service, called a diagnostic clinic. This consists of four full-time certified internists, aided by three full-time medical residents, and functions under the Deans' Committee Program. With the aid of medical and dental personnel from the nearby general medical and surgical hospital, the diagnostic clinic provides a consultation service covering all the medical and surgical specialties. The departments of physical medicine and social service have an indispensable role in the medical program.

A mental hygiene clinic has been established because of the high incidence of psychological problems among the domiciliary patients. It is staffed by two psychiatrists, two clinical psychologists, and five clinical psychology trainees, with one psychiatric social worker. This clinic has as its attending staff the chief of professional services of the adjacent neuropsychiatric hospital and his clinical psychologist. Consultation service is being arranged with the staff of the nearby University of California at Los Angeles medical school.

When hospitalization is necessary, continuance of the doctor-patient relationship essential to the general practitioner system of medicine is made possible through the facilities of a 30 bed ward set up for this purpose. The general practitioner retains responsibility for his own patients. Consultation is obtained, when needed, from the domiciliary consultation service.

A comprehensive screening procedure designed to aid in the rehabilitation of all new domiciliary members has been functioning since April, 1951. The veteran, having been found eligible for admission to the domiciliary unit, is thoroughly evaluated medically by the domiciliary consultation service. This evaluation includes the taking of a complete history and a thorough physical examination, with the use of any indicated laboratory procedures. A similarly thorough psychological, vocational, educational, and social evaluation of the veteran then follows. The veteran's composite picture is completed with an evaluation by the department of physical medicine. The information obtained by this screening procedure forms the basis of a rehabilitation program individually tailored to the needs of each veteran. The rehabilitated veteran is returned to a more useful life either in the outside community or in the domiciliary unit.

1950 DOMICILIARY POPULATION

The mean age of the 3,864 domiciliary patients examined at this center in 1950 was 61 years. Of the total group, 53% were in the age group 55 to 64, while only 4% were under 45, and 9% were 75 or over (*fig. 1*). Analysis of morbidity among the entire group revealed that diseases of the circulatory system were far commoner than any other disease entity (fig. 2). Arterial disease, hypertension, and arteriosclerotic heart disease were the conditions observed in most of the patients in this category. The next commonest group was diseases of the nervous system and sense organs, closely followed by diseases of the musculoskeletal system and the respiratory system. Syphilis occurred in 500 patients; in 248 of these, the central nervous system was affected, and in 51 the

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Reviewed by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions of the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

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cardiovascular system was affected. Malignant neoplasms were found in 200 patients and benign neoplasms in 65. Diabetes mellitus occurred in 172 patients.

In patients under age 45, diseases of the nervous system and psychogenic disorders were the most prevalent, both occurring in almost 50% of the patients in this group. In those over 45, diseases of the circulatory system were the leading causes of disability, occurring in about 75% of the patients and assuming increasing importance with increasing age. Multiple cardiovascular diseases were most prevalent in the older age groups. At all ages diseases of the nervous and musculoskeletal systems played a prominent role. Diseases of the digestive and respiratory systems were present in 25% of the patients over age 45 and seldom occurred in patients below that age. The incidence rate of malignant disease increased from 1 per 100 patients under age 45 to 9 per 100 patients over age 65.

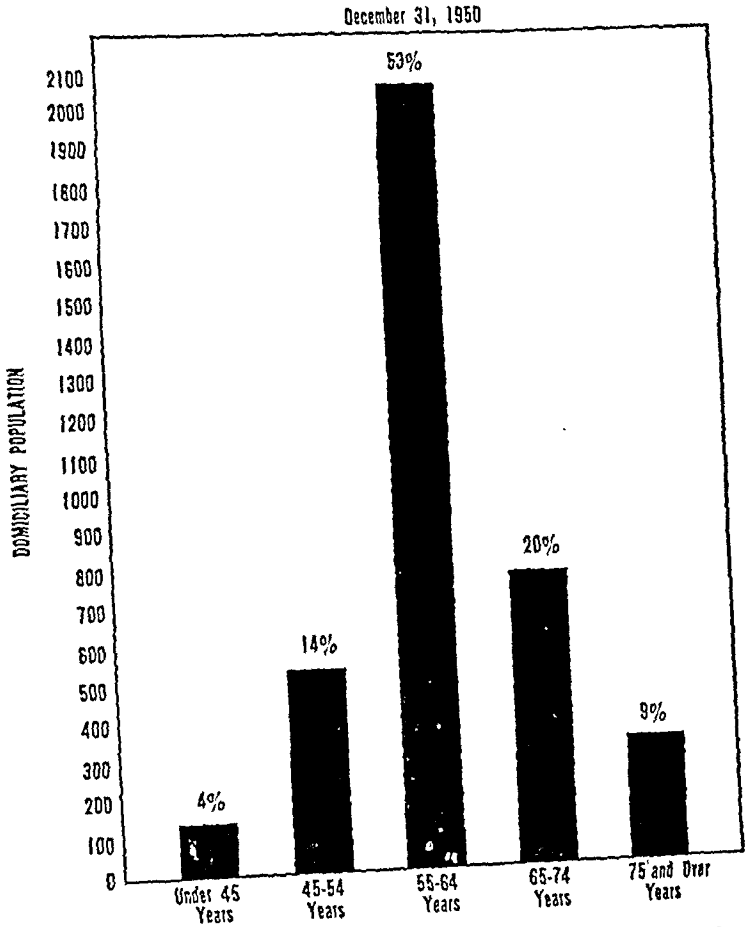

Fig. 1.—Distribution of 1950 domiciliary population by age groups.

Two hundred sixteen deaths occurred in the domiciliary unit in 1950. Diseases of the circulatory system, the leading cause of illness in patients over age 45, was also the leading cause of death, being responsible for 57% of the deaths in this age group. Malignant disease, while not a leading cause of illness, was the second leading cause of death in patients over 45, being responsible for 16% of the deaths in this age group. Two deaths occurred in patients under age 45, both due to urologic disease.

RESULTS OF THE PROGRAM

In 1948, admissions of domiciliary patients to the general medical and surgical hospital for surgical treatment averaged 87 patients for each three month period. The corresponding figure for patients admitted to the hospital for medical care was 213. Shortly after the medical program was started in July, 1949, a sharp and significant rise occurred in the number of patients admitted for surgical treatment. In most cases these were short term admissions for elective surgical procedures. The peak occurred early in 1950, at which time 52% more domiciliary patients were receiving surgical treatment than in 1948. We feel that this was due to the careful medical and surgical evaluation the patients received and to the fact that chronological age alone was not considered a deterrent for elective surgical treatment. No significant effect on the number of patients admitted for medical care was apparent until one year after the program had been in operation. Since then, there has been a steady increase in such admissions, and by early 1951, a decrease of 60% had occurred (fig. 3).

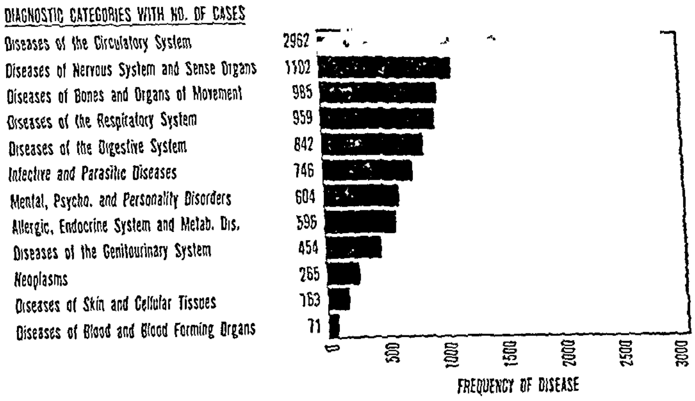

Fig. 2.—Incidence of selected diseases among the 3,864 domiciliary members in 1950. (Includes all disease conditions diagnosed and recorded as first, second, and third diagnoses.)

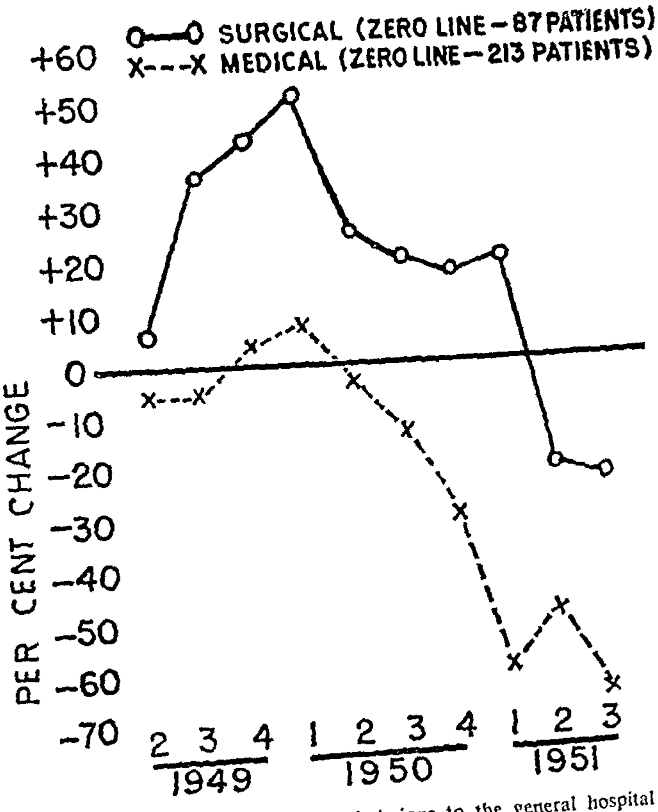

Fig. 3.—Percentage change in admissions to the general hospital since inception of the new medical program.

The rehabilitation program has been in effect too short a time for proper evaluation of the available data; however, only 10 (8%) of the first group of 121 newly admitted patients were considered unsuitable for rehabilitation. Twenty-eight patients (23%) of this group have been rehabilitated to a more useful life in the outside community or in the domiciliary. The remainder have not yet completed the prescribed therapeutic regimen.

COMMENT

The problem of chronic illness in an aging population has become a major one for the Veterans Administration. As the country's veteran population becomes older, it is expected that this problem will increase in importance. The high incidence of disabling organic disease found in this study illustrates the need for a coordinated medical program. It is recognized that medical care in the older age groups must include not only therapy for the patient's disease but proper attention to his psychological, sociologic, and economic problems. During this study, it became evident that not only must the influence of psychological and functional states on organic disease be evaluated, but similar stress must be placed on the influence of organic states on the patient's mental outlook. Both are influenced by the patient's home environment. The effect on the patient of a protected environment, such as is available in the domiciliary, needs further clarification.

The practice of detection medicine by periodic physical examination of the geriatric patient has resulted in earlier diagnosis and treatment of such life-threatening diseases as cancer and tuberculosis. The increase in the practice of surgical correction of partially disabling abnormalities has had similarly favorable results. We believe this program has resulted in improvement in the physical well-being and mental outlook of the patients. This is in part demonstrated by the decrease in medical admissions to the general medical and surgical hospital (fig. 3), with resultant increase in the availability of the hospital's facilities. A large contribution has been made by a program of occupational therapy, which is designed to provide a more useful life for those patients remaining in the domiciliary.

The life expectancy of the veterans analyzed in this study was estimated, using the experience of the white male population in the United States in comparable age groups[1] (see table). Based on the average 1950 per diem rate in all Veterans Administration domiciliary units, it is estimated that an expenditure of at least 55 million dollars for food, clothing, and shelter would be necessary for the future maintenance of these veterans if they spent the rest of their lives in the domiciliary. If a percentage of them can be rehabilitated to a more useful life in the outside community, much of this cost could be defrayed and existing facilities utilized to care for the increasing number of veterans seeking admission to the domiciliary.

Life Expectation in 1950 of Domiciliary Patients, According to Age Groups, and Prospective Cost of Their Domiciliary Care Until Death

Age Group	No. of Patients	Total Years of Life Expectation	Aggregate No. Days Future Care Required	Total Estimated Cost (Rate $3.04/Day*)
Under 45	145	4,438.3	1,621,089	$ 4,928,110.56
45 to 54........	516	9,575.9	3,497,597	10,632,694.88
55 to 64........	1,972	27,314.2	9,976,512	30,328,596.48
65 to 74........	708	6,505.8	2,376,243	7,223,778.72
75 and over...	397	1,619.5	602,068	1,830,286.72
Total, all ages	3,648	49,483.7	18,073,509	$54,943,467.36

* Average 1950 per diem rate in all Veterans Administration domiciliary units.

SUMMARY

1. A coordinated medical program has been instituted in the Los Angeles Veterans Administration Center domiciliary unit, which is designed to meet the problem of chronic illness in an aging veteran population.
2. Better total evaluation and care of the domiciliary patient has been made possible.
3. This program has resulted in improvement in the health, morale, and usefulness of these geriatric patients.

1. Greville, T. N. E.: United States Life Tables and Actuarial Tables, 1939-1941, United States Department of Commerce, Bureau of the Census.

BRAIN FEVER

Louis Casamajor, M.D., New York

In the early years of the 19th century there began to appear in European medical journals—especially the French—descriptions of a disease variously known as brain fever, hydrocephalic fever, and, rarely, encephalitis.[1] At times the name of Pinel is attached to brain fever, and it may be that he originally described it. In any event he is credited with having differentiated it from meningitis. By the middle of the century no further medical articles appeared, although novelists continued to utilize the disease as dramatic incident until well along toward the end of the century. Early American textbooks of neurology contained no reference to the disease.

This disease occurred apparently almost entirely in children. It is difficult to get a clear idea of the clinical picture, for more than one condition was included in this terminology. Some cases were fatal, and the necropsies usually showed meningitis, most likely tubercular. Some patients recovered completely and must have had an entirely different disease. The symptoms recorded are entirely subjective and are those recognized today as indicative of cerebral involvement. The onset was usually sudden, but some were step-like. Often the first symptom was a convulsion; then were added headache, vomiting, coma or delirium, strabismus, aphonia, dyspnea, and in-

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From the Neurological Institute of New York and the Department of Neurology, College of Physicians and Surgeons, Columbia University.

[1] (*a*) Cloquet, H.: Quelques idées sur la fièvre hydrocephalique, ou cérébrale des enfans, Nouv. J. méd., chir., pharm. **1**: 1921, 1815. (*b*) Houdaille, M.: Observations d'une fièvre pernicieuse cérébrale guerie par le quinquina, Nouv. J. méd., chir., pharm. **3**: 30, 1818. (*c*) Mareschal, M.: Quinquina administré das la fièvre hydrocephalique des enfans, Nouv. J. méd., chir., pharm. **4**: 298, 1819. (*d*) Mazet, A.: Histoire de deux fièvres cérébrales chez deux enfans, l'un de cinq ans, et l'autre de trois ans, Nouv. J. méd., chir., pharm. **10**: 17, 1821. (*e*) Brizard, J.: Essai sur l'encéphalite ou fièvre cérébrale (Pinel), These de Paris, 1830, vol. 4. (*f*) Liégard, A. A. L.: Considérations pratiques sur la nature et le traitement de la fièvre cérébrale ou encéphalo-méningite, These de Paris, 1854, vol. 13.

termittent pulse. After a few days to a couple of weeks, the disease subsided and the patients recovered.

Although fever is mentioned in all the reports, there is no record of any temperatures, for temperatures in illness were not taken until after the invention of the modern clinical thermometer, in 1868.[2] No objective neurological signs appear in the reports, because the neurological examination as we know it today did not begin to develop until after Erb [3] described the changes in the tendon reflexes in spinal cord disease and Westphal [4] the loss of knee jerks in tabes dorsalis, both in 1875.

The treatment in these cases of brain fever was that of the times. Bleeding was freely used. The patients were given wine and brandy in different doses. Fantastic enemas consisting of wine, absinthe, asafedita and similar agents were given, which the colons promptly expelled. Some authors, notably Houdaille,[1b] expressed the belief that quinine was specific for the disease. Liégard [1f] recommended antimony inunctions to the scalp till pustules formed. Even with these treatments, apparently most of the patients recovered.

After about 1850 the disease disappeared from the literature. This was probably similar to the way in which the "encephalitis lethargica" of von Economo has disappeared from medical practice. During the 1920's and early 1930's epidemic lethargic encephalitis, with its acute phase and the long chronic stage of parkinsonism, was a very prevalent disease throughout the civilized world. The reports of the Matheson Commission abstract an enormous literature on the subject; yet today one no longer sees cases of the acute disease. One still sees some patients in the chronic stage, but the acute disease is no longer a problem. In a somewhat similar manner brain fever disappeared from the medical literature 100 years ago, but it has not disappeared from medical practice. Undoubtedly the condition does exist today.

REPORT OF CASES

CASE 1.—A. M. was 11 years of age when she entered the Neurological Institute, on Oct. 5, 1935. She was the fifth of eight children, all of whom were normal. Her development had been normal; she was considered to be a friendly, healthy girl whose interests were normal, and she was getting along well in school.

About a month previously her mother noticed a change in her. She "lost interest in things and moped around the house." She was sent to visit an aunt, and there she refused to play with other children or to leave the house. In the week she was away from her home nocturnal enuresis occurred on four occasions, which was the first time that had happened since she was 18 months of age.

On her return home she seemed to be "a different child." She frequently became angry with other children and "acted babyish." She did not want to go to school or leave the house for any other reason. At times, however, she would be very active, helping her mother with the housework and caring for the younger children.

On the evening of Sept. 28 she had her first seizure, involving the right side of her face. She was unable to talk but was conscious and responded to questions with head nodding. After being put to bed she cried all night. On the following morning she appeared to be normal, went to church, and played till late in the afternoon, when she again became irritable.

From this time on she continued having two or three attacks of right facial twitching, without loss of consciousness, with increasing frequency until on the day of admission they were recurring every few minutes. When we first saw her she was having seizures frequently. Most of them were only in the right side of the face, but some spread to involve the right arm and leg. The neurological examination revealed nothing essentially abnormal, but there was a right facial weakness of the central type and the left optic disk appeared blurred. All the deep and superficial reflexes were active and equal on both sides. Between seizures there was no difference in the functions of either arm or leg. The blood cell count, blood chemical studies, and serologic studies disclosed no abnormalities. Examination of the spinal fluid showed five cells and 22 mg. of protein per 100 cc. Later lumbar punctures showed no higher cell count or protein content.

As the child remained on the ward her condition became worse. Seizures on the right side and sometimes of the whole body occurred as often as every minute. Sometimes the seizures were of a psychomotor sort, in which she would shout and scream, beat her head against the wall, tear her clothes, and throw things around the room.

The neurological findings continued as above. On Oct. 10 an ophthalmologist reported increased congestion of nerve heads, with hazier margins. The large and small veins show increased engorgement. On Oct. 15 a pneumoencephalogram showed no significant abnormality, although the ventricles were poorly filled. The right-sided convulsions were still practically continual. On Oct. 29 the right side of the face and the right arm were paralyzed. The child understood speech but was unable to talk. Occasionally her temperature rose to 102 F.

On Oct. 30 improvement was obvious. Her seizures became less severe and less frequent. She could talk and move her right arm, although it was somewhat unsteady and ataxic. The last seizure, a completely right-sided one, occurred on Nov. 5. The right arm after this was ataxic for a few days and showed some involuntary movements. She could not yet talk plainly, and she showed some confusion at times. The results of neurological examination on Nov. 11 were reported as absolutely normal.

When the patient was discharged from the hospital, on Nov. 28, 1935, she seemed normal in every respect. Neurological observations remained normal. There have been no further seizures, and she has received no anticonvulsive medication. She returned to school and, at 14 years of age, was graduated from grade school as fourth in her class, with an average of 92%. In high school she was on the honor roll and was graduated at 18 years. Since then she has held a clerical job. In January, 1946, she married; when last seen, on July 26, 1951, at the age of 27, she appeared to be a healthy, happy, and intelligent woman.

Comment.—This can be taken as a typical case of brain fever. Neurological symptoms and signs are indicative of the location rather than the nature of the brain disease. The spread of the disease is also not always the same. In some cases the disease seems to be restricted to the cerebral cortex and in others to the base and stem of the brain.

CASE 2.—B. W. was 6½ years old when she was admitted to the Neurological Institute, on April 4, 1942. She was the second of two children, a bright girl, and doing well in school. She spoke both English and Polish and acted as interpreter fo her parents. She was well until the middle of March, 1942, whe she began having irregular temperature increases, called "septic." She was kept in bed with conservative treatment. On Marc 30 she complained that everything seemed dim, and in two hou she was completely blind. She was taken to a local hospital an it was reported that she had bilateral papilledema, leukocytos (48,000 with 93% polymorphonuclear cells), increased sedime tation rate, and normal spinal fluid.

On admission to the Neurological Institute she appeared be completely blind and could not stand alone. Her pupils d not react to light; the optic disks were definitely blurred, b the vessels appeared normal. When she attempted to stand alo she fell backward, but none of the other neurological signs were present. The choked disks were considered to be due to retrobulbar neuritis.

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[2] Woodhead, G. S., and Varrier-Jones, P. C.: Investigations on Clinical Thermometers: Continuous and Quasi-Continuous Temperature Records in Man and Animals in Health and Disease, Lancet 1: 173, 1916.

[3] Erb, W. H.: Ueber Sehnenreflexe bei Gesunden und bei Rueckenmarkskranken, Arch. f. Psychiat. 5: 792, 1875.

[4] Westphal, C. F.: Ueber einige Bewegungs Erscheinungen an Gelaemten Gliedern, Arch. f. Psychiat. 5: 803, 1875.

For days she had a low-grade fever, with temperatures between 99 and 100 F. The leukocyte count was 28,000, with 80% polymorphonuclear cells. The count rose to 31,000 and then gradually diminished, but it remained between 13,000 and 20,000 during the months of April and May. By June 8, when she was well on the road to recovery, her leukocyte count was 8,700, with 69% polymorphonuclear cells.

Examination of the spinal fluid on admission showed 30 cells (48% polymorphonuclear cells and 52% lymphocytes) and 43 mg. of protein per 100 cc. This rose as high as 50 mg. per 100 cc. and then decreased, together with the cell count, which was down to 8 on April 28. Dr. Lester Webster, who had organized a laboratory for virus studies in the Rockefeller Institute, examined the blood and spinal fluid of this child at the height of her illness. He found no complement fixation against antigen of any of the known encephalitis viruses, and the spinal fluid proved negative when tested in mice.

At first the child was apathetic and content to lie quietly in bed, but she always cooperated well in the examinations. The blindness persisted, and the eyegrounds remained unchanged. At times she showed a right facial weakness and inability to stand alone, which was considered cerebellar in origin. No real change occurred until April 25, three weeks after admission. On that day the right facial palsy became more marked, her speech was nasal, and she had difficulty in swallowing. These symptoms disappeared after about 15 minutes but recurred together with irregular respiration a number of times during the day.

On April 26, her breathing became purely abdominal and jerky and she was in coma. When this episode passed, her speech was thick and nasal and her deep reflexes were hyperactive but equal. Later a questionable Babinski toe sign was noted on the right. That evening her respiration ceased and she was comatose, with contracted pupils. When placed in a respirator she responded immediately; she remained there for five days.

When the patient came out of the respirator, the picture was one of complete cerebellar asynergia but her vision had improved so that she could count fingers at 5 ft. and recognize colors. Not only was she unable to stand erect but she could not even crawl. When she tried to progress on hands and knees, her hips tended to fall over to the left. The right facial palsy persisted.

This was the first case in which electroencephalographic records were available. Three days after admission the electroencephalogram showed diffuse high voltage one to two per second waves in all leads, most consistently in the occipital areas. Bipolar recording showed widespread phase reversals in all areas. These findings did not change all the time she was hospitalized.

By early June the patient had fully recovered and was considered normal but possibly a little overactive. She was discharged on June 18 and since then has led a normal life. She never had any convulsions. Her progress in school was what one would expect of a child of her background. When last seen in the clinic, in September, 1951, at 15 years of age, the girl appeared normal and the results of the neurological examination were entirely negative.

Comment.—This child had all the signs of severe cerebral involvement except convulsions. This could have been because mainly the base of the brain and the hind brain were involved. At the start the spinal fluid showed signs of meningeal involvement, so that the condition could be called meningoencephalitis. Meningeal signs have been rare in this series of cases, but in the next case they were more prominent.

Case 3.—G. S. was nearly 7 years of age when she was admitted to the Neurological Institute, having been transferred from Babies Hospital, on Feb. 18, 1949. She was an attractive little girl who had always been considered bright. She had always been well until in July, 1948, at the age of 6½ years, when she began to complain occasionally of brief frontal headaches and easy fatigability. This condition continued intermittently for the next five months and did not seem to be getting worse.

On Christmas day, 1948, she suddenly stiffened and became confused. She was unable to walk downstairs and had to be carried. Later in the day she completely recoverd her ability to walk. She remained well until the night of Jan. 9, 1949, when she slept poorly and had a headache. In the morning she had a temperature of 102 F and was drowsy. She continued to be ill, and, as she seemed to be getting worse, she was removed to the Physicians Hospital, Jackson Heights, N. Y.

A neurological examination on Feb. 2 was reported as showing neck rigidity, Kernig's sign on the left side, severe headache, right central facial weakness, hazy optic disks, general hyperreflexes, and a positive Babinski sign on the right. Spinal fluid examination on Jan. 16 showed 53 cells (85% lymphocytes) and 41 mg. of protein per 100 cc. On Jan. 25 the cells had increased to 110, with 62% polymorphonuclear cells, and the protein had increased to 54 mg. per 100 cc.

On Feb. 5 she was admitted to Babies Hospital, with stiffness of the neck, a positive Brudzinski sign, bilateral transient ankle and patellar clonus, blurred optic disks and occasional right facial weakness.

On Feb. 18, when she was transferred to the Neurological Institute, she could support her weight on her feet, but her balance was very poor. The pupils were normal, and the optic disks were blurred, with small arteries and veins. There was slight right facial weakness. Arm reflexes were equal, but leg reflexes were greater on the right. There was no Babinski sign. Spinal fluid examination showed 16 cells, all lymphocytes, and 39 mg. of protein per 100 cc., which later increased to 46 mg. The blood leukocyte count was 5,400 (68 polymorphonuclear cells and 24 lymphocytes). Temperature ranged from 99 to 101.8 F. An electroencephalogram on Feb. 10 was grossly abnormal, with high voltage slow activity, two to four waves per second. This activity appeared focal in the two frontal areas and a large area of the left temporal and occipital. A pneumoencephalogram on Feb. 25 was reported as essentially negative.

The patient's condition improved steadily from the time of admission. By March 2 her gait was practically normal, and there was no facial weakness. All reflexes were active and equal, and optic disks were normal. On March 12 the electroencephalogram still showed partly high voltage three to four per second waves in both parietal and occipital areas but no longer in frontal areas.

On March 13 the patient was discharged from the hospital; she was followed in the clinic. She returned to school, where her progress was normal. The last electroencephalogram, made on Feb. 2, 1950, showed no focal signs, and only on hyperventilation were occasional high voltage bursts of two to three per second waves seen. She remained well, with no abnormal neurological findings. She was last seen on Oct. 3, 1951.

Comment.—In the last two cases the condition was rather severe, with meningeal as well as cerebral signs. In not all the cases is the disease so severe; in some it is milder and of much shorter duration. This is especially true in some of the cases in which convulsions occur.

Case 4.—M. S. was nearly 8 years of age when she was admitted to the Neurological Institute, on June 16, 1950. She was an alert girl, with an I.Q. of 94. On June 10, 1950, she was running down a hill with her 6-year-old brother when they collided and she struck the left side of her head against her brother's. There were no immediate symptoms from the blow, which apparently was a minor one. That night she awoke several times complaining of left-sided headache. The following morning she vomited once and ate very little, but in the afternoon she went outdoors and played normally. She was heard to cry intermittently during the following night. On June 12 and 13 she vomited several times and complained of left-sided headache.

On June 14 she complained of dizziness and did not seem to be able to walk. When she was taken to a hospital for skull x-ray that morning, her father had to carry her. The x-ray was reported as negative. On June 15 she was much improved but still complained of left-sided headache. There was some stiffness of the neck, and her left eyelid drooped.

On the morning of June 16 she awakened early and shortly thereafter was found unconscious in bed, with her head and eyes turned to the right and the eyelids open. She was then taken to St. Vincents Hospital, Staten Island, where she had several generalized seizures, recovering consciousness in the intervals. That day she was transferred to the Neurological Institute.

On admission she was drowsy, with considerable weakness of the right arm and leg. All deep tendon reflexes were sluggish but equal on the two sides. The leukocyte count was 5,200, with 84% polymorphonuclear cells; her temperature was 99.6 F. Spinal fluid examination showed 7 cells, all lymphocytes, and total protein of 33 mg. per 100 cc. The electroencephalogram showed grossly abnormal activity all over the left hemisphere, while the right was fairly normal. The frequency wave range was one to three per second, of high amplitude with phase reversals all over the left side.

The patient remained hospitalized for 12 days. Her temperature never rose above 100.4 F. She never had another seizure of any sort. On the second morning she was alert and free from headache, but there still was some weakness in the right arm and leg, which disappeared the following day. On June 23 a second electroencephalogram was made. The first part of the record showed a striking improvement, in that now only moderate amounts of low to medium voltage of five per second waves were seen over the left temporal and occipital areas. On hyperventilation some bilateral partly high voltage three to four waves per second were seen.

The patient had no further seizures and received no anticonvulsive medication. She returned to school in the fall and made good progress. An electroencephalogram made on May 5, 1951, showed normal activity throughout but a bit more irregular activity in the left temporal and occipital areas. The neurological observations remained normal. The patient was last seen on Dec. 19, 1951.

COMMENT

These patients presented certain features in common. They were all children. Each had a severe disease with signs of cerebral involvement. The course of the disease was more or less acute and terminated rather rapidly in complete recovery.

It was considered that the conditions in these patients were in the group of the encephalitides. This is clinically a very poorly defined group. Specific viruses have been recognized for five encephalitides. In the only case in this series in which virus studies were made, no known viruses were found. The same was true of other similar cases in this hospital. It is not necessary for one to isolate and recognize any virus or other causative agent in order to make a diagnosis of encephalitis. One must remember that the primary causative factor of epidemic encephalitis of von Economo was never isolated or recognized, in spite of the fact that this was a definite and easily recognizable disease and a great deal of intensive work was done on it. All evidence showed that the cause was a filtrable factor, but nothing more definite was ever discovered.

Encephalitis can be regarded as an acute disease in which the symptoms are those of brain involvement. Frequently the blood and temperature show signs of an inflammatory nature. Sometimes this inflammation appears in the meninges and shows in the spinal fluid. Different parts of the brain may be principally involved, with a resulting difference in clinical pictures. The onset in most of my cases extended over a considerable time, with episodes of abnormal behavior alternating with normality before the real onset of the permanent condition appeared. In case 4, however, the onset was rather sudden, following a slight or only moderately severe injury. In such a case the clinician may justly think of a possible subdural hematoma. The course of the disease, however, excludes this possibility. It may well be that, when a brain is beginning to be affected by encephalitis, even the slightest sort of an injury may precipitate the onset and determine the location of the principal lesion, as in case 4.

The diagnosis is made on the basis of the general and neurological signs. By far the most valuable of these are the electroencephalographic changes. In every case on the children's service the electroencephalogram shows most striking signs—a cerebral dysrhythmia with large irregular waves that may be focal. When the disease is of short duration, the dysrhythmia recedes rapidly (case 4). In long-continued cases this dysrhythmia may be present for many months.

Possibly it is an error to consider the condition in the cases reported here to be the same as that the earlier clinicians called brain fever, but they have many features in common. Brain fever does not seem to be such a poor term to apply to them to indicate a special form of encephalitis.

CONCLUSIONS

Clinicians early in the 19th century reported cases of severe disease of an encephalitic sort under the name of brain fever. Many of their patients, in spite of the severity of the disease, recovered. The condition exists today. In this article four cases of a severe encephalitic disease of the present time and with complete recovery are reported.

Usually the onset is irregular and may extend over a number of months. When slight or moderate head trauma has been sustained, the onset may be rather sudden. The symptoms and signs are those of considerable brain involvement, including convulsions, comas, paralyses, cerebellar asynergy, and a sort of bulbar palsy. In the diagnosis the electroencephalogram is of the greatest value.

168th St. and Ft. Washington Ave.

Neurologic Medicine.—Neurology sprang from internal medicine. Its earlier exponents were internists with a special interest in diseases of the nervous system, many of whom, such as Gowers, Charcot, Strümpell and Pierre Marie, have made notable contributions to subjects other than neurology. . . . The rise of neurosurgery as an independent specialty, the success of syphilology in its own field and the mounting aggressiveness of psychiatry soon encroached on what had been considered the neurologist's field, and by the 1930's there were many who predicted the extinction of the genus neurologist. Some professed neurology as an appendage to neurosurgery or psychiatry—and indeed a few professed all three. Many neurologists and neurologic departments became absorbed by departments of neurosurgery or else adopted protective coloring in the form of the title "neuropsychiatrist" and still exist in unhappy dependence on these borderlands. . . . Neurology, to mean anything at all, must mean neurologic medicine and as such must comprise all diseases that in any way affect the nervous system. Since many more diseases may affect the nervous system secondarily than those that attack it exclusively and since more and more of the latter can be shown to be special aspects of the former, neurologic medicine is quite inseparable from internal medicine. The neurologist must be first an internist. His inspiration is in the general medical wards; his techniques are those of internal medicine.—D. E. Denny-Brown, M.D., The Changing Pattern of Neurologic Medicine, *New England Journal of Medicine,* May 29, 1952.

IMPROVED ABRASIVE BALLOON FOR DIAGNOSIS OF GASTRIC CANCER

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The abrasive gastric balloon[1] technique has assumed a place in the diagnosis of gastric cancer because it secures many gastric cells in a good state of preservation. Because of the abrasive balloon test, lesions of the stomach are no longer inaccessible, and the diagnosis of these lesions becomes not only interesting but rapid and reliable. The purpose of this paper is to advance the procedure for general availability.

APPARATUS

The apparatus needed for the abrasive gastric balloon test is easily assembled. The balloon is a thin latex rubber condom 20 cm. long. The surface of the improved balloon is made abrasive by attaching with rubber cement 75 to 100 small, round, 3 mm. fragments of foamed latex rubber. The fragments of foamed latex rubber are arranged in a regular pattern 1 cm. apart. This balloon system has some advantages over that originally reported in that it is made entirely of latex rubber and has a softer consistency, it is a larger balloon with an increased surface area capable of inflating and filling almost the entire stomach cavity, it has the capacity to yield satisfactory cellular material from the stomach and esophagus, it presents a better psychological appearance, and it has a construction more suitable for manufacture.

The improved abrasive balloon may be applied to the double lumen apparatus previously described if aspiration studies are going to be conducted coincident with the cytological studies. Since controlled comparative studies reveal that diagnostic specimens are obtained from the balloon surface, the abrasive balloon may be applied to a single lumen apparatus without an aspirator (fig. 1). This apparatus consists of a no. 18 French single lumen tube 90 cm. long, to which a one way metal adapter is fitted at either end. To the proximal metal adapter is connected a piece of rubber tubing, 10 cm. long, attached to a rubber inflator bulb with an air control valve. To the distal metal adapter is connected a varying 10 to 20 cm. long piece of no. 18 French single lumen tubing perforated in the center. The distal lumen is closed off with a silver plated blind metal tip. In this way, the distal portion of the apparatus is prepared to accommodate an inflatable abrasive gastric balloon. The abrasive balloon may be used in its full length of 20 cm. or may be shortened to 10 cm. as desired. The abrasive balloon fitted on the apparatus is sealed by silk ties distally around the silver plated blind metal tip and proximally around the one way metal adapter. By means of the rubber bulb air may be introduced through the tube and its perforation to inflate the balloon. In the event that the no. 18 French single lumen tube is not rigid enough for handling, more rigidity may be obtained by employing a thicker no. 20 French single lumen tube or by incorporating a cylindrical coiled wire within the lumen of the tubing. The apparatus finally assembled has markings at 45, 55, 65, and 75 cm. from the distal tip for judging its placement in the stomach.

PROCEDURE

Preparation of the patient revolves around the problem of emptying the stomach as much as possible prior to the introduction of the abrasive gastric balloon, so that minimal displacement of the balloon from the stomach mucosal surface is assured. A liquid diet the day prior to examination followed by an 8 hour fasting period prior to the insertion of the abrasive balloon has given good results. A preliminary gastric analysis to determine the index of gastric acidity will usually give the observer an idea of the retentive property of the stomach. In cases of gastric retention, extra preparation may be accomplished by gastric aspiration, longer periods of liquid ingestion, and additional fasting.

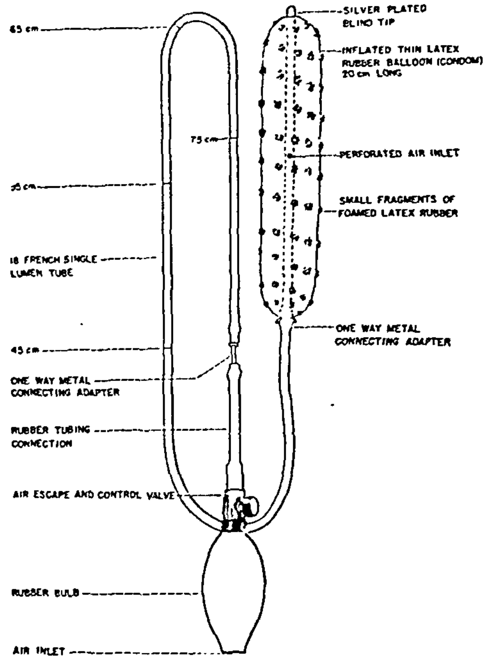
65 cm
75 cm
55 cm
18 FRENCH SINGLE LUMEN TUBE
45 cm
ONE WAY METAL CONNECTING ADAPTER
RUBBER TUBING CONNECTION
AIR ESCAPE AND CONTROL VALVE
RUBBER BULB
AIR INLET
SILVER PLATED BLIND TIP
INFLATED THIN LATEX RUBBER BALLOON (CONDOM) 20 cm LONG
PERFORATED AIR INLET
SMALL FRAGMENTS OF FOAMED LATEX RUBBER
ONE WAY METAL CONNECTING ADAPTER

Fig. 1.—Diagram of the improved abrasive gastric balloon apparatus.

Materials needed for conducting a 15 minute diagnostic procedure are easily procured. They consist of an ethyl aminobenzoate lozenge, an abrasive gastric balloon apparatus, a cylindrical beaker filled with 100 cc. of isotonic sodium chloride solution, two 50 cc. centrifuge tubes, three glass slides, and a container with equal parts of ether and 95% ethyl alcohol fixative.

The prepared patient is seated in an upright position and given an ethyl aminobenzoate lozenge to dissolve under his tongue, and a towel is placed under his chin. The abrasive gastric balloon completely deflated is moistened. As it is placed into the mouth, the patient is asked

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[1] Panico, F. G.; Papanicolaou, G. N., and Cooper, W. A.: Abrasive Balloon for Exfoliation of Gastric Cancer Cells, J. A. M. A. 143: 1308-1311 (Aug. 12) 1950.

to swallow the balloon. In many instances, the patient may direct the swallowing of the balloon without the aid of the examiner. Fluid is administered orally only as necessary. All saliva, postnasal discharge, and pulmonary excretions are discarded to prevent swallowing of unnecessary cellular debris. As the balloon passes through the constrictor muscles of the hypopharynx, there is at times an accompanying cough reflex. This produces no difficulty, and the patient is encouraged to keep swallowing. Once the balloon is in the esophagus, it descends by itself with relative ease, and the patient experiences no discomfort. When the balloon reaches the cardia at a distance of about 55 cm., the direction of passage of the balloon tip toward the pylorus may be facilitated by placing the patient in a right lateral recumbent position. With the patient completely relaxed, the balloon descends to about 75 cm. into the region of the pylorus (fig. 2). With this method, there should be no coiling of the nonrigid tube, and the abrasive gastric balloon is positioned without the aid of fluoroscopy. If the tube is made semirigid, the patient need not be placed in a recumbent position but may be examined throughout the procedure in an upright position. The balloon is inflated slowly by injecting approximately 150 to 250 cc. of air. Ordinarily the free balloon averages 6 cm. in diameter; however, as expansion of the balloon occurs in the stomach, the proximal end of the balloon meets the least resistance and inflates first. This tends to pull the balloon retrograde through the prepyloric antrum into the body of the stomach (fig. 3). As can be seen fluoroscopically, reactive peristalsis may help the balloon in its proximal shift. Hyperperistalsis can be seen in the regions of the stomach not in direct contact with the balloon surface. As the balloon passes through the body of the stomach, peristaltic waves may cause a constricting effect around the balloon. Slowly the balloon is withdrawn into the cardiac region of the stomach where it is checked at the cardia. This is occasionally accompanied by a vomiting reflex (fig. 4). At this point, the balloon is deflated and returned to the pylorus, where it is blown up again. This is repeated three times, an interval of one minute being arbitrarily used for transferring the balloon from the pylorus to the cardia. At the end of three minutes the balloon is withdrawn to the cardia and allowed to collapse completely before being retrieved. The abrasive balloon carrying the specimen of

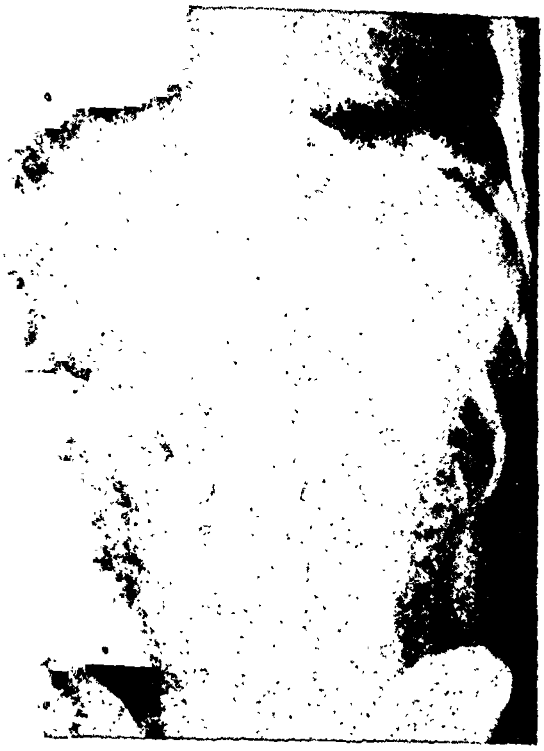

Fig. 2.—Roentgenogram showing uninflated abrasive gastric balloon inserted to the pylorus.

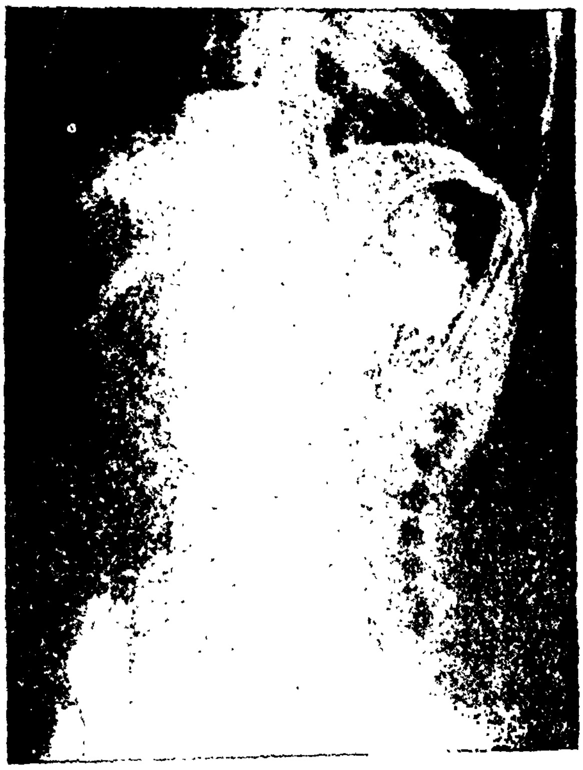

Fig. 3.—Roentgenogram showing inflating abrasive gastric balloon traveling retrograde through the prepyloric antrum and body of the stomach, stimulating reactive peristalsis.

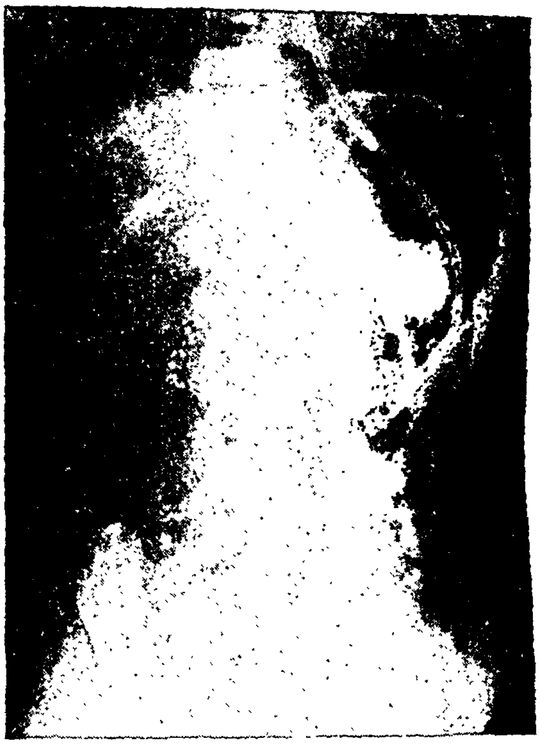

Fig. 4.—Roentgenogram showing inflated abrasive gastric balloon distending the cardiac and fundic regions of the stomach prior to deflation.

gastric and esophageal cells is immediately placed in a beaker containing 100 cc. of isotonic sodium chloride solution. A perforated rubber stopper is placed over the cylinder, which keeps the balloon entirely immersed in the physiological medium. Through the small opening in the rubber stopper, the tube is jiggled up and down in order to free the mucoid cellular particles. The sodium chloride solution becomes cloudy with a large amount of floating cellular material. Some of the malignant cellular clusters may even approach the size of pathological biopsy material. When the surface of the balloon is completely cleansed of cells, the balloon is withdrawn, detached from the apparatus, and destroyed. The apparatus is subsequently cleansed and sterilized preparatory to the attachment of a clean unused abrasive gastric balloon. Throughout the procedure, there is usually no regurgitative vomiting experienced from the fasting stomach. While in the stomach the actual distance that the balloon traverses varies with the length of the balloon and the distensibility of the stomach. An abrasive balloon 20 cm. long almost fills the gastric cavity. A 10 cm. balloon, being shorter, may traverse the stomach over a greater distance to afford an increased abrasive effect.

The specimen of cells contained in the sodium chloride solution represents the entire surface area of gastric and esophageal mucosa. The specimen is placed in two 50 cc. centrifuge tubes. The tubes are centrifuged at 2,500 revolutions per minute for 5 or more minutes. The longer the period of sedimentation the better the cell concentration at the bottom of the centrifuge tube below the mucous layer. After decanting, the sediment is utilized in the preparation of smears. The cellular material is placed on a clean glass slide and spread evenly by means of pressure through another glass slide. Superimposition of the cells may be minimized by making thin smears. A wire loop method of smearing cells may be used. Prepared within a 15 minute period, the gastric smears are placed immediately into a mixture of equal parts of ether and 95% ethyl alcohol to fix the cells. Three slides should suffice for diagnostic purposes. Following fixation the smears are ready for future staining by the Papanicolaou technique. With this procedure, the improved stomach explorer has been tested and found to be satisfactory for gastric diagnosis.

COMMENT

To pass the abrasive balloon into the stomach for cytological diagnosis is no more difficult than to insert a stomach tube through the nose for gastric analysis. The criteria for the use of the abrasive gastric balloon test is the suspicion of a malignant gastric lesion. Age is not a factor, the older age group of patients tolerating the examination extremely well. While the abrasive balloon is in the stomach, it makes immediate contact with the entire mucosal surface. The particles of mucoid cellular material represent, for the most part, abnormal shedding tissue. It is already known that normal gastric mucosa resists mechanical exfoliation. In many ways, the gastric smear is a concentrated reflection of the entire stomach surface. The cytological interpretation of the gastric smear is an integrated picture of the histological and pathological status of the stomach mucosa. The final interpretation may be read as negative or positive for malignant disease, although occasionally an abnormal smear will occur giving a suspicious negative result.

Untoward effects from the abrasive gastric balloon have not been encountered. Gastric ulcers have progressed to healing without event. Accentuation of bleeding has not occurred in patients who have had recent gastric hemorrhage; however, the use of the abrasive gastric balloon test is being avoided in the presence of active gastrointestinal hemorrhage, known esophageal varices, or esophageal obstruction or postoperatively in cases of gastric resection.

How should the abrasive gastric balloon test be used as a diagnostic screening test? The stomach of the patient may be investigated cytologically if he shows (*a*) early symptoms and signs of malignant disease, (*b*) achlorhydria, (*c*) questionable defects seen by gastrointestinal series, or (*d*) suspicious mucosal lesions seen by gastroscopic examination. The individual gastric suspect may be handled in the clinic quickly by first securing a gastric analysis and then performing an abrasive gastric balloon cytological examination. In this way, the achlorhydric stomach would be screened, and the retentive stomach could be recognized for adequate preparation.

If a positive gastric smear is obtained, the problem immediately becomes surgical in nature, and consideration of exploratory laparotomy is warranted. Since carcinoma of the oropharynx may be recognized by direct examination and carcinoma of the duodenum is almost nonexistent, a general localization of the lesion to the stomach or esophagus can be made by cytological examination. If a more definite localization of the lesion within the stomach is deemed necessary, then a gastrointestinal series and a gastroscopic examination may be secured. If a negative or suspicious negative gastric smear is obtained, the problem should be surveyed further by means of an upper gastrointestinal series and gastroscopic examination, along with any other gastric tests that may be utilized. Material obtained from the abrasive gastric balloon yields a sufficient quantity and quality of cells from the stomach and esophagus to minimize the number of false negative and false positive results reported prior to development of the balloon technique. Gastric smear readings for malignant disease can be depended on to have a satisfactory degree of accuracy. As a result of the development of this technique, gastric cytology has become a growing field that offers one potential key to gastric diagnosis.[2]

SUMMARY

The application of the abrasive gastric balloon offers a convenient diagnostic means for revealing gastric malignant disease. The technique is adapted to quick office procedure.

306 S. Drew St.

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PRESENT ROLE OF ZINC PEROXIDE IN TREATMENT OF SURGICAL INFECTIONS

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The antibiotics have so completely taken hold of our minds in the treatment of surgical infections that we are often forgetful of other methods of treatment that are available. I have frequently been asked whether, since the arrival of the sulfonamides and the antibiotics, there are any conditions for which medicinal zinc peroxide is superior to these other chemotherapeutic agents. Experience has demonstrated that there are certain infections that are more amenable to treatment with this agent than to treatment with the antibiotics and that, if medicinal zinc peroxide is properly used, it will bring about the control of the infection and healing of the wound more quickly than will any other form of treatment.

There are two regions of the body where this drug is of particular value: on the outer surface of the skin and along the alimentary tract. It can be used for the interior of the body only if there is a cavity with an opening from which the residue may be readily removed.

LIMITATIONS AND VIRTUES OF ZINC PEROXIDE

The limitations of the drug as well as its virtues must be kept in mind. The limitations are as follows: 1. It must be an active preparation of "medicinal grade" capable of producing nascent oxygen whenever water is added to it. 2. It must be suspended in sterile distilled water so as to form a thick creamy suspension and be applied on cotton or fine mesh gauze in close contact with the infected surfaces. 3. It must be kept wet.

A test to determine the effectiveness of the drug can be easily performed. If a small amount of the powder is placed in a test tube and covered with twice the quantity of water, within an hour the supernatant water will become clear, the sediment will become curdy, and bubbles of oxygen will form in the curd and will gradually lift it to the top of the test tube. Ineffective zinc peroxide will remain at the bottom of the test tube as an inert mass, which may cake, and no oxygen bubbles will be generated.[1]

Before use, zinc peroxide should be sterilized in a dry oven for four hours at 140 C. This not only sterilizes it but renders it more active. Sterile zinc peroxide may be kept in test tubes or bottles for many months without significant deterioration. As far as I know, only the DuPont Chemical Company has been able to make the active "medicinal grade" zinc peroxide, and at present Mallinckrodt Chemical Works is the only company distributing it.

The virtues of the drug are as follows: 1. It will rapidly destroy anaerobic and microaerophilic organisms and inhibit a wide variety of aerobic bacteria. 2. It is particularly effective in the presence of mixed infections. Both proteus and pyocyaneus organisms are inhibited by it when it is used in the concentrations generally employed. 3. It is completely nontoxic and nonirritating to the tissues. 4. After the drug gives off its oxygen, relatively bland zinc oxide and zinc hydroxide remain, which are well tolerated by the tissues. 5. It is a detoxifying agent, inactivating almost every kind of bacterial toxin. 6. It apparently stimulates the growth of healthy, well-vascularized, firm, granulation tissue, which hastens wound healing and permits early skin grafting. 7. It will control hemorrhage from infected surfaces.[2] 8. One very fortunate attribute of zinc peroxide is that it does not stick to the tissues even when applied on cotton. The outer surface must be kept wet so that the dressing will not adhere to the tissues.

Zinc peroxide is most active when used in a watery suspension, but it may be incorporated in a carbowax® (polyethylene glycol) water-soluble base when it is used on areas difficult to keep moist. This ointment may be prepared in any pharmacy with 40 parts of carbowax® 1540, 40 parts of carbowax® 300, and 20 parts of activated zinc peroxide. The carbowaxes® are melted together over a free flame and then sterilized in a dry oven at 140 C for two hours. The mixture is then allowed to cool; as it begins to solidify, the activated zinc peroxide is added. It is mixed well with an egg beater and placed immediately in the refrigerator. It is again stirred with a rod just before solidification and then refrigerated until it is solid. Reid and Altemeier found that 1% calcium peroxide might be combined with 10% zinc peroxide in such a base; the combination seemed to be more effective than zinc peroxide alone.[3]

ULCERS AND OTHER SKIN INFECTIONS

Undermining Burrowing Ulcers.—It was with the undermining burrowing ulcers that medicinal zinc peroxide first demonstrated its clinical usefulness.[4] This lesion is caused by a microaerophilic hemolytic Streptococcus that has a propensity for burrowing in the subcutaneous tissues and down in between muscles and along lymphatic channels. It frequently involves neighboring lymph nodes, and the infection often smolders there until the nodes are surgically removed. These ulcers are chronic, often lasting years until the proper treatment is administered. The infection frequently burrows through the skin from beneath. Starting as a pinpoint opening, a daughter

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ulcer may form, gradually enlarge, and then either fuse with the main ulcer or leave a bridge of skin that is completely surrounded by epithelium. The value of medicinal zinc peroxide in the treatment of these ulcers has been amply confirmed.[5]

It is obvious that the local application of any medicament may not reach every part of the infected surface and its activity certainly will not readily spread up to the neighboring lymph nodes, if they are involved. The blood vessels in the periphery of such lesions are usually blocked by blood clots, making it difficult for any medicament given systemically to reach the infected surface from the inside. If contact cannot be maintained with every part of the infected surface, it becomes necessary to excise the lesion and the grossly involved lymph nodes *in toto*. Then the application of medicinal zinc peroxide on the raw surface quickly disposes of the microaerophilic hemolytic Streptococcus and all other susceptible organisms. Cultures have been made during the course of wound healing in numerous cases in which this treatment was used. The first cultures, particularly those obtained from the innermost recesses of the wound, show the microaerophilic hemolytic Streptococcus, but within 24 to 48 hours the progeny becomes aerobic. With continued treatment the organisms become green. Soon afterward they disappear from the wound.

Progressive Bacterial Synergistic Gangrene.—There is one type of gangrenous ulcer of the skin that has characteristic clinical features. This is due to the presence of a microaerophilic nonhemolytic Streptococcus and a Staphylococcus aureus. It is therefore called synergistic gangrene and is inexorably progressive unless it receives the proper treatment.[6] Its pathognomonic symptom is extreme pain and tenderness, and it is readily recognized by its typical three zones: an outer erythematous zone, a middle raised purple necrobiotic zone, and an inner zone of "suede leather" gangrene. It often starts in an infected operative wound, particularly one that has drained a putrid abscess of the abdomen or chest, and one that has been closed with retention sutures. The tension of these sutures seems to establish the conditions under which the microaerophilic nonhemolytic Streptococcus can gain a foothold.

A number of authors have reported cases of synergistic gangrene and have stated that they have treated the condition with zinc peroxide without effect, but it is obvious that medicinal zinc peroxide or any other medicament applied locally could never reach the spreading periphery of this lesion. In order for one to treat it with medicinal zinc peroxide, it is necessary first to widely excise the lesion beyond the zone of erythema. Then closely applied medicinal zinc peroxide suspension will kill the microaerophilic organism and will prevent a recurrence of the infection. However, it has recently been demonstrated that this condition can often be cured and surgery obviated by the systemic administration of bacitracin.[7] Medicinal zinc peroxide, therefore, should not be used for treatment of this lesion unless the organisms are resistant to both penicillin and bacitracin therapy. Then excision becomes necessary.

Other Ulcers and Skin Lesions.—There are various other kinds of ulcers that may occur on any part of the body surface but are particularly prevalent on the legs. If infection plays a major part in their occurrence or in their persistence, many of them respond to treatment with medicinal zinc peroxide, especially those caused by the hemolytic Streptococcus or the hemolytic Staphylococcus aureus. Hemolytic streptococci promptly disappear from ulcerated surfaces when medicinal zinc peroxide has been applied. Most of these ulcers do not have the typical undermined and rolled in edges, which are characteristic of, and should not be confused with, the undermining burrowing ulcer.

Decubital ulcers clean up and heal more rapidly if medicinal zinc peroxide is employed on cotton balls or flat cotton wet with a suspension of the powder in water and kept moist with an overlying layer of wet flat cotton sealed with a double thickness of zinc oxide ointment gauze.

Other skin lesions that are particularly amenable to treatment with medicinal zinc peroxide are the very common exudative lesions produced by contact with the leaves, branches, or roots of poison ivy and poison oak. If medicinal zinc peroxide powder is applied to the lesions in the form of a lotion, they usually dry up quickly and spread no further. A rapid cure almost invariably results, probably owing to the detoxication of the Rhus toxin. The pain and vesiculation of herpes zoster is also amenable to medicinal zinc peroxide treatment. Again the medicament should be applied in the form of a lotion, which can be renewed at frequent intervals.

In the foul-smelling gangrenous lesions of the extremities in diabetes or arteriosclerosis, the use of medicinal zinc peroxide will prevent the activity of the odoriferous organisms and sometimes permit healing after conservative removal of the dead tissue.[8]

Gas Gangrene.—The results of medicinal zinc peroxide therapy in both experimental and clinical gas gangrene are somewhat equivocal, probably because of the difficulty of getting and maintaining contact with the area of infection.[9] With excision of the necrotic muscle, however, treatment with medicinal zinc peroxide has been found able to take care of the residual infection. The benefit obtained by intramuscular and local application of bacitracin and the oral administration of aureomycin and chloramphenicol may well take the place of medicinal zinc peroxide in the treatment of this condition.

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Putrid Lung Abscess.—In foul-smelling lung abscesses that have been surgically drained, medicinal zinc peroxide may be instilled and gently applied on cotton balls, with prompt disappearance of the putrid odor, which is due to the anaerobic streptococci, spirochetes, and fusiform bacilli.[10]

Human Bites.—Lesions resulting from human bites often fall into the same category as lung abscesses as far as the bacteriologic aspects are concerned, and medicinal zinc peroxide is effective both in the prophylactic and in the active treatment of these wounds.

ALIMENTARY TRACT INFECTIONS

Mouth and Throat Lesions.—Infections along the alimentary tract are successfully treated with medicinal zinc peroxide, for example, ulcerative stomatitis and Vincent's infection of the mouth and gums.[11] Often the pain of sore throat produced by the hemolytic Streptococcus is promptly relieved by the use of medicinal zinc peroxide. This may be applied to the surface as a mouthwash or gargle, or, in order to maintain contact with the infection for as long a time as possible, it is a good practice to make a thick suspension of medicinal zinc peroxide on a flat piece of wet cotton and apply it directly to the lesion, maintaining contact for an hour at a time several times a day. The tonsils and pharynx may be swabbed with cotton on an applicator wet with the zinc peroxide suspension. Medicinal zinc peroxide may be used as a prophylactic before plastic operations on the mouth or before dental extractions. After extractions it may be applied on a small pledget of cotton directly into the tooth socket.

If infections occur from the mouth organisms spreading down into the neck with a rapid breakdown of the nodes and surrounding tissues, after incision for drainage, medicinal zinc peroxide may be applied to the abscess cavity, with a rapid resolution of the infection.[12] The foul odor of these infections is due to gas-forming anaerobic streptococci with or without spirochetes. When their growth and activity is prevented by treatment with medicinal zinc peroxide, the odor rapidly disappears.

Mediastinal Infections.—Likewise, the foul-smelling abscesses that form in the mediastinum or in the pleura after perforation of the esophagus, by a foreign body, by an ulceration, or by a dilating instrument, respond to zinc peroxide after surgical intervention, because the most important organism in such infections is a gas-forming anaerobic Streptococcus. Medicinal zinc peroxide cannot be given by mouth because it causes nausea when it reaches the stomach, unless it is taken in enteric-coated capsules. However, only small quantities can be delivered to the intestine in that way. The antibiotics and the sulfonamides are more dependable for reducing the susceptible organisms of the intestine.

Ulcerative Colitis.—In ulcerative colitis, medicinal zinc peroxide may be extremely useful.[13] If the lesion is limited to the lower part of the large bowel in the rectum and sigmoid, medicinal zinc peroxide may be delivered by retention enemas. In some cases when there is extensive ulceration and extreme irritability, it cannot be retained very long at first, but, if it is so introduced and expelled, it leaves a coating of zinc peroxide that has an inhibiting effect on the growth of anaerobic organisms and favors the healing of ulcerations. With repeated instillations it can be tolerated for a longer and longer time. However, if a retention enema containing medicinal zinc peroxide, 6 to 8 oz. (170 to 227 gm.), cannot reach the upper limits of the lesion, it is necessary to perform a cecostomy and deliver it above the lesion, so that it may reach all of the infected surfaces. In many cases delivering it in this way results in rapid healing of the ulcerated area of the intestine from the cecum downward, even when the drug is mixed with fecal material. However, in some cases in which the ulceration is extreme, it is better to perform either an ileostomy or a colostomy above the lesion, thus cutting off the fecal stream and giving the medicament a better chance to act on the ulcerated areas of the mucous membrane. I feel certain that, with medicinal zinc peroxide therapy, a colectomy can be avoided in some cases or a less extensive colectomy than would otherwise be required can be performed.

Whenever medicinal zinc peroxide is used in an enema or is to be delivered into the cecum or lower ileum, it is advisable to suspend it in a 2% polyvinyl alcohol solution rather than in sodium chloride solution or in water. In this mucilaginous suspension it does not become dry and hard during the dehydration process as the fecal material passes on toward the rectum.

Anorectal Infection.—Medicinal zinc peroxide is extremely useful after operations on fistulous tracts and on hemorrhoids as well as on infected pilonidal cysts. It may be applied in a watery suspension on cotton balls that fit readily into the cavity from which the fistulous tract has been excised or the pilonidal sinus has been removed.[14] After hemorrhoid operations a gauze rol may be soaked in the medicinal zinc peroxide suspensio and inserted into the anus. I have yet to find a patien who objects to the presence of the plug. The gentle pres sure that it exerts tends to stop all bleeding and inflam mation. One is particularly struck by the absence of swelling, redness, and pain around the site of operation. On the second, third, or fourth day this roll may be readily removed from the anal canal, and by that time the danger of infection from intestinal contents has passed.

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VAGINAL INFECTION

The organisms most to be feared in the vagina are the hemolytic streptococci, the anaerobic cocci, and the gas gangrene organisms. These are all inhibited and destroyed by medicinal zinc peroxide therapy. Infections of the vagina caused by these organisms are generally quickly brought under control by the nightly instillation of a suspension of this drug. This may also be used as a prophylactic against infection before plastic operations on the vagina and before supravaginal hysterectomy.

AS AN ADJUNCT IN CANCER THERAPY

Those who deal with cancer find medicinal zinc peroxide very useful, particularly in minimizing the foul odor and bad taste of inoperable cancerous lesions of the mouth during and after radiotherapy.[15] In the Memorial Hospital in New York it is employed constantly for these purposes.[16] In cases in which there is a radionecrosis of skin—for example, in the mammary region—medicinal zinc peroxide therapy will prevent odor and favor wound healing. When a colostomy is formed, light packing around it with gauze soaked with a suspension of medicinal zinc peroxide or medicinal zinc peroxide ointment will usually prevent the development of infection in the surrounding tissues and prevent the sloughing of fascia, which may lead to a ventral hernia. After an abdominoperineal resection of the bowel, the perineal cavity may be kept free from infection by instillation of medicinal zinc peroxide and packing of the cavity with a 5 yd. (4.67 meters) gauze roll inside a China silk tampon.

SUMMARY

Medicinal zinc peroxide is still useful in the treatment of surgical infections even though the sulfonamides and antibiotics are available. Only the medicinal zinc peroxide approved by the United States Pharmacopoeia should be used. It is more effective if activated and sterilized by heating the powder in a dry oven at 140 C for four hours. After this procedure it may be kept in sterile containers for several months without significant deterioration.

The most important conditions better served by medicinal zinc peroxide therapy are undermining burrowing ulcers; synergistic gangrene, only after excision; decubital ulcers; other ulcers in which there is a large element of infection; diabetic gangrene; ulcerative stomatitis; foul-smelling lesions of the mouth and neck, especially in the presence of cancer; radiation burns; ulcerative colitis; and nonvenereal vaginitis. It is useful as a prophylactic around colostomies and after dental extractions. It is also very effective for the skin lesions of herpes zoster and poison ivy.

630 W. 168th St.

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LONGEVITY IN BENIGN ESSENTIAL HYPERTENSION

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This is a report of a study of 100 cases of benign essential hypertension followed personally by us for 10 to 34 years. Our purpose in making this study was threefold: (1) to add to the meager volume of valid published data concerned with the ordinary course of this disorder; (2) to seek clues that might explain the long life and favorable course in these cases, thereby increasing our capacity for making accurate prognoses; and (3) to make physicians more conscious of the fact that the course in many hypertensive patients may be a very benign one, compatible with long and effective living. If this last can be accomplished, judgments concerning patients with high blood pressure will be dominated less by the end-stages of this disorder, with its dreaded hemiplegia, heart failure, or uremic poisoning, and patients should receive better and more reasonable therapy than many now do. It is our hope that this paper may also dispel much of the confusion created by the literature concerning length of life in patients with hypertensive vascular disease.

Reports concerned with mortality rates among patients with hypertensive vascular disease have been contradictory. For example, Ranges[1] reported no deaths whatsoever among 241 patients observed for 10 to 25 years, whereas Hamman's[2] 10-year death rate was 78%. Statistics from other authors fluctuate markedly between these extremes. Such apparent lack of unanimity is readily explained by analysis of the various methods of selection used by different authors. Cases of mild disease predominate in some papers[1] and cases of severe disease in others.[3] In many,[4] cases of both benign and

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malignant disease are grouped together. Some authors[5] make little if any distinction between the various diseases that have hypertension as a common factor. Some publications deal only with special groups, such as those that have come under the scrutiny of insurance companies.[6] The reports of Hamman,[2] Paullin,[7] Riesman,[8] Rice,[9] and Burgess[10] deal exclusively with private patients. Most of the other writers have utilized only hospital or clinic patients. Their papers have the inherent weakness of being essentially collections of data taken from hospital records. The observations recorded had been made by numerous observers with varying interests and standards of accuracy.

Some of the confusion has been dispelled in the last few years by the excellent articles of Burgess,[10] Blood,[11] Perera,[12] Palmer,[13] Ranges,[1] Smith,[14] and Griep.[15] Many more will have to be written with data carefully determined and compiled by uniform methods before adequate information is available.

Papers based on a continuing series of documented observations made by trained persons are few indeed. Seventy-eight per cent of our patients were private patients of one of us (J. P. O'H.). Twenty-two per cent were patients in the clinic at the Peter Bent Brigham Hospital, and all the observations on them were made by one of us (J. P. O'H.) or his associates.

Cases were selected on the basis of (1) a documented history of 10 or more years of hypertension (systolic 145 mm., diastolic 95 mm., minimum); (2) absence of primary renal disease (glomerulonephritis or pyelonephritis); (3) absence of coarctation of the aorta, valvular heart disease, hyperthyroidism, and similar disorders causing hypertension; and (4) absence of any radical therapy, such as sympathectomy and rice diet. (A very small group of these patients, in their early years, had an unsuccessful trial with diet extremely low in salt. Another small group received "cyanate" therapy for a few weeks.) Excluding these few, the condition in all patients was classifiable as benign essential hypertension treated throughout by conservative methods only, i. e., sedation, weight reduction, and general advice.

Of the entire group of 100, all were white and predominantly of Irish, American, or Jewish extraction; 67 were women and 33 men. The age distribution at the time of discovery of the hypertension ranged from 21 to 68 years, with an average of 45 years. Initial systolic pressures varied from 130 mm. to 250 mm.; diastolic pressures from 80 mm. to 140 mm. (Normal systolic pressures were accompanied by elevated diastolic pressures and vice versa.) The period of documented hypertension ranged from 10 to 34 years, with an average of more than 17 years. Since 71% of these patients are still alive, including our "34-year" patient, this range and average will in all probability increase. A breakdown of the group by five-year periods is shown in table 1.

TABLE 1.—*Duration of Hypertension*

Period, Yr.	No. of Patients	No. Living	No. Dead
30-34	4	3	1
25-30	8	5	3
20-25	11	8	3
15-20	42	31	11
10-15	35	24	11
Total	100	71	29

It is particularly impressive that 23 patients have lived 20 or more years (average 25) with their hypertension and that 16 of these are still alive and have a fair chance of living several more years.

Of the group of 71 still alive, 47 are in "good" condition, 19 in "fair" condition, and only 5 in "poor" health. Such data clearly point to the fact that essential hypertension may be compatible with relatively long life. Furthermore, such long life has been, with some exceptions, reasonably comfortable and effective.

Thus far only 29 of our patients have died. The causes of death are indicated in table 2. The incidence of cerebral accident and myocardial failure among these 29 is at variance with that in other published data. However, this is a very small group; when death eventually comes to the entire 100, the percentages, in all probability, will be quite different. It should be noted, too, that the figures in table 2 represent only the principal cause of death. Few patients have escaped one or more of the complications commonly associated with hypertensive vascular disease. For example, coronary insufficiency has been noted at some time in 51% of the entire group of 100 patients. In the 29 who have died, this symptom had occurred in all but 8. Coronary thrombosis, though listed as the principal cause of death in only 8 patients, occurred at least once in 12 others. Myocardial failure was the chief cause of death in only 3 patients, and yet it was noted in 19% of the entire group and played a prominent role in 13 patients who died. This disorder appeared late in the disease, usually during the last two years of life. Cerebral vascular accident, credited with being the principal cause of death in 14 patients, occurred in 28 of the entire group. Chronic nephritis developed in 12 patients but played a significant role in only one of those who died. Diabetes

TABLE 2.—*Principal Cause of Death in Twenty-Nine Patients*

Cause	No. of Patients
Cerebrovascular accident	14
Coronary thrombosis	8
Myocardial failure	3
Cancer	1
Auto accident	1
Unknown	2
Total	29

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5. King, Carlile, and Blackford.[4a] Janeway, T. C.: A Clinical Study of Hypertensive Cardio-Vascular Disease, Arch. Int. Med. **12**: 755, 1913.

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15. Griep, A. H.; Barry, G. R.; Hall, W. C., and Hoobler, S. W.: Prognosis in Arterial Hypertension: Report on 117 Patients Under 53 Years of Age Followed 8 to 10 Years, Am. J. M. Sc. **221**: 239, 1951.

mellitus was noted in nine patients but was an important feature in only three of those who died.

In any disease as prevalent as hypertension, it is obvious that observations on 100 patients, even when such observations are as numerous and accurate as ours, may not be statistically significant. Others, no doubt, will record similar observations, and eventually there will be accumulated sufficient accurate material to make the total significant. Nevertheless, we would be remiss if at this time we did not analyze our voluminous data to check the clinical impressions gained from these patients and to seek clues that might explain their longevity and their favorable courses. We believe the results are of considerable interest and importance, particularly with respect to the light they throw on the course of benign hypertension and the suggestions they offer for making more satisfactory prognoses.

It may be suggested, with some degree of justice, that ours is an exceptional group. To the extent that these patients were deliberately chosen because they had essential hypertension, had lived with this disorder at least 10 years, and had accurate documentation, this is correct. We have no means of knowing what percentage of the total number of patients with essential hypertension is represented by patients with such benign disease. Too few groups have been followed for 10 or more years to permit us to answer this question. We strongly suspect that the percentage is far greater than is commonly believed.

It may be argued that the group is not truly representative, since four-fifths of the patients were private and only one-fifth clinic patients. It can be truthfully stated that as a whole the patients were from typically "middle-class" American families. Few were wealthy, and few were very poor. Practically none was able to give himself exceptional care. Not more than 4 out of the entire 100 could be said to be even reasonably free from strains of one kind or another, real or imagined.

Our series contains a high proportion of women (67 women to 33 men). This proportion is, however, in keeping with that considered usual by Boas [16] and by Blackford.[17] It is generally agreed that women as a class withstand hypertensive disease better than do men. Our data support this conclusion. It is of considerable interest to note that of the 23 patients who have lived the longest (20 or more years) only 5 were men.

AGE AT ONSET

The predominance of the younger age group among the patients with chronic nephritis or "malignant" hypertension must account, in part at least, for the generally accepted opinion that the younger the age when hypertension starts, the poorer the prognosis, and the older the age at onset, the more favorable the outlook. While this statement is generally true, there must be many exceptions. Some of these are found in this series. In our patients, all of whom had high blood pressure for at least 10 years, the age at discovery ranged from 21 to 68 years, with an average of 45 years. In 5 patients the disease began before age 30 and in 28 before age 40.

It is of additional interest to note in the 23 patients who have lived the longest—20 or more years—that 10 were discovered to be hypertensive before they were 40 years of age and one before he was 30. Furthermore, of the 28 with onset before 40 years, only 8 have died. Perhaps the ratio of 20 women to 8 men in this group may be a factor.

RANGE OF BLOOD PRESSURES

With three important exceptions, to be discussed later, blood pressure levels, per se, in the individual patient were of little value in helping us to determine his true condition and make a prognosis. This is particularly true of the initial readings. These readings in some patients were exceedingly high, reaching in one, a top level of 250/140. The lowest initial recording was 130/80. (Sub-hypertensive systolic and diastolic levels were associated with corresponding hypertensive diastolic and systolic readings.)

False conclusions could easily have been drawn—and probably were—from such high figures, undoubtedly recorded at times of great stress. It should be again pointed out that all these patients lived at least 10 years after such high levels were observed. In the group that have lived 20 years or more, the initial blood pressure was found to be as high as 224/120. Three of this same group had initial systolic pressures over 200, and 2 had diastolic levels over 120. (It seems reasonable to draw another inference from these high first readings: that these patients probably had had increased pressure for some time previous to its discovery.)

Throughout the years of observation, the highest systolic pressure was 300 and the highest diastolic pressure 170; the lowest systolic pressure was 108 and the lowest diastolic pressure 60. The range of individual variations was as follows: systolic 20 to 150 (average 68) and diastolic 10 to 81 (average 40).

Over the years, too, individual blood pressure readings and curves of pressures by themselves contributed little toward a proper prognosis. Wide variations, in the forms of peaks or slower wave-like rises and falls, were the rule. There was little "trend" pattern and, except in a group of 17 patients, little constancy or stabilized levels of pressure.

These 17 patients are of particular interest in that, throughout their courses, they have never shown pressures above 180/120. Six of these are in the group of 23 who have lived more than 20 years. One is still alive and well after 34 years of such hypertension. Four patients have constantly shown diastolic levels of 110 mm. or less. Only 4 of the entire 17 have died, all with serious heart failure. Patients of this type, who maintain a constant low level of hypertension for months or years, are exceptional, and the statement made previously that blood pressure levels, per se, gave little indication of the ultimate prognosis, is not true of them. A second exception is the type of patient having progressive, steady rise of diastolic pressure into high levels, foreboding strain on the whole circulatory apparatus. However, this occurred rarely in our group, and then only near the end of life.

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16. Boas, E. P., and Fineberg, M. H.: Hypertension in its Relationship to Mitral Stenosis and Aortic Insufficiency, Am. J. M. Sc. 172: 648, 1926.

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AVERAGE BASAL DIASTOLIC BLOOD PRESSURE

In the group that has lived 20 years or more, the average basal[18] diastolic pressure was 91 mm. The average in the entire group was 95 mm. and in those that had died, 99 mm. The most important exception to the generally poor prognostic value of blood pressure readings, and one of the most important conclusions we have been able to draw from our studies, is that low basal diastolic pressures suggest a favorable course. It seems highly significant that these patients, who have at times shown such extremely high levels of systolic and diastolic pressure and yet have lived so long and so well, have had basal diastolic pressures that averaged throughout the years only 95 mm. For the 23 who have lived an average of 25 years, the average "basal" diastolic pressure was even lower (91 mm.); for the 29 dead, who lived an average of 17 years, it was somewhat higher (99 mm.).

In our judgment, basal diastolic pressures represent more nearly the ordinary day by day, hour by hour, arterial pressure and circulatory strain than do the higher "spot" readings made in the physician's office or in the hospital, readings so often affected by the emotional strain of the examination.

Summarizing the analysis of our data on blood pressures in this group, we conclude that, with two important exceptions, single readings at any given time, or multiple readings over long periods, by themselves, were of little help in prediction of the course of the disease. The two exceptions were in (1) the relatively small group of patients whose pressures were stabilized at comparatively low levels of hypertension, and (2) those with low average basal diastolic pressures. We may also conclude that, in these 100 patients, the degree and constancy of strain were far less than were suggested by the frequent high levels of blood pressure recorded.

FUNDI

For many years we have believed that careful examination of the fundi was one of the most fruitful studies that could be made of the hypertensive patient. We felt that the degree of retinal arteriolar sclerosis revealed the results of strains and the seriousness of the disease better than any other single sign until the appearance of the advanced cardiac and cerebral changes that presage the "end of the road." Sporadic hemorrhages and white spots, except in association with severe retinal arteriolar sclerosis or papilledema, seemed to have little prognostic value. The analysis of the data in this series confirms our previous opinions.

It is extremely significant that of 98 patients the results of whose most recent examinations were satisfactory, only 22 showed marked arteriolar sclerosis (grade 3). One-third never revealed more than a moderate amount (grade 2), and almost one-half with all their years of hypertension, still had only grade 1 arteriolar sclerosis. The older group, those with more than 20 years of high blood pressure, are even more interesting, in that 6 of the 22 had grade 1, 8 had grade 2, and only 8 had grade 3 arteriolar sclerosis. The slight increase in the number with grade 3 arteriolar sclerosis among the group of dead patients is hardly significant, since it is to be remembered that even these patients averaged 17 years of high blood pressure.

As indicated in table 3, the relative infrequency of advanced changes in the retinas and disks speak for themselves. Only three of the entire group had many hemorrhages and/or exudates, and none had severe papilledema.

Since the arterioles are the blood vessels most concerned in the hypertensive process, it seems reasonable to infer that these vessels in our long-lived patients tolerated the pressures very well and that the strains, in spite of the occasional high pressures recorded, were not so great as might have been expected. This idea fits well with that indicated by the low level of basal diastolic pressures previously mentioned.

ELECTROCARDIOGRAPHIC AND ROENTGENOGRAPHIC OBSERVATIONS

Two other divisions of our data seem to point in the same direction: the electrocardiographic and roentgenographic observations of the hearts.

TABLE 3.—*Observations of the Fundi*

Arteriolar Sclerosis

No. of Patients	Grade 1	Grade 2	Grade 3
Total, 98 *	43	33	22
Living average of 25+ yr., 22	6	8	8
Dead, 28 †	10	8	10

Maximum Retinitis

	Grade 0	Grade 1	Grade 2	Grade 3
Total, 98 *	54	29	12	3
Living average of 25+ yr., 23 *	13	5	4	1
Dead, 28 †	9	12	6	1

Papilledema

	Grade 0	Grade ±	Grade 1	Grade 2
Total, 97 *	90	2	4	1
Living average of 25+ yr., 22 *	22	..	..	..
Dead, 28 †	23	2	2	1

* *Apparent discrepancy in numbers* is due to lack of recent observations or to inadequate observations because of cataracts and the like.
† Lived an average of 17 years.

Among 91 patients, the electrocardiographs showed normal axis in 26, left axis deviation in 50, left axis deviation with left ventricular hypertrophy in 14, and left ventricular hypertrophy alone in 1.

In the light of the previously expressed opinion concerning the degree of strain, it is important to note that 26 of the group of 91 have never shown anything but a normal axis, never having showed even the left axis deviation so commonly associated with hypertension. Only 15 revealed the greater strain indicated by the left ventricular hypertrophy pattern.

Of course, there were other electrocardiographic abnormalities of greater or lesser magnitude. The most common was a progressive slight lowering of the T waves in the four principal leads. This change was noted in 49 patients. Inversion of T waves in these same leads was next in frequency, discovered in 36 patients. Diphasia of T waves developed in 15. Premature beats, both auricular and ventricular, were more often heard than recorded in our tracings, only 11 of which revealed these common abnormalities.

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[18] The term "basal" as used here differs somewhat from the usual understanding of that term. In the earlier days no such readings were made. The term as we have used it refers to the lowest "spot" readings, in three or five-year periods, according to the amount of available data. True basal readings would, no doubt, have been even lower.

In the patients with more serious cardiac involvement, major electrocardiographic changes were recorded. Heart block, varying in degree from a prolonged P-R interval to bundle branch block, was noted in seven of the group. In the 20 instances of myocardial infarction, the diagnosis was confirmed by characteristic electrocardiographic patterns.

Roentgenograms of the heart in 61 patients showed no enlargement (10% or less) in 41 and enlargement (15 to 40%) in 20. Lack of strain and wear and tear is still further suggested by roentgenograms of the heart taken at a distance of 7 ft. and fluoroscopic examination. Two-thirds of the patients who had satisfactory x-ray examinations late in the period of observation revealed no significant enlargement of this organ. It should be said, however, that fluoroscopic study almost always revealed a prominent left ventricle. Many showed elongation and tortuosity of the aorta; a few showed calcification of this vessel. Most of those with large hearts had coronary artery disease. However, several patients with known coronary symptoms had normal-sized hearts.

COMPLICATIONS

In trying to account for the long course of these patients, we sought to ascertain whether they had been unusually free from the complications that so often accompany hypertensive vascular disease. Table 4 suggests that they were not. However, it is a fact that these disturbing complications occurred late in the disease with few exceptions, and often they contributed to the terminal event. As seen in table 2, cerebrovascular accident was the cause of death in 14, coronary thrombosis in 8, and congestive failure in 3.

TABLE 4.—*Incidence of Complications*

Complication	Among 100 Patients	Among 29 Dead
Cerebrovascular accident	28	15
Coronary insufficiency	51	21
Coronary thrombosis	20	12
Congestive failure	19	13
Chronic nephritis	12	9

Angina pectoris always warrants caution in the prognosis; yet, 12 of the 51 patients who had this symptom lived many years after the initial attack. One patient had her first severe attack 26 years before her death. In some of these patients, pain occurred irregularly over several years. A few had several attacks followed by long intervals of freedom, indicating, probably, the development of collateral channels. The prognosis for patients with angina pectoris, though always uncertain, is far from hopeless and may be compatible with many years of life. Even coronary thrombosis may be followed by several years of comfortable living, as it was in 7 of the 20 patients who had such a complication. At least six patients who experienced a cerebral vascular accident have lived 2 to 13 years after the first "stroke." One is still alive after 13 years.

Two complications were invariably late in these patients: congestive failure and uremia. The former rarely developed earlier than two years before death. It was always serious. While slight proteinuria was noted at some time or other in 58% of our series, true chronic nephritis developed in only 12 patients and played a prominent part in the death of only one. The progress of renal failure was very slow, but, when it became marked, the end was near.

The presence of any of these circulatory complications warrants a cautious but not hopeless prognosis in all patients with hypertensive vascular disease. Their absence warrants a more favorable outlook. Ten of the 23 patients who have lived 20 or more years with their hypertension never showed any of these symptoms or signs. By contrast, all but one of the patients who have died (and he from cancer of the pancreas) have had one or more of these complications.

SUMMARY AND CONCLUSIONS

A study of 100 patients with benign essential hypertension followed by the authors for 10 to 34 years (average 17+ years) is presented. Seventy-one per cent are still alive, and only five of these are in poor condition. Reasonable explanations for their longevity and generally favorable course seem to be the predominance of the female sex in this series and objective evidence that the circulatory strains were not so great as the generally high levels of pressure recorded suggested.

This evidence suggests that the determination of "basal" diastolic pressures and the careful evaluation of the degree of retinal arteriolar sclerosis are of maximum importance in making accurate prognoses in patients with hypertensive vascular disease. Of lesser importance, perhaps because less readily available, are electrocardiographic and roentgenographic observations of the heart.

This report indicates that essential hypertension may be compatible with a relatively long and effective life. One must give this fact serious consideration in making prognoses and planning therapy, to avoid creating "blood pressure neurotics" and subjecting patients to difficult, expensive, prolonged, and often ineffective treatment.

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Cervical Lymphadenopathy.—Asymmetric enlargement of one or more cervical lymph nodes in the adult is almost always cancerous and usually is due to metastasis from a primary lesion in the mouth or pharynx. The foregoing statement may appear dogmatic, but as a basic principle for the physician's clinical attitude toward cervical tumors it will prove to be both sound and dependable, and will result in the early discovery of many cryptic primary growths. . . . The phrase, usually due to metastasis from a primary lesion in the mouth or pharynx, is wholly true if the examiner is sufficiently alert and experienced to exclude from consideration such benign and readily recognizable tumors as lipoma, branchiogenic cyst, etc., which are not highly suggestive of metastatic cancer. . . . The crux of the problem . . . is the frequent failure to recognize the probable cancerous nature of cervical tumors in the adult and, furthermore, the failure to realize that practically all malignant cervical tumors are metastatic rather than primary in the neck. Unless he understands the basic nature of this clinical phenomenon, the first physician consulted by a patient with a cervical tumor will not examine the mouth and pharynx for a "silent" primary lesion. In other cases, although the possibility of an oral or pharyngeal growth is appreciated, too much dependence is placed on a single—even cursory—examination. As a result, precious time is lost and the primary growth may advance to an incurable stage before it is discovered.—Hayes Martin, M.D., and Claude Romieu, M.D., The Diagnostic Significance of a "Lump in the Neck," *Postgraduate Medicine*, June, 1952.

CORTICOTROPIN (ACTH) IN THE TREATMENT OF ACUTE SUBACROMIAL BURSITIS

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The multiplicity of "successful" treatments advocated for subacromial bursitis indicates that failures are common. Various treatments that have been used are injection of the subacromial bursa with procaine hydrochloride, iron cacodylate injections, short wave local treatment, hot moist packs, immobilization, surgical excision of the bursa, radiation therapy, and manipulation of the shoulder with the patient under a general anesthetic. Radiation therapy has been more or less accepted as being the most efficacious treatment for acute subacromial bursitis. Meyerding and Ivins [1] stated that acute lesions have responded to roentgen treatment in contrast to failure in cases of more chronic lesions. Forty of the 150 patients in their series were treated with roentgen rays. Lattomus and Hunter [2] found that the best results were obtained in the cases of acute disease, either first attacks or acute exacerbations of a chronic condition. Leucutia (in Portmann [3]) stated that the severe pain in the cases of acute subacromial bursitis is relieved by roentgen therapy within 48 hours.

Successful treatment of this condition should accomplish first and foremost relief from the severe pain. The follow-up care should be directed toward obtaining normal or near normal motion of the shoulder joint. The advantages of roentgen therapy are that it is applied easily and is usually effective within 48 hours. However, it is to be noted that at least 50% of patients so treated experience intense aggravation of the shoulder pain and require opiates during this 48 hour period. Another disadvantage to this treatment is that, although relief follows within a period of 24 to 48 hours, a frozen or less obile shoulder joint results unless a carefully planned ogram of physical therapy is soon inaugurated.

The recent dramatic relief of joint symptoms associated with cortisone, hydrocortisone, or corticotropin (ACTH) therapy has stimulated us to study the effects of corticotropin therapy on acute subacromial bursitis. This is usually a self-limiting condition associated with severe pain and disability of the shoulder joint. Since the condition is self-limiting and therefore would require a short period of treatment with corticotropin, none of the complications associated with treatment with this hormone should be encountered. It was therefore tried in the following six patients with various dose schedules.

REPORT OF CASES

CASE 1.—E. O., a white man aged 45, was first seen June 1, 1951. He had had some discomfort in his left shoulder for one week. The pain became intense on May 27, 1951 (four days before treatment was started), after he had lifted approximately 50 lb. (28 kg.). The pain permitted him no sleep at night. There was severe tenderness over the left subacromial bursa. No abduction was possible in the left shoulder. A roentgenogram showed an area of calcification in the bursa. He was given 50 U. S. P. units of corticotropin intramuscularly on two occasions on June 1. When seen three days later, he had normal motion and no pain in the left shoulder joint. He reported that the acute pain in the left shoulder had subsided within a period of three hours after the first injection. He was followed for one month, with no evidence of recurrence. Corrected hourly sedimentation rates were as follows: June 1, 28 mm.; June 4, 21 mm.; June 12, 23 mm.; June 26, 15 mm.; and July 3, 3 mm. At the time of writing there had been no recurrence.

CASE 2.—M. C., a housewife aged 65, was first seen on Sept. 11, 1950. The diagnosis was osteitis deformans (Paget's disease of bone) and a mixed type of arthritis. She was next seen on July 24, 1951, at which time she experienced severe pain in the region of the right shoulder after a day of intensive gardening. Examination revealed severe tenderness over the subacromial bursa and no abduction of the shoulder because of severe pain. She was given 40 units of corticotropin intramuscularly. She reported the following day that the pain had subsided within a period of three hours. There was still 50% limitation of motion of the shoulder joint. She was therefore given 40 units of corticotropin on July 25. Six days later she reported complete relief of all symptoms and a normal range of motion of the right shoulder joint. No roentgenograms were taken. The corrected hourly sedimentation rates were as follows: May 7, 1951 (before the attack of acute subacromial bursitis), 13 mm.; July 25 (one day after the acute attack of bursitis), 26 mm.; and Aug. 21, 14 mm. At the time of writing she had had no recurrence of difficulty in her right shoulder.

CASE 3.—G. B., a white woman aged 35, who did janitor type work, was first seen Nov. 14, 1951, with severe pain in her right shoulder of one day's duration. She awakened at 3 a. m. the morning of Nov. 14 with severe pain in her right shoulder joint. The pain permitted no motion of the right shoulder. Severe tenderness was present over the right subacromial bursa, and no motion was possible in this joint. She was given 80 units of corticotropin intramuscularly, with pronounced relief of pain within three hours. The following day she was able to abduct her right arm to 10 degrees. She was again given 80 units of corticotropin intramuscularly. The following day abduction was possible to 20 degrees. She was given 40 units of corticotropin intramuscularly on Nov. 16 and again on Nov. 17, on which date she was able to abduct her shoulder to 45 degrees. There was no tenderness present and abduction of the right shoulder was normal on Nov. 24. The sedimentation rates remained normal throughout the period of treatment. Roentgenograms of her right shoulder taken Nov. 15 showed an area of calcification subjacent to the deltoid muscle (fig. 1*A*). No calcification was seen in a roentgenogram taken on Dec. 12 (fig. 1*B*). At the time of writing there had been no return of her symptoms.

CASE 4.—P. B., a white man aged 53, was first seen Nov. 15, 1951. He moved a heavy object three days previously and noticed severe pain in the region of his right shoulder. The pain became progressively intense during the next three days. There was exquisite tenderness over the right subacromial bursa, and the slightest touch of his right arm caused the patient to wince with pain. He was given 80 units of corticotropin intramuscular-

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1. Meyerding, H. W., and Ivins, J. C.: Causation and Treatment of Painful Stiff Shoulder: Subdeltoid Bursitis, Periarthritis, Tendinitis and Adhesive Capsulitis, Arch. Surg. **56**: 693 (June) 1948.

2. Lattomus, W. W., and Hunter, L. M.: Roentgen Therapy of Subdeltoid Bursitis: Review of 235 Cases, Delaware State M. J. **21**: 115 (June) 1949.

3. Portmann, U. V., editor: Clinical Therapeutic Radiology, New York, Nelson & Sons, 1950, p. 531.

ly. He was able to abduct his right arm to 60 degrees and had practically no pain the following day. He reported that the pain had subsided within a period of one hour after the first injection. He was given 40 units of corticotropin intramuscularly on Nov. 16. The date of the next dose of corticotropin was Nov. 20, at which time 40 units was given intramuscularly. The patient had complete normal range of motion and no pain the following day. He was able to return to work on Nov. 19. He reported on Nov. 24 that he had been able to work since Nov. 19 without pain. There was complete normal range of motion of the shoulder joints. His corrected hourly sedimentation rates were as follows: Nov. 15, 23 mm.; Nov. 17, 21 mm.; Nov. 20, 18 mm.; and Nov. 24, 18 mm. A total of 160 units of corticotropin had been given.

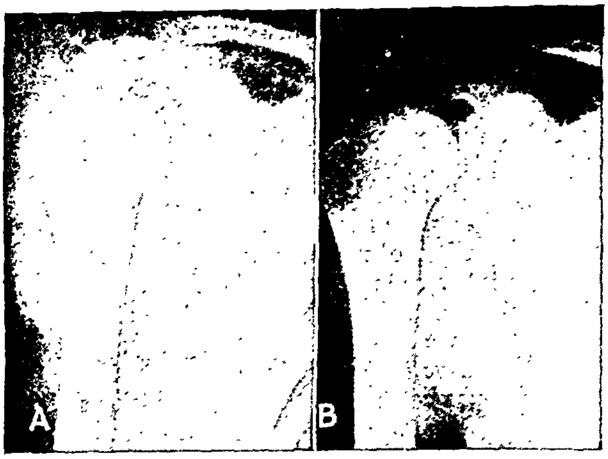

Fig. 1 (case 3).—*A*, calcification in the right subacromial bursa before treatment. *B*, roentgenogram taken one month after the initial treatment, showing the disappearance of calcification in the bursa. The total dose of corticotropin was 240 units. It is usual for calcification to disappear from such bursae in six to eight months after roentgen treatment.

CASE 5.—M. W., a white woman aged 43, was first seen in the office on Oct. 17, 1951. She had had severe disabling pain in the right shoulder one year previously. The right subacromial bursa was aspirated at that time. This resulted in relief of pain, but no increase in joint mobility. She next received roentgen therapy to her right shoulder. The pain in the right shoulder gradually subsided, but pain in the left shoulder, which started after the first roentgen treatment, became more intense. Diathermy directed to the left shoulder joint resulted in no relief of pain. Recently, pain had also returned in the right shoulder joint.

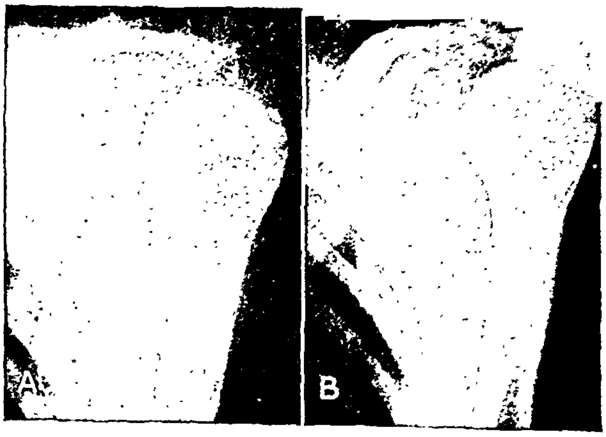

Fig. 2 (case 5).—*A*, calcification in the left subacromial bursa before treatment. *B*, there is little change in calcification of the subacromial bursa three weeks after initial treatment with corticotropin.

Examination on Oct. 17, 1951, revealed a frozen right shoulder joint. Attempts at passive exercise of this joint resulted in severe pain. There was pronounced tenderness over the left subacromial bursa, and abduction was limited to 70 degrees. A roentgenogram of the left shoulder (fig. 2*A*) showed a calcium deposit in the left subacromial bursa. Intramuscular injections of corticotropin were given according to the following schedule:

Oct. 19	20 units
Oct. 20	20 units
Oct. 21	40 units
Oct. 25 and 29	20 units twice daily
Oct. 30	10 units twice daily
Nov. 11	10 units twice daily
Nov. 12	10 units daily
Nov. 13	10 units daily
Nov. 14	5 units daily
Nov. 15	5 units daily

The patient experienced pronounced relief of pain in her shoulder joint about six hours after the first injection. Further relief was experienced by the following day. Abduction of the right shoulder on Oct. 24 was possible to 45 degrees and in the left shoulder to 70 degrees. On Nov. 26, abduction of the left shoulder was possible to 85 degrees and of the right shoulder to 75 degrees. The patient was last seen on Jan. 16, 1952, and reported no recurrence of pain. The range of motion reported above was maintained. Complete range of motion was not possible in the period of observation. The follow-up roentgenograms of the left shoulder showed no change in the calcium shadow (fig. 2*B*).

CASE 6.—K. B., a physician aged 51, awakened early on the morning of Jan. 31, 1952, with soreness and lameness in the right upper arm at the point of insertion of the deltoid muscle. Roentgenographic examination revealed calcification in the right subacromial bursa. Roentgen treatment was given over this area on Feb. 3 and 4, 1952. The pain in the right shoulder became progressively worse after the second roentgen treatment. The physician reported that, on Feb. 5, he took a total of 65 mg. of morphine, 150 mg. of meperidine hydrochloride, some secobarbital sodium, phenobarbital sodium, and acetylsalicylic acid, with no relief of pain. An injection of 25 mg. of hydrocortisone (compound F) was given into the right subacromial bursa on the morning of Feb. 6. The excruciating pain disappeared within a period of three hours after this injection. However, motion of the right shoulder joint was limited to 10 degrees of abduction the following morning, and there was still moderate soreness in the region of the right subacromial bursa. A dose of 40 units of corticotropin was administered intramuscularly on Feb. 7 and again on Feb. 8. Abduction of 45 degrees was present on Feb. 8, and there was only a slight degree of soreness in the region of the bursa. Complete range of motion of the right shoulder joint returned on Feb. 11 and all soreness disappeared. At the date of writing, there had been no recurrence of the condition.

COMMENT

Hollander and co-workers[4] treated three patients with acute and seven with chronic subdeltoid bursitis by local injection of 15 mg. of hydrocortisone. The three patients with acute subdeltoid bursitis responded within a three hour period. The authors stated, "There was complete alleviation of pain and tenderness, and full range of shoulder motion was possible without pain." The relief had persisted for four months in one case and three months in the other two cases at the time their article was written. No other treatment was used. The local injection of cortisone into inflamed bursae in two previous cases produced no such benefit.

Three of their patients with chronic subdeltoid bursitis responded in a similar manner to treatment with a single injection of hydrocortisone. However, in four other cases of chronic subacromial bursitis no relief was obtained even after repeated trial, and in two instances shoulder pain was reported worse for several days after each injection.

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[4] Hollander, J. L.; Brown, E. M., Jr.; Jessar, R. A., and Brown, C. Y.: Hydrocortisone and Cortisone Injected into Arthritic Joints: Comparative Effects of and Use of Hydrocortisone as a Local Antiarthritic Agent, J. A. M. A. **147**: 1629 (Dec. 22) 1951.

SUMMARY

Six patients with subacromial bursitis were treated with corticotropin (ACTH). In five the condition was acute and in one chronic. Roentgenograms of the shoulder were taken in five cases; these showed calcification in the subacromial bursa. Follow-up roentgenograms showed disappearance of calcification within a period of one month in one case and no change in calcification in another case. Complete relief of pain and return to normal function of the shoulder joint was obtained in the five cases of acute bursitis. Complete relief of pain and almost complete return of normal function was obtained in a case of chronic bursitis. We used various dosages of corticotropin in order to determine which dose was optimal. From this study it is suggested that 50 U. S. P. units of corticotropin given twice on the first day and once daily on two successive days is sufficient treatment in the usual case. The ease with which corticotropin may be administered and the clinical response obtained make this method of treatment worth while. Insufficient experience has been obtained with hydrocortisone therapy to determine whether this method is superior. Hydrocortisone must be injected directly into the bursa in order to be effective. This in itself is a problem in some instances. Future experience will indicate the proper role of hydrocortisone in the treatment of this condition. Our present meager experience seems to indicate that corticotropin is more effective than roentgen radiation in the treatment of acute subacromial bursitis.

277 Alexander St. (7).

Treatment of Acute Cholecystitis.—The hazards of acute cholecystitis and its proper treatment are so much greater than the danger of operation at an earlier stage of the disease that every effort should be made to perform cholecystectomy at the time cholelithiasis is first discovered, particularly if it is symptomatic. Serious attacks of acute cholecystitis could thus be largely prevented.

The case fatality rates [in a large series of cases of acute cholecystitis] were significantly affected by the age of the patient and the severity of the acute attack. The fact that the severity of the disease increased with age, and that other age-related factors also contributed to a high fatality rate in the aged again emphasizes the desirability of definitive surgical treatment of cholelithiasis and cholecystitis in the individual at an earlier age.

Serious complications of empyema, gangrene, perforation, and peritonitis were prone to occur, particularly if surgical treatment was delayed. The case fatality rate in patients seen more than 72 hours after onset was apparently significantly greater than that in cases seen earlier. Careful analysis of the deaths revealed that 50% might have been prevented had surgery been employed earlier in the course of the disease.—Paul T. DeCamp, M.D., and others, Timing in the Surgical Treatment of Acute Cholecystitis, *Annals of Surgery*, May, 1952.

CLINICAL NOTES

GASTRIC HODGKIN'S DISEASE

W. Ross Morris, M.D., Washington, D. C.

Hodgkin's disease is one of unknown causation. It is a disease of lymphoid tissues usually affecting the lymph nodes primarily; it also involves the spleen, liver, and, in varying degrees, the lymphatic and reticular structure of almost any organ of the body. It may occur at any age, although it is rare in infancy and old age. In Hodgkin's disease the stomach is involved only infrequently; however, cases have been reported in which the disease was thought to have its origin in the stomach. Such a case may be the so-called isolated gastric Hodgkin's disease, in which there is apparently no involvement other than in the stomach. This is rare indeed. There may be gastric involvement secondary to involvement elsewhere, as in the spleen. Although this is rare it is encountered more frequently than the isolated form. Such a case is reported in this paper with a consideration of the best form of treatment.

REPORT OF A CASE

A white American housewife, aged 71, entered Doctors Hospital on Aug. 4, 1950. On admission she complained of weakness, nervousness, depression, shortness of breath, anorexia, and loss of weight. The patient had a history of marked anemia of seven or eight years duration despite dietary treatment, supplemented by vitamins, injections of liver and iron, and several transfusions. The anemia was very severe at times. After a careful study, including a negative roentgenographic examination of the gastrointestinal tract, a tentative diagnosis of pernicious anemia had been made.

Physical Examination.—The patient had a pale, warm, and moist skin. The blood pressure was 146/86 mm. Hg, the pulse rate 96, and the temperature 98.6 F. There was moderate generalized arteriosclerosis. Examination of the mouth showed upper and lower plates, and the tongue appeared redder than normal. There was a well-healed scar from a thyroidectomy and a well-healed scar from a left simple mastectomy. A grade 2 systolic murmur was heard over the pericardium, but it was not transmitted. There was slight tenderness on deep epigastric pressure, but no abdominal organs or masses were palpable. There was a shortening and slight atrophy of the left leg and a limitation of motion of the left hip. No glandular adenopathy was found.

Laboratory Examination.—Blood studies on Aug. 5, 1950, showed a white blood cell count of 17,500 per cubic millimeter: differential count, young forms 2%, band forms 3%, segmented forms 78%, neutrophils 83%, eosinophils 1%, basophils 0, monocytes 0, and lymphocytes 16%; red blood cell count 2,400,000 per cubic millimeter; and hemoglobin of 6 gm. (spectrophotometer method). The urine was normal. A roentgenographic examination of the gastrointestinal tract made several months previously revealed no pathological condition except a diverticulum of the second portion of the duodenum. A roentgenographic examination of the gastrointestinal tract made on Aug. 5, 1950 showed an ulcerative lesion 5 × 10 cm. on the greater curvatur side of the pars media. This was thought to be a large malignan growth, probably of the lymphoblastoma group. The diverticulu of the second portion of the duodenum was still present. In th gastric analysis the fasting specimen showed no free hydrochlori acid. There was no free hydrochloric acid after histamine a ministration.

From the Doctors Hospital.

A transfusion of 500 cc. of whole blood was given daily for three days. The patient improved greatly, and a blood test showed the hemoglobin to be 56% and the red blood cell count 3,010,000 per cubic millimeter.

Operation.—On Aug. 9, 1950, the abdomen was explored through an upper left paramedian incision converted into a T incision. The gallbladder was chronically inflamed and filled with stones. Along the lesser curvature and posterior wall of the stomach, about the middle third, there was a large ulcerated lesion, which appeared to be malignant; it was about twice the size of a silver dollar. There was no evidence of metastasis in the liver, but the glands in the gastrocolic and gastrohepatic omentum and around the celiac axis were enlarged and apparently metastatically involved. The spleen was very large (about the size of a small football) and contained large nodules, particularly on its posterior and superior surfaces where it was adherent to the peritoneum and the diaphragm. A subtotal gastrectomy and gastrojejunostomy (anterior Polya type) were done, with the removal of all the omentum, the gastrohepatic ligament, and the spleen. In doing the splenectomy it was necessary to remove a portion of the lateral part of the left diaphragm. After the lung was inflated by the anesthetist the opening in the diaphragm was closed. All involved tissue appeared to be removed except for the glands around the celiac axis. The wound was closed in layers around a drain placed in the splenic bed. The pathological examination revealed a malignant lymphoma, Hodgkin's type. The tumor appeared to be primary in the spleen with involvement of the stomach and regional lymph nodes.

Postoperative Course.—The patient had an unusually favorable postoperative course and was discharged from the hospital in good condition. She had gained weight and was eating well. A blood examination showed a white blood cell count of 11,300 per cubic millimeter: differential count, young forms 0, band forms 5%, segmented forms 84%, neutrophils 89%, eosinophils 0, basophils 0, monocytes 0, and lymphocytes 11%; red blood cell count 4,300,000 per cubic millimeter; and hemoglobin 13 gm.

Approximately three weeks postoperatively the patient was given a course of roentgen ray therapy (2,000 r) in an effort to destroy the glands that had been left in the region of the celiac axis. This produced marked radiation sickness evidenced by anorexia, nausea, loss of weight, and decrease in the blood cell count. She recovered gradually from this and continued in fairly good condition until January, 1951 (approximately five months postoperatively). At this time increasing weakness, loss of weight, enlarged glands in the left axilla and left cervical region, a left pleural effusion, and anemia developed. On Jan. 3, 1951, a blood examination showed a red blood cell count of 3,200,000 per cubic millimeter: differential count, young forms 1%, band forms 3%, segmented forms 80%, neutrophils 84%, eosinophils 1%, monocytes 2%, and lymphocytes 13%; white blood cell count 13,400; and hemoglobin 10 gm. Despite nitrogen mustard therapy, blood transfusions, removal of fluid from the left chest, and other procedures, the patient's progress was gradually downhill. On May 11, 1951, the patient was transferred to the Marine Hospital in Baltimore, where she died in June, 1951, from generalized Hodgkin's disease.

REVIEW OF THE LITERATURE

A review of the literature on gastric Hodgkin's disease revealed this to be a rare manifestation of the disease, whether it be found only in the stomach (so-called isolated Hodgkin's disease of the stomach) or whether it be gastric involvement secondary to involvement of the spleen or elsewhere. A clinical entity characterized by involvement of the glandular system, spleen, and, at times, other organs was first described by Thomas Hodgkin [1] in 1832. Since that time much has been learned about the disease; however, its cause remains unknown. There is no specific cure, nor even a satisfactory palliative treatment, known. In 1913 Schlagenhaufer [2] reported the first case of Hodgkin's disease in which the specific lesion was found in the stomach. Mead,[3] in a review of 16,254 autopsies, found only 3 cases of isolated involvement of the gastrointestinal tract. In a study of 212 cases of Hodgkin's disease Goldman [4] found the stomach to be involved in only 1 case. In a review of 54 cases of Hodgkin's disease Burger and Lehman [5] found only 2 cases with stomach involvement. Jackson and Parker [6] reported 3 cases of gastric involvement in a study of 174 cases. Goldman and Victor [7] reported only 1 case of gastric Hodgkin's disease in a series of 319 cases. In a review of 18 cases of the disease seen at Doctors Hospital, Washington, D. C., there were no cases of Hodgkin's disease of the stomach. These cases were diagnosed by postmortem examination, biopsy of a gland, or roentgenographic examination.

SYMPTOMATOLOGY AND DIAGNOSIS

The symptomatology of abdominal Hodgkin's disease is indefinite, varied, and bizarre. There may be present in varying degree any one or several of the following symptoms: anorexia, abdominal pain, nausea, constipation, hematemesis, persistent occult blood in the stools, fever, diarrhea, secondary anemia, leukopenia, and cachexia. There are no pathognomonic symptoms. Hayden and Apfelback [8] classified the symptoms in three groups: (1) ulcerative enteritis and colitis, (2) gastric carcinoma, and (3) intestinal obstruction. Primary anemia might also be added. Bockus [9] stated that a syndrome similar to that of duodenal ulcer has been reported in several cases. In this case the patient presented symptoms indicative of pernicious anemia for some time; finally the picture became that of a gastric malignancy.

ROENTGENOGRAPHIC STUDY

In cases of Hodgkin's disease of the stomach the roentgenographic findings have usually been interpreted as being indicative of carcinoma and, less frequently, of ulcer. Jungmann [10] and others, however, have emphasized a characteristic roentgenographic finding: persistence of peristalsis, even though sluggish, despite extensive involvement of the mucosa of the stomach. Now the correct diagnosis is made frequently by the roentgenologist from the roentgenographic appearance alone. In this case the roentgenologist stated that the gastric lesion had the appearance of a "very large malignancy of the lymphoblastoma group." The diagnosis of Hodgkin's disease of the stomach, however, is difficult and is usually not made before operation or necropsy. A final absolute diagnosis can be made only on histological section; there is no distinct clinical picture.

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[1] Hodgkin, T.: On Some Morbid Appearances of the Absorbent Glands and Spleen, Tr. Med.-Chir. Soc., London **17**: 68-114, 1832.

[2] Schlagenhaufer, F.: Über Granulomatosis des Magendamtrokts, Zentralbl. f. allg. Path. u. path. Anat. **24**: 965-966, 1913.

[3] Mead, C. H.: Chronic Lymphatic Leukemia Involving the Gastro-intestinal Tract, Radiology **21**: 351-365, 1933.

[4] Goldman, L. B.: Hodgkin's Disease: An Analysis of 212 Cases, J. A. M. A. **114**: 1611-1616 (April 27) 1940.

[5] Burger, R. E., and Lehman, E. P.: Hodgkin's Disease: Review of 54 Cases, Arch. Surg. **43**: 839-849 (Nov.) 1941.

[6] Jackson, H., Jr., and Parker, F., Jr.: Hodgkin's Disease: Involvement of Certain Organs, New England J. Med. **232**: 547-559, 1945.

[7] Goldman, L. B., and Victor, A. W.: Hodgkin's Disease: Salient Clinical Features and Relative Value of Various Methods of Treatment Based upon Study of 319 Cases, New York State J. Med. **45**: 1313-1318, 1945.

[8] Hayden, H. C., and Apfelback, C. W.: Gastrointestinal Lymphogranulomatosis, Arch. Path. **4**: 743-770 (Nov.) 1927.

[9] Gastro-Enterology, edited by H. L. Bockus, Philadelphia, W. B. Saunders Company, 1944, vol. 1, pp. 755-757.

[10] Jungmann, H.: Hodgkin's Disease of the Stomach, Brit. J. Radiol. **16**: 386-387, 1943.

TREATMENT

As has been stated previously, Hodgkin's disease is usually a generalized disease; an occasional case reveals a pathological condition that is confined largely to the stomach. Cases have been reported in which postmortem examination confirmed the gastric limitation of the disease. In some of these cases perigastric node involvement or extension into the pancreas, or both, is found. It may be very difficult to demonstrate the extragastric lesion at operation. For these reasons an attempt to cure or prolong life by surgical intervention may be thought to be contraindicated. At present there is no satisfactory treatment known for this disease. The use of roentgen ray therapy, the use of nitrogen mustard, or the use of these two methods combined give palliative results in some cases; however, the use of these methods does not result in a cure, and they are especially ineffective in cases having gastric involvement. It should be remembered that generalized Hodgkin's disease may be asymptomatic for many years, while involvement of the stomach may result in death long before this would have occurred otherwise. This disease in the stomach causes loss of appetite, digestive disturbance, and loss of blood; these conditions speed the downward progress of the patient. Gastric resection, even though it is not curative, relieves these symptoms, makes the patient more comfortable, and prolongs life; I believe surgery is indicated and justified.

A review of the literature revealed approximately 43 cases of gastric Hodgkin's disease in which the patient was treated by surgical excision; perigastric node involvement was not found in all cases. A few patients died in the immediate postoperative period, and the survival time in the majority of cases varied from 5 months to 13 years.

Marshall and Brown[11] made a study of 406 patients who had been operated on at the Lahey Clinic from 1941 to 1945 for malignant disease of the stomach. They found that 16 patients (4% of the group) had noncarcinomatous malignant disease of the stomach, and 14 patients (3.4%), or 1 in every 30 cases, had malignant lymphomas; of these there were 8 cases of the localized form of gastric Hodgkin's disease. They came to these conclusions. 1. Malignant tumors of the stomach were relatively frequent. 2. The prognosis for patients with malignant lymphomatous tumors of the stomach was probably better than that for patients with gastric carcinoma. 3. Treatment should be radical surgery, as it is impossible to make a definite diagnosis of a lymphoid tumor before operation. Radiation therapy should be recommended following resection and should be recommended for those cases diagnosed inoperable after a laparotomy and biopsy. 4. A biopsy should always be obtained for definite identification of malignant gastric tumors considered inoperable at laparotomy, since there have been long-term survivals reported for patients with tumors of the lymphoma group following radiation therapy alone.

In 1950 Sandick[12] reported five cases of gastric Hodgkin's disease and reviewed the literature. He found only two cases in which postmortem examination confirmed the absolute limitation of the disease to the stomach. In our patient the Hodgkin's disease of the stomach was secondary to the splenic involvement, and there was no further evidence of disease except in the perigastric nodes. A subtotal gastric resection, excision of regional nodes, and a splenectomy were done. No similar case was found in the literature.

SUMMARY AND CONCLUSIONS

The literature on gastric Hodgkin's disease is reviewed briefly. There are no diagnostic symptoms. Roentgen findings may be quite indicative. A patient with Hodgkin's disease with gastric involvement treated by subtotal gastric resection is reported on. It is suggested that Hodgkin's disease of the stomach should be treated as any other gastric malignant disease: by surgical extirpation.

The case reported in this paper illustrates further that Hodgkin's disease may involve the stomach. When located in this area, it may be an isolated gastric Hodgkin's lesion or it may be secondary to a primary involvement in the spleen or elsewhere. The patient's life may be prolonged greatly by surgical excision of the gastric lesion, any diseased glands, and the spleen, if necessary. Results from some of the cases reported indicate that a cure may even be obtained. Therefore, it appears that the treatment of choice in gastric Hodgkin's disease is surgical extirpation.

1801 Eye St., N. W.

[11] Marshall, S. F., and Brown, L.: Primary Malignant Lymphoid Tumors of the Stomach, S. Clin. North America **30**: 885-892, 1950.

[12] Sandick, H.: Hodgkin's Disease Involving the Stomach, Gastroenterology **15**: 135-143, 1950.

ABSENCE OF COMMON BILE DUCT

Allan E. Sachs, M.D., Seattle

Anomalies of the biliary ducts are a constant source of concern to physicians performing surgery in that area. Numerous variations in the position and length of the cystic duct as well as the cystic artery have been described in detail. Mention has frequently been made of aberrant hepatocystic ducts; however, absence of the common bile duct, discussed in the following case report, has been reported briefly only once before, when it occurred in an infant who died because of this anomaly. The ducts were all atretic.[1]

This anatomic variation, if it had not been recognized in the usual technical performance of cholecystectomy, would surely have led to death or unfortunate complications. The maneuver emphasized by Gatch[2] of tracing the biliary ducts from within after emptying the gallbladder was helpful in tracing the anomaly. This procedure is a sound, valuable aid in preventing surgical accidents in the biliary ducts. I have seen no untoward complications resulting from this procedure, and I have been helped on several occasions in tracing ducts by probing from within.

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[1] Flint, E.: Abnormalities of the Right Hepatic Cystic, and Gastro-Duodenal Arteries, and of Bile-Ducts, Brit. J. Surg. **10**: 508, 1923.

[2] Gatch, W. D.; Battersby, J. S., and Wakim, K. G.: Nature and Treatment of Cholecystitis, Tr. West. S. A. (1945) **53**: 380-394, 1946.

REPORT OF A CASE

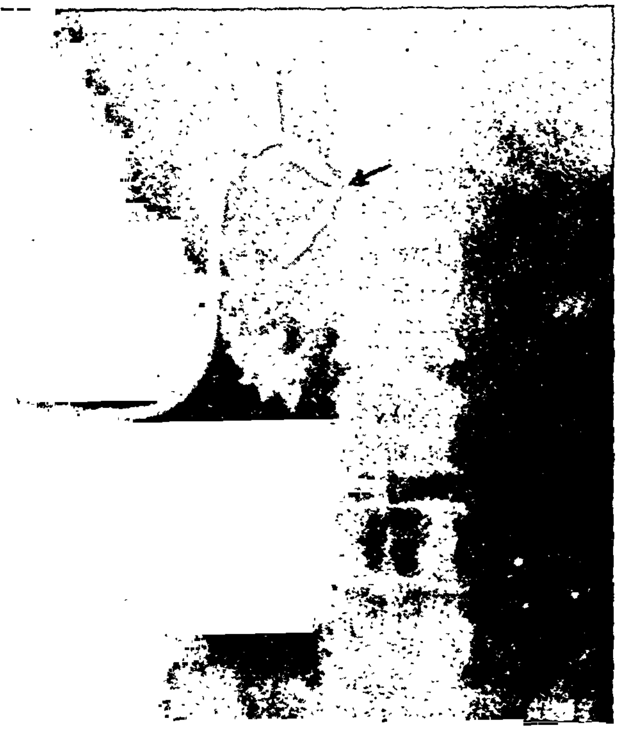
A 54-year-old schoolteacher was admitted to Group Health Hospital on Oct. 28, 1949, with the following history. Severe pain had developed in the right upper abdomen two days prior to admission. This was accompanied by nausea and one episode of vomiting. There was associated pain between the scapulas. At the onset, there had been diarrhea, but the stools then became clay-colored and floated on water. She noticed slight jaundice and pruritus. On questioning, it was learned that she had experienced pain of a milder nature during the last two years. Along with this, she had flatulence and constipation. Gallbladder disease had been suspected two years before, and, at that time, a Graham test showed no concentration of dye.

Physical examination revealed a well-developed, somewhat obese, white woman. Her temperature was 99 F orally, her pulse rate 76, and her blood pressure 124/80 mm. Hg. Positive findings included a slight icterus of the skin and conjunctiva. There was slight right upper abdominal tenderness but no rigidity. Laboratory tests revealed an acid urine with 50 mg. per liter of albumin; urobilinogen was 1:20; the white blood cell count was 8,900, with normal differential count; and the sedimentation rate was 22 mm. per hour. The total protein level was 5.8 mg. per 100 cc.; the albumin was 3.8 mg. per 100 cc.; the globulin was 2.0 mg. per 100 cc.; the serum bilirubin was 6.3 mg. per 100 cc.; the serum alkaline phosphatase was 21.5 King units; and the cephalin flocculation was negative in 24 hours. The provisional diagnosis was chronic cholecystitis with cholelithiasis and choledocholithiasis.

On Nov. 1, 1949, after suitable preparation with fluids, glucose, and vitamin K, the patient was operated on. The anesthetic, a tetracaine (pontocaine®) hydrochloride-dextrose mixture, was administered intraspinally. A right paramedian incision was made, and the gallbladder was visualized. It was distended, and the walls were inflamed and contained areas of purulent infection. Stones could be palpated in the cystic duct. The cystic duct was dissected free, and the cystic artery was identified. An attempt was then made to visualize the common duct; however, the duct could not be found in its usual location, and the hepatic duct was also absent in the gastrohepatic ligament. On dissection, the portal vein and the hepatic artery were found. The cystic duct was then dissected toward the duodenum; it was not dilated and did not enter a common duct. In order to study the anatomy before clamping the cystic duct more accurately, the fundus of the gallbladder was opened. Bile, pus, and pigment stones were removed from the gallbladder. The empty gallbladder was then probed from within with a small catheter. The catheter passed through the cystic duct easily and continued into the duodenum; however, no hepatic ducts could be found traversing backward from the cystic duct to the liver. On further probing, two hepatic ducts were found entering the midportion of the gallbladder. These ducts could be easily probed retrograde into the right and left lobes of the liver.

In order to perform a cholecystectomy, a bile duct anastomosis would have had to be done in the presence of acute infection. It was therefore decided that it would be wiser to do a cholecystostomy after removing the gallstones. A catheter was sewed in the gallbladder with pursestring sutures and brought out through a stab wound. After the cholecystostomy, the patient made an uneventful recovery. The catheter drain functioned well. After three weeks, the bile drainage was clear, and a direct cholangiogram was done with iodized oil (lipiodol®). The roentgenogram showed the anomaly clearly. After this study was completed, the drain was removed, and the wound healed promptly. The patient has now been followed for two years, during which time she has been asymptomatic.

This case is of interest because it is a rare but important deviation from the normal anatomy. It is interesting to note that the jaundice was easily explained by stones blocking the cystic duct, which, in this patient, was also serving as a common duct. In reviewing the literature for similar conditions and embryology of the biliary tract, one finds that such an anomaly might be expected. The gallbladder develops as an evagination from the common duct, and it could carry the hepatic ducts out from the common duct. It is impossible to be sure whether this case represents an absence of the common bile duct per se or the absence of a common hepatic duct. In certain species of reptiles, in most birds, and in one mammal, a type of otter, Lutra vulgaris, a somewhat similar condition exists.[3] A hepatoenteric duct drains directly into the duodenum, and a hepatocystic duct drains directly into the gall bladder. The latter duct has no direct communication with the duodenum. This patient had two hepatocystic ducts, possibly, with no common hepatic duct.



Direct cholangiogram three weeks after operation; the iodized oil injected through the catheter reveals the gallbladder with the hepatic ducts entering its midportion. The arrow points to the cystic duct. A single duct leads from the cystic duct to the duodenum.

SUMMARY

1. An unusual but important anomaly of the biliary tract is discussed.
2. The value of tracing the biliary ducts from within the gallbladder lumen is reemphasized.

501 Medical-Dental Bldg.

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[3] Huntington, G. S.: Anatomy of Human Peritoneum and Abdominal Cavity, Philadelphia, Lee Brothers & Company, 1903.

USE OF SILICONE OILS IN MAINTAINING SURGICAL INSTRUMENTS

Frank W. Crowe, M.D., Ann Arbor, Mich.

Since the introduction of the organosilicon products into industry in 1943, the potentialities of these compounds appear to be unlimited. Their more recent introduction into cutaneous medicine [1] suggests many uses for their water repellent and lubricating properties in general medicine and surgery.

The physical and chemical differences of the silicones and organic compounds are marked, because the silicones have a quartz-like skeleton consisting of alternating silicon and oxygen atoms rather than the carbon atom framework of the organic compounds.[2] The silicones are water repellent, superior lubricants, which are chemically and physiologically inert and show marked surface adhesiveness. Physical properties [3] are remarkable in that the freezing point for the 500 centistoke (viscosity) DC 200 fluid is –50 C (–58 F). The boiling point for this fluid is 200 C (392 F), and the viscosity-temperature coefficients (viscosity-temperature coefficient equals $1-\frac{\text{viscosity at 210 F}}{\text{viscosity at 100 F}}$) are in the range of 0.60 to 0.625, showing that there is little change in viscosity with elevation of temperature. Volatility is less than 2% after 48 hours at 200 C (392 F). There is no significant toxicity from the silicones in the amounts used in this study. They have been fed to laboratory test animals in doses as high as 2% of their body weight without discernable ill effects.[4]

It is the standard procedure in many hospitals for surgical instruments to be cleaned in warm, soapy water after each operation. After drying, the individual instrument is usually oiled with high-grade petroleum lubricating oil. The clean instruments are then boiled and wrapped in sterile covers or wrapped and autoclaved in preparation for the next surgical procedure. In either method, the protective lubricating film is reduced or destroyed by the moist heat, and the free action of the instrument is frequently either impaired or lost. Much valuable time is spent in repetitious oiling of instruments, and inferior results are often obtained.

During the initial work with silicote® (a petrolatum base ointment containing 30% of the 60,000 centistoke DC 200 silicone fluids) many possibilities for utilizing the repellent and lubricating properties were apparent to the investigators. Among these was the application of pure silicone oils to surgical instruments prior to boiling or autoclaving to insure free motion of the instrument after sterilization. Initial coating of the instruments by using 60,000 centistoke DC 200 silicone oils proved reasonably satisfactory to both the surgeon and the operating room nurses, but it was difficult to handle because of its high viscosity.[5] It did, however, show that the oiling of instruments could be reduced to every fourth or fifth boiling without impairing the efficiency of the instruments.

For the past three months a preparation called "surgicote," a mixture of silicone oils, has been used in the University Hospital operating rooms, in the Dermatology Out-Patient Clinic, and in the University of Michigan Student Health Service Dermatology Clinic on all surgical instruments. The nurses and aids cleaning the instruments, the physicians using them, and the central supply room that was responsible for their care all felt that there was a significant improvement in ease of care and efficiency of the instruments while this agent was being tested.

The procedure used in caring for all instruments was as follows. All old, rusted, or corroded instruments were first treated in a rust removing solution then washed and dried. A thin coat of the "surgicote" was applied with an applicator stick to the entire instrument, making sure that the joints or fulcrum had been coated with the oil. A cotton flannel rag was used to wipe off the excess and to insure even distribution over the instrument. New instruments were treated in this manner as soon as they were obtained. It was observed that the older instruments remained rust-free and showed no signs of corrosion when this agent was used. Both the old and new instruments showed a freedom of action even after the most vigorous sterilization. Cleaning time was appreciably reduced, and the instruments required less oiling, often going through three or four sterilizations without further oiling.

Barondes [6] has also found the silicone fluids ideal for instrument sterilization; however, he used the method suggested by Crowley and Ostrander,[7] in which the instruments are placed directly in the hot silicone oils for sterilization. Although Barondes points out that the loss of fluid caused by carry-out on the instruments is minimal, it is felt that such a procedure was more practica in the small office than in a hospital because of this flui loss.

SUMMARY

A new agent is described in the care of surgical i struments. This agent is principally a silicone oil and an effective lubricant and water repellent while bei inert chemically and physiologically. It is nontoxic to t tissues with which it might come in contact. The ti involved in the care of surgical instruments is reduc with this agent, and the efficiency of the instruments appreciably increased.

The "surgicote" used in this study was furnished by the Silicote Corporation, Oshkosh, Wis.

From the Department of Dermatology and Syphilology, University Hospital, University of Michigan.

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3. Dow Corning Corporation Bulletin, No. 2001, Midland, Mich., 1948.
4. Rowe, V. K.; Spencer, H. C., and Bass, S. L.: Toxicological Studies on Certain Commercial Silicones and Hydrolyzable Silane Intermediates, J. Indust. Hyg. & Toxicol. **30**: 332 (Nov.) 1948.
5. Talbot, J. R.: Personal communication to the author, 1951.
6. Barondes, R. de R.; Judge, W. D.; Towne, C. G., and Baxter, M. L.: Silicones in Medicine: New Organic Derivatives and Some of Their Unique Properties, Mil. Surgeon **106**: 379 (May) 1950.
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PERIODIC SIALORRHEA

REPORT OF A CASE

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and
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Attention has been called elsewhere to an assortment of periodic disorders of unknown cause that recur at more or less regularly predictable short intervals, last for years, do not respond to therapy, and do not affect the patient's general health.[1] The disorders were classified according to their respective outstanding features: fever, abdominalgia, arthralgia, neutropenia, purpura, and edema. It was predicted that interest in the problem and further search would reveal other bizarre examples of periodic disorders to add to the list. The purpose of this paper is to describe such a case and to refer to reports of similar ones published elsewhere.

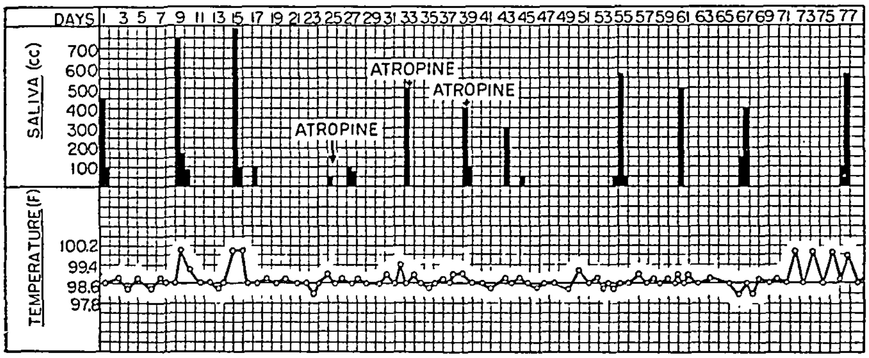
REPORT OF A CASE

Mrs. E. P., aged 44, had pain in her shoulders, arms, fingers, and toes in September, 1948. Her case was studied at a hospital, and a diagnosis of arthritis was made. Several teeth were extracted, against her protest, and excessive salivation was noted for one day. Thereafter, periods of salivation lasting a day occurred at about monthly intervals, until January, 1949, when the rest of her teeth were extracted. The intervals between the spells of salivation gradually shortened to 7 to 10 days. According to the patient's and her husband's statements, as much as 4 qt. (3,785 cc.) of saliva would be expectorated into a basin in a period of 24 to 36 hours. The flow was distressing, especially at night when saliva would flood over the pillow while the patient slept or the flow would keep her awake. She lost 50 lb. (22.7 kg.) during the next year, partly because of malnutrition owing to her inability to masticate. She has one son, aged 20, and has had four miscarriages. Her menstrual history is normal.

A study at another hospital made late in 1949 revealed she had hypotension, with 90 mm. Hg systolic and 60 mm. Hg diastolic pressure, and persistent eosinophilia of 23 to 44%. Polyarteritis, trichinosis, Addison's disease, rheumatoid arthritis, malnutrition, and sialorrhea were considered as diagnoses, but no conclusion was reached. Many consultations, special studies, and laboratory tests, including the Cutler-Powers-Wilder test, a Trichinella skin test, and roentgenographic examination, were all unrevealing. The patient's condition persisted, and in 1950, she came under our observation in the hospital for three months. Her chief complaints were spells of excessive salivation, occurring about once every week or 10 days and pains in her shoulders and calves, which became worse at night. Her intelligence was low, she was reticent and suspicious, and minor contradictions and inaccuracies were evident in the history she gave. Physical examination showed a normally developed, thin woman, 64 in. (162.6 cm.) in height and 123 lb. (55.8 kg.) in weight. Both submaxillary salivary glands were enlarged and palpable; the right was larger than the left, measuring about 3 by 4 cm. No evidence of arthritis could be seen in her roentgenograms. The muscles of her limbs were atrophic from self-induced inactivity. Psychiatric investigation revealed that the patient's childhood had been unhappy. Marriage at the age of 16 had caused more unhappiness. The patient is unkempt, and, since the onset of arthralgia in 1946, she has neglected housework, preferring to do nothing much of the time. Her outlook is severely restricted; she is hostile, dependent, and incapable of facing reality. She had made several visits to faith healers to obtain relief. Her response to examination and to questions indicated the existence of conversion hysteria. She complained of arthritis in the absence of objective evidence of the disease, while her other troubles were accepted passively. She refused to submit to an interview under narcosis. Her periodic bouts of salivation were thought by the psychiatrist to be psychically determined.

Results of laboratory studies were as follows: erythrocyte count, 4 million per cubic millimeter; hemoglobin, 12 gm. per 100 cc.; leukocyte count, between 7,000 and 9,000, with constant eosinophilia of 30 to 44%; the hyperplastic marrow contained many eosinophilic cells and their precursors; sedimentation rate, 29 mm. in 60 min. (Cutler method); basal metabolic rates, −27 and +4; the blood pressure varied from 80 to 130 mm. Hg systolic and from 40 to 80 mm. Hg diastolic; the pulse rate varied from 70 to 90 beats per minute. The temperature is shown in the figure. Roentgenograms revealed no abnormalities of the viscera and general demineralization of the bones. A sialogram showed a normal system of ducts in the submaxillary salivary glands and that the right gland was larger than the left. In an electroencephalographic examination done in a period between episodes of salivation, low voltage and abnormally fast activity were recorded, but no focal cerebral dysrhythmia was shown.

As shown in the figure, 11 episodes of sialorrhea were observed in 77 days, occurring at intervals of 4 to 12 days, the average being 7.6 days. The long periods after the 7th and 10th episodes indicate irregularities in the rhythm of recurrence, or that 2 episodes were skipped, or that inapparent episodes occurred in the regular rhythm of about 6 day intervals. The last possibility is suggested by slight rises in the patient's temperature that were evident on the 50th and 71st days. Similar fever accompanied or preceded most of the recorded episodes.



Schedule showing eleven periodic episodes of sialorrhea and fever in this patient. Note that atropine therapy greatly retarded the flow on one occasion.

An episode usually is preceded by a "scratchy sensation" of the eyes and a sense of fulness in the neck. The submaxillary glands, which are large, become larger, tender, and visibly swollen. Salivation begins abruptly and continues for 24 to 36 hours, occasionally ceasing for a period of several hours to a day and then resuming. Soreness in the mouth, headache, and pain in the chest are noted at times. As shown in the figure, the saliva collected in a container measured from 350 cc. to almost 1,000 cc. in different episodes. Much saliva was lost to measurement by swallowing and by drooling during sleep. The nocturnal flow is especially distressing to the patient. On inspection saliva could be seen welling in spurts from the sublingual ducts. Pressure on a swollen gland increased the flow. The secretion from the parotid glands seemed to be normal in amount. The fever that precedes or accompanies these episodes may be caused by the distention or overactivity of the glands, or, as in other periodic disorders, may be another aspect of the disorder. Occasionally, according to the patient, the prodromal symptoms mentioned may occur on time with slight or no salivation. During several episodes, slight vaginal bleeding was observed apart from four normal menstrual periods that took place during our study.

The patient left the hospital in August, 1950. Episodes continued unchanged, and she was induced to return in January, 1951, for a further study period of 53 days. Seven episodes of salivation were observed, spaced at intervals of 5 to 11 days (average 6.5 days). The largest amount of saliva collected in one period was 1,700 cc. and the smallest, 150 cc., but as before, much saliva was lost during sleep. The temperature, as in the previous study, rose slightly over 98.6 F during most of the episodes. All routine laboratory studies again gave normal results.

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[1] Reimann, H. A.: Periodic Disease, Medicine **30**: 219-245, 1951.

Experimental Studies.—Because of the patient's suspiciousness, apprehension, and hostility to certain personnel, it was only with persuasion that she submitted to tests. Permission to perform biopsy of an affected gland was refused. It was desired to anesthetize the chorda tympani by injecting cocaine into the middle ear to observe the effect of eliminating stimuli over the parasympathetic nerves to the submaxillary and sublingual glands. The otic ganglion of the parotid gland could have been influenced by infiltrating the temporal fossa, or the effect of blocking the sympathetic nerve supply could have been observed by anesthesia of the superior cervical ganglion. These procedures were not permitted. The only suggestive evidence of an increased parasympathetic tone during the episodes were the accompanying slight rises in temperature. The pulse rate did not increase in frequency and the pupils did not change in diameter.

The saliva was mucinous and faintly opalescent. Its viscosity, measured with the Hess apparatus on many occasions during episodes and the intervals between them, varied within the normal range of 1.4 to 2.8 without relation to the amount. The pH varied from 6 to 7.7, usually tending toward greater alkalinity when the flow was copious. Measurements of the amount of amylase gave highly variable amounts from 600 to 80,000 units per 100 cc., the amount having no relation to the occurrence of the episodes. The amount of urea varied from 8 to 26 mg. per 100 cc. No eosinophilic cells were observed.

Four days after an episode the patient was given 10 mg. of pilocarpine hydrochloride by subcutaneous injection. Five minutes later salivation began and lasted 3 hours, a total of 200 cc. being secreted. An injection of isotonic solution of sodium chloride as a placebo failed to cause salivation. Pilocarpine hydrochloride injected during an episode apparently did not augment the flow. The flow stopped 10 minutes after 0.6 gm. of quinine sulfate was given orally during an episode and resumed in its usual volume after 4 hours. On another occasion, the same dose was given at three hour intervals with no effect. On one occasion, 50 mg. of methantheline (banthine®) bromide was given at 3 hour intervals after salivation had been in progress for several hours. Salivation was arrested after only 150 cc. of saliva had been collected. Seven days later, methantheline bromide given in similar amounts again apparently reduced the amount of saliva secreted to about 100 cc. The patient refused further therapy because the drug made her feel ill. The effect of atropine on the amount of saliva expelled is illustrated in the figure. The patient had eosinophilia of the blood and marrow as before, with 3,000 eosinophils per cubic millimeter. She was given an injection of 0.3 mg. of epinephrine, and 4 hours later the eosinophil count was 2,800 per cubic millimeter. On two occasions, the injection of 25 mg. of corticotropin failed to alter the count. Surgical removal of the affected glands or roentgenotherapy was suggested by consultants, but the patient refused further study and left the hospital.

COMMENT

Only one other reference to a nearly identical case of periodic sialorrhea was found in the literature; it was originally described in an inaccessible essay written by Emden in 1897.[2] It occurred in a woman, aged 54, who had, at first at long intervals and later regularly every eight days, bilateral swelling of the submaxillary and sublingual salivary glands. The swelling process would subside after three days, with expulsion from the salivary glands of fibrinous plugs and with profuse salivation. The possibility of a secretory neurosis was considered. Since 1879,[3] about 44 cases in adults and many in children of recurrent swelling of the parotid glands have been described.[4] Mention seldom was made of the submaxillary and sublingual glands. In many cases pyogenic infection, apparently caused by obstruction of the ducts, swelling of the glands, and secondary bacterial invasion, complicated the picture and interfered with clear-cut evidence of regular periodicity. Little attention was given to exact periodicity of glandular swelling, except for casual mention of recurrences in days, weeks, months, or two or three times a year over many years.[5] Sialorrhea was noted only in Vogeler's patient [6]; xerostomia was prominent in others. In one case, colic accompanied the glandular swellings.[7] The disorder was hereditary in a boy whose mother and grandmother had similar complaints.[8] Of Payne's 19 patients, 16 were women.[5b]

In cases described before 1935, infection was thought to be the cause, but later, as in other periodic disorders, allergy was suspected.[9] Of the 44 cases found in the literature by Bass, allergic manifestations were present in 12.[4] Bigler, however, saw no relation to allergy in his patients.[5c] According to Weber,[10] acute bouts of ptyalism are induced at times by gastrointestinal disturbances and muscular exercise. He suspected an allergic influence to be at fault, since persons so afflicted often have relatives who have allergic disorders. There seems to be no doubt that, at times, allergy may incite recurrent salivary disturbances. In some instances allergy or infection may serve as an inciting factor, but neither can account for uniform episodes of the disease regularly recurring for years without affecting the general health and resistance to therapy. Continuous sialorrhea occurred in a man, aged 50, two weeks after he received an injection of penicillin.[11] It was accompanied by urticaria, which vanished after six weeks during therapy with antihistaminic drugs, but the ptyalism continued. Ptyalism is a rare complication of pregnancy and is thought to be related to hyperemesis, which it may accompany.[12] Amounts as great as 2,000 cc. of saliva expelled in a day are recorded. Excessive salivation occurs in rabies, epilepsy, and other neurological disorders, particularly with lenticular involvement.[13] Normally, the amount of salivary flow varie greatly in different persons. In one study,[14] the amount secreted in a minute ranged from 0.08 cc. to 0.88 cc. Calculated at those rates the range for normal persons for a 24-hour period is from 115 cc. to 1,267 cc., an average of 345 cc.

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[2] Emden, F.: Sitzungsbericht Aerzt. Ver., Hamburg, 1897. Osler, W., and McCrae, T.: Modern Medicine, Philadelphia, Lea & Febiger, 1908, vol. 5, p. 71.

[3] Kussmaul, I.: Anfallsweise auftretende Speichelgeschwulst in Folge von chronischer eitrig fibrinöse Entzündung des stensonsche Ganges, Berl. klin. Wchnschr. **60**: 209-211, 1879.

[4] Bass, H. E., and Mendlowitz, M.: Relationship of Chronic Recurrent Sialadenitis to the Alarm Reaction, Ann. Otol., Rhin. & Laryng. **58**: 868-872, 1949.

[5] (*a*) Pearson, R. S. B.: Recurrent Swelling of the Parotid Glands, Arch. Dis. Childhood **10**: 363-376 (Oct.) 1935. (*b*) Payne, R. T.: Recurrent Pyogenic Parotitis: Its Pathology, Diagnosis, and Treatment, Lancet **1**: 348-353, 1933. (*c*) Bigler, J. A.: Symposium on Pediatrics: Recurrent Parotitis, M. Clin. North America **36**: 97-104, 1946.

[6] Vogeler, K.: Über die chronische Entzündung der Parotis, Arch. f. klin. Chir. **122**: 655-663, 1923.

[7] Von Reuss, A.: Ueber chronische Erkrankungen der Parotis in Kindesalter, Jahrb. f. Kinderh. **70**: 161-173, 1909.

[8] Meyer, H. S.: Chronic Sialodochitis: Recurrent, Alternating, Familial, Hereditary, J. Pediat. **4**: 248-250, 1934.

[9] Pearson, R. S. B.[5a] Waldbott, G. L., and Shea, J. J.: Allergic Parotitis, J. Allergy **18**: 51-54, 1947.

[10] Weber, F. P.: Endocrine Tumours, and Other Essays, London, H. K. Lewis & Co., Ltd., 1936, p. 132.

[11] Ptyalism, Queries and Minor Notes, J. A. M. A. **143**: 1297 (Aug. 5) 1950.

[12] Ptyalism During Pregnancy, Queries and Minor Notes, J. A. M. A. **146**: 1545 (Aug. 18) 1951.

[13] Furstenberg, A. C., and Crosby, E.: Disturbance of the Function of the Salivary Glands, Ann. Otol., Rhin. & Laryng. **54**: 243-264, 1945.

Whether the case of periodic sialorrhea we have described is of the same nature as the other reported cases of recurrent parotitis cannot be said. It probably is. Differences in glands affected and in the signs, symptoms, and behavior are understandable considering the complexity of the neurological control of the salivary glands as discussed in Furstenberg's paper.[13] In our patient and in others, additional convenient signs and symptoms suggest the presence of a systemic disturbance, with salivary disorder as the outstanding feature. Recurrent parotitis and periodic sialorrhea are similar to other periodic disorders in that they occur fairly regularly at short intervals, last many years without affecting the patient's general health, and resist therapy. The frequent occurrence of overlapping characteristics among other periodic disorders, suggesting a unity, is evident also in recurrent salivary disorders, in which abdominalgia, fever, eosinophilia, arthralgia, uterine bleeding, and evidence of hereditary transmission may be noted. Sialorrhea was observed in patients during episodes of periodic abdominalgia [15] and periodic neutropenia.[16] The cause of such disorders is unknown, but there is reason to suspect a common, underlying rhythm, which, in rare persons, provokes different symptoms and signs to recur in synchrony and which in the present case affects the salivary glands. Atropine and methantheline, at times, temporarily reduced the excessive flow of saliva, but tests with these drugs revealed no further hint as to the cause than those used in studies of other periodic disorders. The persistent eosinophilia of this patient may be inherent, but it was not present in her son. There was no evidence of allergy, polyarteritis, or other disease. The significance of the patient's abnormal electroencephalogram is undetermined.

SUMMARY

The occurrence of episodes of swelling of the submaxillary salivary glands with excessive salivation lasting a day or more and occurring at intervals of about 6 days for over 3 years in a 44-year-old woman is described. Studies failed to reveal the cause, and therapy, except for transient inhibition of the flow, was unsuccessful. Numerous cases of recurrent swelling of the parotid glands are on record. Because of their characteristic rhythmicity of recurrence over a long period and their lack of effect on the patient's general health, periodic sialorrhea and recurrent swelling of the parotid glands may belong to the group of diverse periodic disorders previously described.

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[14] Spealman, C. R.: The Volume Flow of Resting Salivary Secretion, Am. J. Physiol. 139:225-229, 1943.

[15] Janeway, E. G., and Mosenthal, H. O.: An Unusual Paroxysmal Syndrome, Probably Allied to Recurrent Vomiting, with a Study of N Metabolism, Tr. A. Am. Physicians 23:504-518, 1908.

[16] Rolland, C. F., and Davidson, L. S. P.: A Case of Cyclic Neutropenia, Edinburgh M. J. 57:205-210, 1950.

CARCINOMATOUS MUCOSAL EXCRESCENCE OF THE RECTUM

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There has been much discussion as to the pathogenesis of intestinal polyps and the possible evolution of benign adenomas into invasive carcinomas. No direct valid evidence of such a transformation has been offered, because of the necessity for complete removal of a lesion for microscopic examination to permit determination of its true histopathological character, thus rendering further study in situ impossible. However, considerable collateral evidence for malignant transformation is presented both on histological grounds, by Atwater and Bargen,[1] and on a clinical basis, by Helwig.[2] It is generally agreed that colonic polyps, if not already malignant, are potentially malignant and that, whenever observed, they should be extirpated, whether or not they are at the time producing symptoms. With regard to the smaller polyps, sometimes referred to as mucosal excrescences, there are, however, statements in the literature that tend to create a more complacent attitude. In many instances, these smaller lesions are either ignored or are fulgurated without being submitted for histological study, because their gross characteristics are considered unmistakably benign. Castro and co-workers [3] state that "on numerous occasions we have seen sessile mammilations 2 to 3 millimeters in size during routine proctoscopy. These are often gone when proctoscopy is repeated a few weeks later." In the light of such a statement, the following case is considered of interest.

REPORT OF A CASE

S. S., a 51-year-old white physician, while serving as a medical officer in the United States Army in December, 1943, began to experience fatigue and had an occasional loose bowel movement accompanied by slight discomfort in the lower part of the abdomen. He was admitted to a military hospital, where all his examinations yielded negative results except for the proctosigmoidoscopic findings. A "tiny polyp" was observed on the posterior wall of the rectum about 5 in. (12.5 cm.) from the anal orifice by a well-known and competent gastroenterologist. The patient was advised that there was no necessity for removal of this lesion, since it was so small, but that it should be observed periodically. In 1946, he had a recurrence of loose bowel movements and slight cramps for a short time but did not have further examinations until Feb. 17, 1950, when he consulted one of us (M. W.). The results of general physical examination, blood cell count, and urinalysis were normal. A warm, purged stool was negative for ova and parasites. Proctosigmoidoscopic examination was performed. The instrument was introduced for a distance of 10 in. (25 cm.). Five and one-half inches (about 14 cm.) from the anal verge, a small sessile polyp, about 2½ mm. in diameter, was seen. Its apex was raised about 1 mm. from the surface of the mucosa. The color was that of the surrounding mucosa, which appeared normal. When pressure was made with the rim of the sigmoidoscope against the surrounding rectal mucosa with the lesion held at the center, the polyp flattened out and became invisible. When the pressure of

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From Beth Israel Hospital.

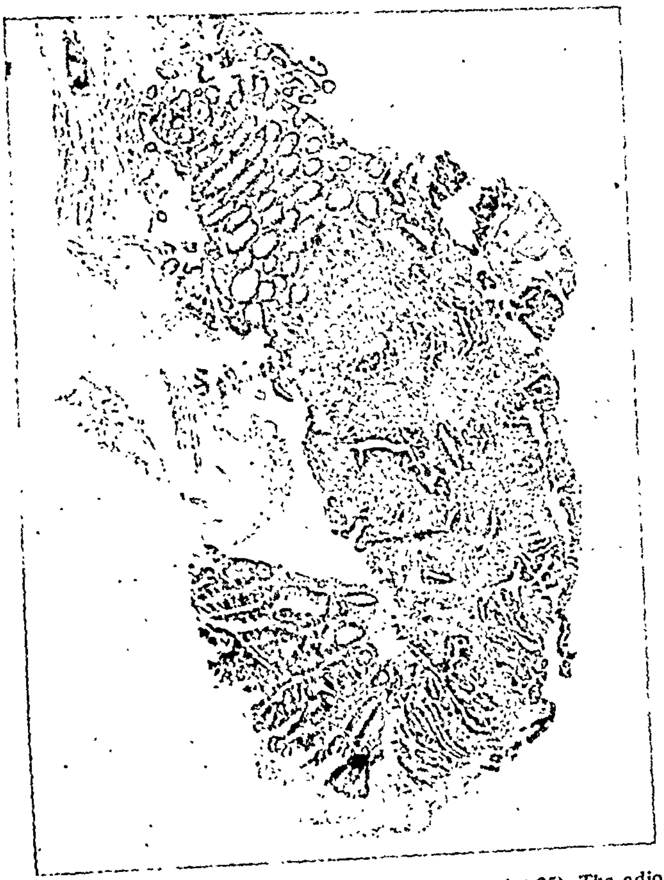
[1] Atwater, J. S., and Bargen, J. A.: The Pathogenesis of Intestinal Polyps, Gastroenterology 4:395 (May) 1945.

[2] Helwig, E. B.: The Evolution of Adenomas of the Large Intestine and Their Relation to Carcinoma, Surg., Gynec. & Obst. 84:36 (Jan.) 1947.

[3] Castro, A. F.; Ault, G. W., and Smith, R. S.: Adenomatous Polyps of the Colon and Rectum, Surg., Gynec. & Obst. 92:164 (Feb.) 1951.

the instrument was released, it was apparent that a small polyp was present. The lesion was removed completely with a cold angulated, punch biopsy forceps and submitted for histological examination to Dr. William Antopol who reported that the specimen consisted of a fragment of soft gray tissue measuring 4 by 3 by 2 mm. Microscopic examination revealed normal glands at each end of the mucosal surface. The middle third of the mucosa was composed of glands that contained no mucous secretory glands. The acini had cells with scant cytoplasm and prominent nuclei. The nuclei were present at all levels, often reaching the free surface of the cell. Mitoses were frequent. These atypical glandular elements penetrated beyond the muscularis mucosae. In the region of the atypical glands there was erosion with numerous polymorphonuclear leukocytes. Because of the abnormal glandular pattern, the pronounced proliferation and the invasive tendency, the lesion was diagnosed as an adenocarcinoma.

The area from which the polyp was removed was fulgurated several days later by one of us (R. T.). Results of the barium enema examination of the colon, including air contrast study, were normal. The sigmoidoscopic examination has been repeated several times since, without evidence of recurrence. Results of a barium enema study done in May, 1951, were also normal.



Carcinomatous mucosal excrescence of the rectum (× 25). The adjoining mucosa is normal.

COMMENT

Mucosal excrescences seen on proctosigmoidoscopic examination are sometimes considered as being of no consequence or else are fulgurated thoroughly without biopsy, either to save the patient the cost of an additional laboratory procedure or because the observer feels confident that the gross appearance indicates that the lesion is benign. The foregoing experience with an excrescence of the rectum of less than 3 mm. in diameter, which proved to be an adenocarcinoma, emphasizes the necessity of submitting any accessible abnormal protrusions of the rectal or sigmoidal mucosa for histological indentification. One of us (R. T.), while experimenting with the construction of a cold, angulated biopsy forceps in 1940, removed a grossly benign-appearing mucosal elevation that was less than 2 mm. in diameter and which proved to be an adenocarcinoma on microscopic examination.[4] Hines and Riddler [5] recently reported the removal with a biopsy forceps of a sessile, submucosal nodule measuring 3 mm. in diameter from the anterior rectal wall, 4 cm. above the dentate line. Histological examination disclosed the nodule to be a carcinoid tumor. It is apparent that one cannot assume on the basis of the gross appearance or the small size of a mucosal excrescence that it is innocuous.

An interesting aspect of the case herein reported is that the description and location of the lesion as stated in December, 1943, corresponded with that observed in February, 1950. Although one cannot be certain, it would appear that we were dealing with the same "tiny polyp." It seems highly improbable that the lesion when first observed was carcinomatous in nature, since one would then have to assume that it remained dormant for six years and did not increase in size. In all likelihood, this mucosal excrescence was a benign adenoma, when it was seen in December, 1943, and underwent malignant changes shortly before it was removed. One may therefore state that in all probability this is an instance in which a benign adenoma of the rectum was observed and left in situ and became carcinomatous approximately six years later. Added weight is thus given to the generally held opinion that all abnormal protrusions of the rectosigmoidal mucosa should be completely removed when observed and submitted for histological examination.

SUMMARY

A case is reported in which a 2.5 mm. mucosal excrescence of the rectum, which had been observed six years earlier, proved to be carcinomatous in nature. The importance of removing and examining histologically every abnormal protrusion from mucosa of the instrumentally accessible terminal portion of the colon, no matter how small or innocent in appearance, is emphasized.

24 E. 36 St. (16) (Dr. Weingarten).

[4] Turell, R., and Wilkinson, R. S.: Adenomas of the Colon and Rectum, Surgery **28**: 651 (Oct.) 1950.

[5] Hines, M. O., and Riddler, J. G.: Unusual Lesions of Rectum and Anus, Surgery **29**: 496 (April) 1951.

Wet X-Ray Films.—It is the practice for some people to make a habit of asking to see wet [x-ray] films, before the processing is completed by drying. This seems to be an opportune moment to point out that this procedure lays one open to making many errors. When a film is taken it is dry. When it is processed, the emulsion, including the layers of sensitized material, of which there may be 2 or more, swells up to between 4 and 8 times its original thickness. This swollen emulsion is particularly susceptible to physical damage from contact with other materials. What is, perhaps, more important, is that when there is any slight irregularity of the coating—even though this may not be sufficient to affect its photographic qualities—during the swelling the images formed become distorted and indistinct. It is therefore frequently impossible to detect minor alterations of structure, particularly in the study of bone trabeculation, until the film has been dried and the emulsion has returned to the physical state in which it existed when the film was exposed. Neither knowledge nor experience can overcome this physical deficiency in the images on a wet film.—Anthony A. Vickers, F.F.R., A Dissertation on X-Ray Diagnosis, *The British Journal of Physical Medicine,* May, 1952.

TOPICAL CHLORAMPHENICOL (CHLOROMYCETIN®) THERAPY OF PYOGENIC DERMATOSES

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June Carol Shafer, M.D., Arlington, Va.

The continuing search for more effective agents for the treatment of pyogenic infections of the skin has led to the development of many improved preparations over the past few years.[1] In the evaluation of a new agent, two basic factors must be observed. The agent must possess an active bacteriostatic effect, preferably in low concentrations, against a wide spectrum of microorganisms that commonly affect the skin. It must also show a low index of cutaneous sensitivity. Some of the newer antibiotic ointments appear to meet both of these criteria.[1] Another factor worthy of consideration is the stability of the preparation in various vehicles. This report evaluates topical chloramphenicol with reference to effectiveness in the treatment of pyodermas, to cutaneous allergenicity, and to applicability in a greaseless base.

HISTORICAL DATA

In 1947, Ehrlich and co-workers[2] announced the recovery of a new antibiotic, chloramphenicol (chloromycetin®), from Streptomyces venezuelae after a tedious search of thousands of soil samples from all over the world. In vitro testing by McLean and co-workers[3] revealed a wide spectrum of susceptibility to the antibiotic by microorganisms commonly invading the skin, including Staphylococcus, Streptococcus, Aerobacter, Bacillus, Corynebacterium, Hemophilus, and other bacteria, certain spirochetes and Protozoa, fungi of the Actinomyces group, and some of the rickettsias and large viruses.

Recently, Cornbleet and Schorr[4] reported the results of topical chloramphenicol therapy in 18 cases of "eczema and pruritus of the ear canal," associated with bacterial infection in some cases. They used an ointment containing 0.275 gm. of chloramphenicol in 30 gm. of polyethylene glycol (carbowax 1500®). Initially, there was improvement in 16 of the 18 patients treated, but recurrences were frequent in those patients reached for follow-up inquiry. There was no instance of sensitivity to the preparation in the series. More favorable results were recorded by Newman and Feldman[5] in the treatment of 51 patients with various pyogenic dermatoses. They used a 1% chloramphenicol cream (0.300 gm. of chloramphenicol in 30 gm. of oil-in-water emulsion).[6] In patients with superficial pyodermas, there was rapid recovery, but less dramatic improvement was noted in patients with deeper infections. An additional 75 patients were treated later with the cream, and one instance of hypersensitivity was encountered.

ALLERGENICITY

For this report, patch tests of 2% chloramphenicol cream (0.600 gm. of chloramphenicol in 30 gm. of base)[6] were applied to 100 consecutive patients admitted to the dermatology section, Walter Reed Army Hospital. A total of 50 persons were retested 14 days later. There were no positive reactions from either the original or provocative tests. Of the treated patients, one showed a flare-up of the underlying dermatitis, but a patch test for chloramphenicol cream was negative.

Kerby and Muller[7] recently investigated a group of antibiotics, including chloramphenicol, with reference to the Shwartzman phenomenon. In their animal experiments, no hypersensitivity of the Shwartzman type was demonstrated. No cutaneous sensitivity was noted by Newman and Feldman[5] in 100 consecutive patients dressed with chloramphenicol cream after surgical procedures. As previously mentioned, one of their patients had a contact type of reaction. Patch tests were positive to chloramphenicol and negative to the cream base.

Results of Treatment with Chloramphenicol Cream of Eighty-Six Patients with Pyogenic Disease

Disease	Number of Patients	Clinically Cured	Improved	No Improvement
Impetigo	8	6	2	..
Folliculitis of trunk or extremities	9	6	3	..
Folliculitis of beard or scalp	16	13	3	..
Sycosis vulgaris	6	2	2	2
Otitis externa *	20	2	18	..
Infected dermatitis of intertriginous areas *	4	1	2	1
Infected dermatitis of hands and feet *	11	..	8	3
Ecthyma	3	1	1	1
Hidradenitis suppurativa	2	..	2	..
Pustular acne	2	..	1	1
Furunculosis	2	..	1	1
Fox-Fordyce disease	2	..	..	2
Aphthous stomatitis of lips	1	1	..	..

* Secondary infectious element cleared rapidly almost without exception.

CLINICAL MATERIAL AND RESULTS

A total of 100 patients with superficial and deep pyogenic infections from the outpatient clinic and inpatient service of the dermatology section of this hospital were selected for the study. Of this group, adequate follow-up examinations of 86 persons were available. The majority

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The chloramphenicol (chloromycetin®) cream used in this study was supplied by Parke, Davis & Company.

From the Dermatology and Syphilology Section, Walter Reed Army Hospital, Lieut. Col. William N. Piper, Medical Corps, Chief of Section (Captain Trice), and the Dermatology and Syphilology Department, Georgetown University Medical School (Dr. Shafer).

1. (*a*) Miller, J. L.; Slatkin, M. H., and Lingamfelter, C. S.: Symposium on Diseases of the Skin: Antibiotics in Dermatology, M. Clin. North America **35**: 341-354 (March) 1951. (*b*) Miller, J. L.; Slatkin, M. H., and Johnson, B. A.: Local Use of Bacitracin, J. Invest. Dermat. **10**: 179-188 (March) 1948. (*c*) Derzavis, J. L.; Rice, J. S., and Leland, L. S.: Topical Bacitracin Therapy of Pyogenic Dermatoses: Clinical Report, J. A. M. A. **141**: 191-192 (Sept. 17) 1949.

2. Ehrlich, J., and others: Chloromycetin, a New Antibiotic from a Soil Actinomycete, Science **106**: 417 (Oct. 31) 1947.

3. McLean, I. W., Jr.; Schwab, J. L.; Hillegas, A. B., and Schlingman, A. S.: Symposium on Antibiotics; Susceptibility of Micro-Organisms to Chloramphenicol (Chloromycetin), J. Clin. Investigation **28**: 953-963 (Sept.) 1949.

4. Cornbleet, T., and Schorr, H. C.: Chloromycetin for Eczema and Pruritus of the Ear Canal, A. M. A. Arch. Dermat. & Syph. **62**: 907-908 (Dec.) 1950.

5. Newman, B. A., and Feldman, F. F.: Treatment of Pyogenic Dermatoses with Topical Chloramphenicol (Chloromycetin), A. M. A. Arch. Dermat. & Syph. **64**: 212-214 (Aug.) 1951.

6. The commercial base contained cetyl alcohol, petrolatum, sodium lauryl sulfate, propyl parahydroxybenzoate and distilled water.

7. Kerby, G. P., and Muller, J. C.: An Experimental Study of Antibiotics for their Activity in the Shwartzman Phenomenon, J. Invest. Dermat. **15**: 385-388 (Nov.) 1950.

of the patients were given a 2% chloramphenicol cream to be applied three times daily for from 3 to 14 days, depending on the severity of involvement. A few patients used a 1% cream, which, when applied more frequently, appeared to be as effective as the 2% preparation. Some patients in the series received nonspecific supportive measures, such as aluminum subacetate compresses and disinfection of shaving instruments.

Results of therapy with chloramphenicol cream are briefly summarized in the accompanying table. In general, there was clearing of most superficial infections within 3 to 10 days. In the group of patients with otitis externa, the underlying dermatitis remained in most instances, so that the final result was recorded as "improved." Chloramphenicol was effective, however, in clearing and preventing secondary bacterial infection. Deeper infections showed favorable results when treated early in the acute phase. In other chronic cases, chloramphenicol was no more effective than other antiseptic preparations.

COMMENT

In this series, chloramphenicol cream was found to be as effective in the therapy of pyodermas as other topical antibiotic preparations previously evaluated at this hospital, such as bacitracin[1c] and aureomycin.[8] These results are not so impressive as those reported by Newman and Feldman.[5] Perhaps this can be explained by the fact that the pyodermatous infections in patients referred to an Army general hospital have frequently reached a chronic stage and have been relatively resistant to ordinary treatment.

The oil-in-water emulsion base was particularly efficient as a vehicle for the drug for treating hairy and intertriginous areas. It was especially useful when applied before and after shaving by patients with recurrent folliculitis of the bearded area. Patients who had used ointment preparations previously found the cream base mechanically more suitable when used as a part of the shaving routine. Incorporation of 2% chloramphenicol in basic lotion[9] for patients with secondarily infected intertrigo (not included in this series) was also found to be efficient.

SUMMARY AND CONCLUSIONS

A wide spectrum of microorganisms that commonly cause pyogenic infections of the skin are susceptible to chloramphenicol. A total of 86 patients with superficial and deep pyodermas were treated with chloramphenicol cream. Favorable responses were noted in treating superficial infections. The response of deep pyodermas was no better than to some of the other topical antibiotic preparations. The oil-in-water emulsion base afforded an efficient vehicle for the drug in treating hairy and intertriginous areas. No proved instance of hypersensitivity developed in the patients treated. Patch tests in 100 persons and provocative tests in 50 of them failed to demonstrate an allergic response.

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[8] Fink, J. C., and Grauer, F. H.: Unpublished data.

[9] Though chloramphenicol is compatible with most ointments and lotions, its stability is destroyed by glycerin or wool fat (lanolin).

J.A.M.A., Aug. 16, 1952

MECHANICAL AID FOR THE ARM AMPUTEE

Jerome Weiss, M.D.
and
Otto Eisert, M.D., Brooklyn

In recent years considerable advance has been made in the rehabilitation of the arm amputee by means of working prostheses. These are supplied with a prehensile hook operated by the opposite shoulder or by muscle motors produced surgically. For special purposes, modifications of the hook have been devised. Some prostheses provide for quick removal of the hook and replacement with a nonfunctioning hand for cosmetic effect.

These prosthetic devices have done much to restore the usefulness and to elevate the morale of the arm amputee; however, there remain two important functions that are still difficult for him. These are the opening of an ordinary can, and the removing or replacing of a bulky top from a jar. Dunton and Licht[1] recognize the difficulty that the usefulness of only one arm imposes. No present arm prosthesis that is useful for ordinary purposes will satisfactorily hold a can or jar unless modified in some way. The slipping of a can or jar can be dangerous, particularly for the one-armed amputee or hemiplegic who is using his good hand to do the work.

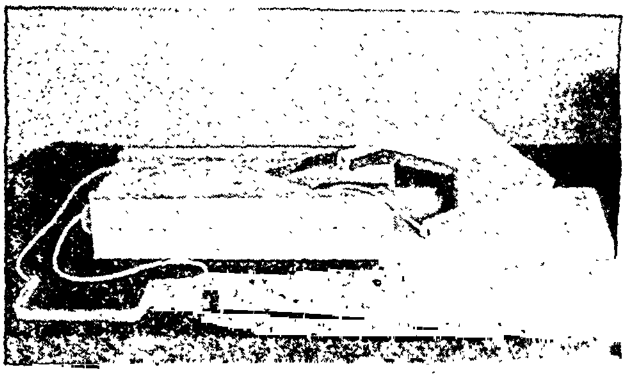

Fig. 1.—The holder with the movable jaw completely removed from its track. The stirrup appears at the left.

To make this chore safe and easy for the one-armed amputee or hemiplegic, a simple device has been developed by the physical medicine rehabilitation service of the Brooklyn Veterans Administration Hospital. It is an adaptation of the saddler's bench, in which foot power is used to clamp the leather and hold it while it is being stitched. Figures 1 and 2 are fully descriptive of the construction and use of the device.

Although many modifications in construction are possible, the device, as shown, is held to the simple form that can easily be produced in from three to four hours in the manual arts or occupational therapy departments of any good service of physical medicine and rehabilita-

From the Veterans Administration Hospital, Brooklyn.

Mr. William Conlon, manual arts therapist, constructed the mechanical aid from plans furnished by the authors.

Mr. F. Hertling, of the Medical Illustrations Section, Veterans Administration Hospital, Brooklyn, prepared the illustrations.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

[1] Dunton, W. R., and Licht, S., editors: Occupational Therapy: Principles and Practice, Springfield, Ill., Charles C Thomas, Publisher, 1950.

tion. The clamps and the extra length of the lower board are not required if the holder is screwed to the table top. The holding screws, if used, must be of the flat-headed type and must be countersunk below the top of the lower board. The addition of removable jaws to the fixed and moving members will make the device still more versatile. It will then approximate the saddler's bench and can be used to hold parts for soldering or for joining by other means.

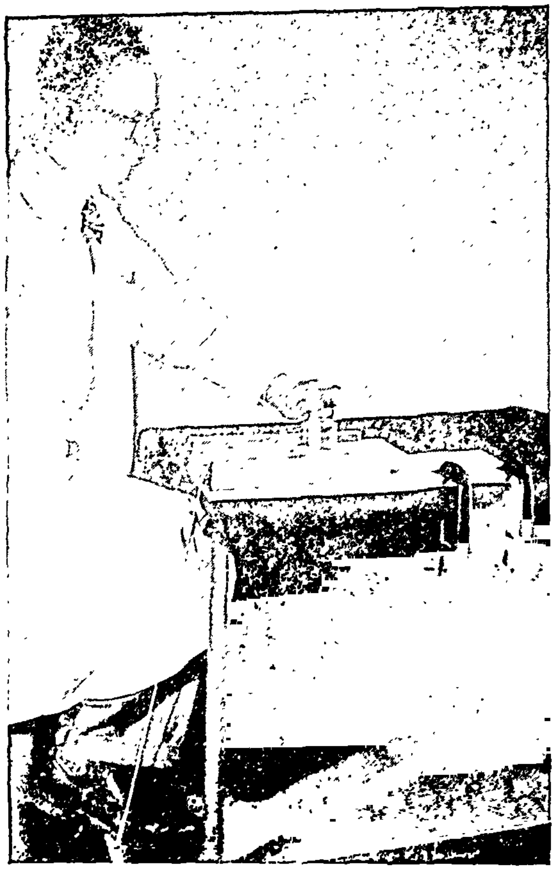

Fig. 2.—Here is demonstrated the use of the apparatus to facilitate the opening or closing of a jar by a one-armed person. The clamps are not needed if the holder is screwed to the table top.

The required materials, easily obtained, are given below:

Wooden boards—¾ in. plywood is excellent
Two small single awning pulleys—nonswivel type
About 5 ft. of strong awning cord
One 9 in. length of ⅛ or ¼ in. iron pipe
Two flat-headed iron wood screws—no. 8, 1 in. long
Three flat-headed iron wood screws—no. 10, 1½ in. long
Strips of rubber from an old innertube for the jaw faces
Two clamps, if the hold-down screws are not to be used

A 6 in. square sheet of rubber cut from an old innertube will improve the patient's grip on the jar cap or bottle top.

The amputee or hemiplegic will be able to use the holder perfectly after brief instruction, and, with a little practice, he will usually do better than an able-bodied person can without the holder. A simple device such as this can do much to help the arm amputee or the hemiplegic to attain independence.

MENINGITIS DUE TO KLEBSIELLA PNEUMONIAE

REPORT OF TWO CASES, WITH THE USE OF STREPTOKINASE AND STREPTODORNASE IN ONE CASE

Peter A. Trice, M.D.
and
Thomas E. Townsend, M.D., Fairfield, Ala.

In a recent article by Thompson and associates,[1] the rarity of meningitis due to Klebsiella pneumoniae was emphasized. These authors, in a comprehensive review of the literature, found only 118 cases, to which they added 1 case of their own. We wish to add two cases of meningitis due to K. pneumoniae diagnosed at the Lloyd Noland Hospital and to discuss the use of streptokinase and streptodornase in the management of one case.

REPORT OF CASES

Case 1.—I. F. B., a 2-year-old white girl, was admitted to the Lloyd Noland Hospital on Nov. 7, 1946, in a comatose state. The history revealed that the child had been well until the previous day, when fever with occasional vomiting developed. The child was examined by a local physician on the morning of admission. A diagnosis of "sore throat" was made and a sulfonamide drug prescribed. After taking approximately 0.5 gm. of the prescribed drug, she seemed much worse and was brought to the Lloyd Noland Emergency Clinic, where she was immediately admitted to the hospital. On admission, at 6:30 p. m., the child was comatose and cyanotic. The pulse was palpable in the legs and temples; the heart sounds were of poor quality and rapid. The right pupil was irregular and fixed to light. The left pupil was pinpoint sized. It was apparent that she was dying. She did not respond to supportive therapy and died an hour and 20 minutes after admission.

Autopsy revealed purulent meningitis and pneumonia. Sterile blood cultures and spinal fluid cultures done at autopsy revealed K. pneumoniae.

Case 2.—N. E. M., a Negro infant, was born prematurely at the Lloyd Noland Hospital Sept. 5, 1951. The delivery was sterile with spontaneous left occiput anterior presentation. The cry was weak and delayed; the lungs were not fully expanded; and slight cyanosis was present around the mouth. The birth weight was 4 lb. 2 oz. (1,871 gm.), and the length was 17½ in. (44 cm.). The infant was placed in an incubator with oxygen, and there was rapid improvement. He was removed to a bassinet on the second day. The weight gain was slow for the first five days, but after the caloric intake was increased the gain was satisfactory.

On Sept. 24, at 19 days of age, the infant became cyanotic and experienced some respiratory difficulty. The lungs were clear to auscultation; the heart rate was rapid, but the sounds were of good quality. He responded well to treatment with oxygen and a stimulant. The possibility of an intracranial hemorrhage or aspiration of formula was considered at that time. Immediately after this episode some spasticity of the extremities was noted. This was believed to be a result of anoxia and disappeared in approximately 24 hours. After this, the infant's condition remained good. He took his formula well, and the weight gain was satisfactory.

On Oct. 1, when the patient was 26 days of age, it was noted that the anterior fontanel was full but not tense. A lumbar puncture revealed xanthochromic fluid with 1,750 cells per cubic

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From the Pediatric Department, Lloyd Noland Hospital.

[1] Thompson, A. J.; Williams, E. B., Jr.; Williams, E. D., and Anderson, J. M.: Klebsiella Pneumoniae Meningitis: Review of the Literature and Report of a Case with Bacteremia and Pneumonia, with Recovery, Arch. Int. Med. 89: 405-420 (March) 1952.

millimeter, 87% of which were neutrophils and 13% lymphocytes. The smear was negative. Treatment with dihydrostreptomycin, 0.25 gm. every eight hours, and penicillin, 100,000 units every three hours, was started. The following day spinal fluid culture revealed an organism resembling K. pneumoniae. At this time chloramphenicol therapy, 250 mg. every four hours, was started. On Oct. 3 the organism was definitely identified as K. pneumoniae. Tests revealed the organism was sensitive to chloramphenicol, streptomycin, and aureomycin, in the order named. On Oct. 4 the fontanel was tense and the general condition poor. The temperature at this time was 99.8 F per rectum, and at no time during the course of the illness was it any higher. The infant continued to take his formula well but required supplements of fluids, given parenterally. On Oct. 6 he had a generalized, clonic convulsion, which was controlled by sedation. On Oct. 10 a lumbar puncture was done, but no spinal fluid was obtained. A ventricular tap was therefore performed. The ventricular fluid revealed 1,250 cells per cubic millimeter, 90% neutrophils and 10% lymphocytes. Smear and culture were negative. The protein was 800 mg., sugar 16.9 mg., and chlorides 603.1 mg. per 100 cc. At this time a gradual increase in the circumference of the head was noted, with a tense and bulging fontanel. On Oct. 18, 20 cc. of xanthochromic fluid was obtained by ventricular tap, and 125,000 units of streptokinase and 25,000 units of streptodornase ("varidase")[2] diluted in 10 cc. of distilled water were instilled. The ventricular fluid contained 4,300 cells with 70% neutrophils, and 30% lymphocytes. Protein was 640 mg., chlorides 586 mg., and sugar 30 mg. per 100 cc. Smear and culture were negative. After this there was definite clinical improvement. The weight gain was steady; feedings were taken well; no fluids were required parenterally; there was no further increase in circumference of the head; and the fontanel returned to normal. Penicillin therapy was discontinued on Oct. 20 and streptomycin and chloramphenicol therapy on Oct. 24. On Nov. 7 a lumbar puncture for therapeutic evaluation was done. No fluid was obtained. Ventricular tap revealed 325 cells, 60% neutrophils and 40% lymphocytes. The protein was 164 mg., chlorides 659 mg., and sugar 32.5 mg. per 100 cc. Smear and culture were negative. Treatment was discontinued, and the infant was discharged to be followed in the outpatient clinic. At this time the infant weighed 5 lb. 15 oz. (2,693 gm.). The diagnoses on discharge were prematurity and meningitis due to K. pneumoniae, with acquired hydrocephalus. The follow-up on this patient was inadequate, owing to lack of cooperation by the family.

The patient was not seen again until readmission, Jan. 1, 1952, some 54 days after discharge. At this time he was 4 months of age. The mother stated that the child's head had suddenly become larger the night before admission but that he had been well until that time. However, physical examination revealed severe malnutrition, the weight being 7 lb. (3,175 gm.), and severe hydrocephalus. The respirations were 20 to 30 per minute and very shallow; the pulse was weak and the rate 60 per minute; and the temperature was 94.8 F per rectum. The infant appeared both acutely and chronically ill. Lumbar puncture was done on admission, but no fluid was obtained. Ventricular tap revealed 10 cells per cubic millimeter, all of which were lymphocytes, and protein was 435 mg. per 100 cc. Smear and culture were negative. Treatment with chloramphenicol, 250 mg. every four hours, and dihydrostreptomycin, 0.25 gm. every eight hours, was started. Supportive measures consisted of the use of plasma, fluids parenterally, oxygen, and heat. Ventricular taps were done on Jan. 3, 4, and 6, with instillation of 50,000 units of streptokinase and 10,000 units of streptodornase each time. After this, there was some improvement in the general condition and the fontanel became less tense. However, the circumference of the head steadily increased.

Ventricular taps were repeated on Jan. 16, 21, 22, and 23, with instillation each time of 87,500 units of streptokinase and 17,500 units of streptodornase. On Jan. 24, ventricular tap was performed, with instillation of 125,000 units of streptokinase and 25,000 units of streptodornase. All ventricular taps revealed normal cell counts, negative smear and culture, and an elevated protein level. After the last ventricular tap, it was felt that maximum benefit had been obtained from the streptokinase and streptodornase therapy and that further treatment with these agents was not indicated. The infant was removed from the hospital by the parents on Jan. 26, on the 26th day of his second hospital admission and 116 days from the time his illness was first diagnosed. The weight on discharge was 9 lb. (4,082.33 gm.), a gain of 2 lb. (907.18 gm.) during the last hospitalization. Follow-up was to be done through the outpatient clinic, with treatment to be reinstituted as indicated. Once again, cooperation on the part of the family was poor. The patient was not seen again and died at home on Feb. 12.

COMMENT

In case 1, the infant was moribund on admission and died an hour and 20 minutes after admission to the hospital. There was time for no treatment except supportive measures, to which the infant failed to respond. The diagnosis was made at autopsy.

In case 2, owing to our failure to obtain spinal fluid on repeated lumbar puncture, we felt that the flow of fluid into the spinal canal was probably obstructed by fibrinous exudate. Because of the favorable reports of the fibrinolytic effect of streptokinase and the hydrolytic effect of streptodornase,[3] it was hoped that these drugs would liquefy the fibrinous exudate. Cathie and MacFarlane[4] reported the use of streptokinase intrathecally in cases of tuberculous meningitis, with favorable results when used before organized exudate was formed. As noted in the case report, definite improvement followed the initial instillation of 125,000 units of streptokinase and 25,000 units of streptodornase. Prior to the use of these fibrinolytic agents, the anterior fontanel was consistently distended and there was gradual increase in the circumference of the head. After the first instillation of streptokinase and streptodornase, the fontanel remained soft and the circumference of the head remained the same. However, lumbar puncture was still not productive of spinal fluid. On the initial hospital admission only one ventricular tap was performed after the instillation of the agents (on the 20th day), and this was for follow-up study rather than for therapeutic purposes.

On the patient's readmission to the hospital, 54 days after discharge, there was pronounced distention of the anterior fontanel and hydrocephalus. It is regrettable that the patient had not returned to the clinic during this interval. Because of the apparent beneficial results obtained with the use of streptokinase and streptodornase on the first admission, it was felt that further treatmen with these agents was warranted. Benefit was minimal even with daily instillation. The patient was remove from the hospital against our advice, so that furthe observation was not possible. He died at home 16 day after leaving the hospital.

Since the infecting agent apparently was controlled b streptomycin and chloramphenicol therapy, as indicate by sterile cerebrospinal fluid on repeated cultures, it felt that death was due to the complications and not primarily to the infecting organism.

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[2] The streptodornase used in this case was supplied by Lederle Laboratories Division, American Cyanamid Company, Pearl River, N. Y.

[3] Tillett, W. S., and Garner, R. L.: The Fibrinolytic Activity of Human Streptococci, J. Exper. Med. 58: 485-502 (Oct.) 1933. Tillett, W. S., and Sherry, S.: The Effect in Patients of Streptococcal Fibrinolysin (Streptokinase) and Streptococcal Desoxyribonuclease on Fibrinous, Purulent, and Sanguinous Pleural Exudations, J. Clin. Invest. 28: 173-190 (Jan.) 1949. Miller, J. M.; Ginsberg, M.; Lipin, R. J., and Long, P. H.: Clinical Experience with Streptokinase and Streptodornase, J. A. M. A. 145: 620-624 (March 3) 1951. Miller, J. M.; Long, P. H., and Stafford, E. S.: Clinical Experience with Streptokinase and Streptodornase in Tuberculosis, ibid. 148: 1485-1489 (April 26) 1952.

[4] Cathie, I. A. B.: Streptomycin-Streptokinase: Treatment of Tuberculous Meningitis: Preliminary Communication, Lancet 1: 441 (March 12) 1949. Cathie, I. A. B., and MacFarlane, J. C. W.: Adjuvants to Streptomycin in Treating Tuberculous Meningitis in Children, ibid. 2: 784 (Dec. 16) 1950.

SUMMARY

Two cases of meningitis due to Klebsiella pneumoniae are reported. Sensitivity studies in case 2 indicated the organism was most sensitive to chloramphenicol. The meningitis was controlled by the use of chloramphenicol and streptomycin. The use of streptokinase and streptodornase in one case is discussed. It is felt that some benefit was obtained from the use of streptokinase and streptodornase. Further investigation of the use of these agents in cases of meningitis in which blocks to the circulating cerebrospinal fluid are developed would seem justified. No untoward results from the administration of the streptokinase and streptodornase were detected.

P. O. Box 358.

COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION

APPARATUS ACCEPTED

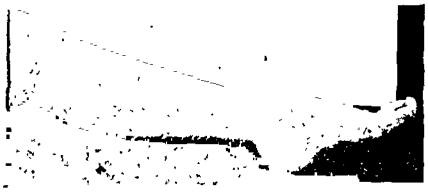
The following additional products have been accepted as conforming to the rules of the Council on Physical Medicine and Rehabilitation of the American Medical Association for inclusion in Apparatus Accepted. A copy of the rules on which the Council bases its action will be sent on application.

Ralph E. De Forest, M.D., *Secretary.*

The Jobst Stocking

Conrad Jobst, P. O. Box 653, Toledo, O.

The Jobst Stocking is intended for use in the treatment of circulatory disorders of the legs and differs from other available elastic stockings in the special arrangement for fitting the stocking to the individual patient's leg. This consists of a tape that automatically affords measurements of circumference at predetermined levels on the leg.



The Jobst Stocking.

The unpacked stocking weighs 70 to 90 gm. (2½ to 3 oz.). Its shipping weight is 78 to 98 gm. (2¾ to 3½ oz.).

Evidence obtained from sources acceptable to the Council indicated that The Jobst Stocking was well made and served the purposes for which it was designed. It was emphasized that elastic stockings are not suitable for all types of circulatory disease, that the physician, not the patient, must decide whether the stocking is needed in a given case, and that the measurements must be made by someone with experience and skill.

Bauer & Black Nylon Elastic Stockings, Models N-1 and S-1

Bauer & Black, Division of The Kendall Company, 309 W. Jackson Blvd., Chicago 6.

The two types of elastic stocking referred to in this report are made of a combination of nylon and elastic in such a way as to give a two-way stretch. The heel and the top are nonelastic, and the stocking is maintained in position by garters or a girdle support. Both are of a beige color, light in weight, and of sufficient strength for use in slight swellings and milder forms of edema.

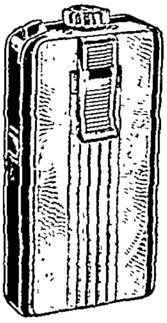
Evidence from sources acceptable to the Council showed that these stockings were well constructed. It is pointed out that elastic stockings can be effective in some forms of peripheral vascular disease, but injurious in others, and that the physician, not the patient, is the one to decide whether elastic stockings are advisable in any given case.

Sonotone Hearing Aid, Model 966

Sonotone Corporation, Elmsford, N. Y.

The Sonotone Hearing Aid, Model 966, is an electric hearing aid housed in a case of plastic and metal. The user may wear it with batteries or connect it to external batteries. With batteries it weighs 326 gm. and measures 2.6 by 6.8 by 12.5 cm.

Evidence from an acceptable source indicated that this instrument met the published requirements of the Council for hearing aids.



Sonotone Hearing Aid, Model 966

Monaghan Hospital Respirator, Model 110

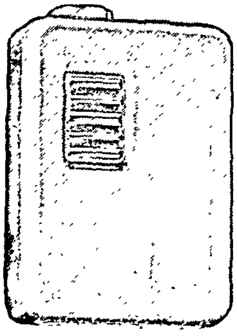
J. J. Monaghan Company, 500 Alcott St., Denver 4.

The Monaghan Hospital Respirator, Model 110, is a cuirass type respirator designed to supply the needs of two patients at once and also permit the administration of positive intratracheal pressure in time with the cycles of negative extrathoracic pressure.

The apparatus is housed in a large cabinet which stands on casters on the floor. Unpacked, it measures 94 (height) by 95 by 51 cm. (36½ by 37 by 20 in.) and weighs 66 kg. (145 lb.). The series of plastic shells (six shells of graduated sizes) are contained in one door of this cabinet; the positive pressure unit with other accessories are contained in the other. The cabinet also houses the pump and motor, which operates on 50 to 60 cycle alternating current at 110 volts and draws 130 watts.

Packed for shipment, the unit measures 107 by 101 by 62 cm. (41¾ by 39¼ by 24 in.) and weighs 94 kg. (206 lb.).

Evidence from sources acceptable to the Council showed that the device satisfied the published requirements of the Council for acceptance of respirators of the cuirass type as stated in The Journal, April 30, 1949, page 1273.



Telex Hearing Aid, Model 500

Telex Hearing Aid, Model 500

Telex, Inc., Telex Park, St. Paul 1.

The Telex Hearing Aid, Model 500, is a conventional electric hearing aid in shape and general design. It measures 61 by 44 by 21 mm. without clothing clips and weighs 82.5 gm. without batteries. The batteries weigh 25 gm., and the receiver and cord weigh 10 gm.

Evidence of good workmanship and satisfactory performance was obtained from a laboratory acceptable to the Council.

Dome-Paste Bandage

Dome Chemicals, Inc., 109 W. 64th St., New York 23.

The Dome-Paste Bandage consists of strips of impregnated gauze intended for application to the legs, as with Unna's Paste Boot, in the treatment of peripheral circulatory disease. Each strip, measuring 914 cm. (10 yards) in length and 10.2 cm. (4 in.) in width, is wrapped in wax paper and enclosed in a tin can. Twelve such bandages, separately packed, fill a multiple-unit container.

From acceptable sources the Council obtained evidence indicating that the Dome-Paste Bandage was convenient and efficient.

Dome-Paste Bandage

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Cable Address . . . . "Medic, Chicago"

CORTISONE, CORTICOTROPIN, AND TUBERCULOSIS

Considerable uncertainty has arisen concerning the use of cortisone and corticotropin in the presence of suspected or proved tuberculous lesions. Sufficient evidence appears to have been accumulated to at least suggest caution in the use of these drugs in a patient with tuberculosis.

Two recent reports now reveal without any doubt the need for this conservative approach.[1] The Committee on Therapy of the American Trudeau Society and an associated committee, the Laboratory Subcommittee, have offered evidence that should be kept in mind when cortisone and corticotropin are used clinically. The latter committee, after examining 39 published papers and personal communications, concluded that, insofar as the data from studies on experimental tuberculous infections in animals are concerned, the drugs may produce a variety of effects, depending on the species of animal and the experimental conditions. The hormones produce a decrease in reactivity of the small blood vessels, which in turn reduces the inflammatory response of sensitized tissue to the tubercle bacillus. Cortisone can cause a flare-up of a tuberculosis infection in mice. When the hormones are withdrawn, a tuberculosis lesion may become more active. Further, the hormones have no significant action on the tubercle bacillus itself in vitro.

The Committee on Therapy analyzed data on 81 patients treated with cortisone and/or corticotropin in whom tuberculosis was present or became a complicating factor. Autopsies had been performed on many of the subjects. From their study the members of the committee concluded that an exacerbation of tuberculosis infection does not always follow use of cortisone or corticotropin, but that these drugs do have an effect on active and apparently inactive tuberculosis. For example, there often were found in their studies a spectacular dissemination of the disease and unsatisfactory localization of the lesions. They also report development and progression of excavation during and after the hormone therapy. Yet these drugs may cause signs of clinical and symptomatic improvement even as the lesions are becoming progressively worse.

Therefore, the Committee on Therapy has concluded that cortisone and corticotropin appear to have no place in the treatment of active tuberculosis as such, and in the presence of active tuberculosis infection the use of these drugs should be regarded as potentially hazardous. While the presence of old, apparently inactive, tuberculosis is not an absolute contraindication to therapy, the condition should be studied carefully; if one of the drugs is used, there should be frequent examinations of roentgenograms and the sputum. Even in the absence of known tuberculous infection there should be careful examinations before, during, and after therapy, as there may be exacerbation of an unsuspected lesion. If a pulmonary infiltrate develops during or after therapy with cortisone or corticotropin, the presence of tuberculosis should be suspected until it is proved otherwise. When these drugs must be used when a tuberculous infection is known to exist, it may be wise to employ at the same time antimicrobial agents such as streptomycin and para-aminosalicylic acid. The findings and conclusions of these two committees of the American Trudeau Society emphasize the need for caution when new drugs are employed.

RHODOPSIN

It has long been known that optimal function of the eye is associated with adequate nutrition, and, once the nutritive importance of vitamin A had been established, this food factor was shown to be involved in the photochemical cycle that is the physiological basis of sight. The retina is known to be extremely rich in vitamin A potency,[1] and it has been shown [2] that when visual purple, or rhodopsin, is acted on by light, a carotenoid yellow pigment (retinene) is formed, which, in turn, gives rise to vitamin A. In natural dark adaptation this process is reversed and rhodopsin is regenerated in the rods of the retina. In the course of the dissociation of rhodopsin, there is formed, in addition to the retinene, a protein called opsin. In recent reports [3] successful experiments are described in which rhodopsin has been synthetized from opsin and high concentrations of synthetic retinene; the resulting synthetic product was shown to be visual purple by physical and chemical tests, although the efficiency of the in vitro reaction does not approach that in the intact retina.

Further studies [4] of the circumstances attending the effect of light on rhodopsin have shown that the in vitro synthesis of this pigment can be accomplished when the retinene (vitamin A aldehyde) is prepared in the laboratory from fish liver oils but not when crystalline vitamin A is the source of the retinene. This observation has led to the conclusion that some cis-isomer of vitamin A rather than the trans- form is essential for the reformation of retinene. It appears that the principal action of light on retinene is to isomerize it rather than to decompose it. By further analysis of the response of retinene to various wavelengths of light, the conclusion has been reached that the retinene emerging from the bleaching of rhodopsin is a different isomer from that subsequently entering into the synthesis of the visual purple.

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[1] Committee on Therapy, American Trudeau Society: The Effect of Cortisone and/or Corticotropin on Tuberculous Infection in Man, Am. Rev. Tuberc. **66**:254-256 (Aug.) 1952. Laboratory Subcommittee of the Committee on Medical Research, American Trudeau Society: The Effect of Cortisone and/or Corticotropin on Experimental Tuberculous Infections in Animals, ibid. **66**:257-259 (Aug.) 1952.

[1] Yudkin, A. M.; Kriss, M., and Smith, A. H.: Vitamin A Potency of Retinal Tissue, Am. J. Physiol. **97**:611, 1931.

[2] Wald, G.: Carotenoids and Visual Cycle, J. Gen. Physiol. **19**:351, 1935.

[3] Wald, G., and Hubbard, R.: Synthesis of Rhodopsin from Vitamin A_1, Proc. Nat. Acad, Sc. **36**:92, 1950. Wald, G., and Brown, P. K.: Synthesis of Rhodopsin from Retinene, ibid. **36**:84, 1950.

[4] Hubbard, R., and Wald, G.: Cis-Trans Isomers of Vitamin A and Retinene in Vision, Science **115**:60, 1952.

It becomes of considerable interest to determine the nature of the chemical bond between the protein (opsin) and the chromatophore (retinene) of the rhodopsin. In a recent report [5] experimental observations are described that throw light on this point. Although the aldehyde group of the retinene might be expected to combine with the amino groups of the protein, evidence is established to show that the retinene combines with opsin through the sulfhydryl groups of the latter. To further prove this point, it was shown that, in the course of bleaching of rhodopsin, reactive sulfhydryl groups appear in the system. Although these circumstances indicate that the bleaching of visual purple by light is similar to the well-known denaturation of proteins, the changes in rhodopsin during bleaching suggest that the structure of visual purple is more complex than reactions of the sulfhydryl group might indicate.

Despite the newer observations that a definite spacial relationship in the atoms of vitamin A is required for the synthesis of rhodopsin, the fact remains that the body has no other source of this indispensable factor than the food. The factor of safety lies in the fact that, when one isomer of vitamin A is fed, the liver soon contains a mixture of known isomers.

VACATIONS AND DIARRHEA

Each year, particularly in the summer and fall, physicians must treat patients with serious diarrheal disease resulting from vacation trips. Study of this problem for many years [1] shows there are three basic aspects, involving the layman, the family physician, and public health officials. The family physician is the most important link between the patient and public health department. Without him it would be impossible to carry out the epidemiological and bacteriological studies necessary for control. Since 1934 the Dysentery Registry has carried out educational campaigns each summer for laymen. The film, "From Hand to Mouth,"[2] has been in continuous use in schools, colleges, and hospitals both here and abroad since its first showing at the New York World's Fair.

Physicians have been alerted to the necessity for isolation and fecal cultures and recognition of new clinical forms of diarrheal disease. Public health agencies are now cognizant of the wide prevalance of infectious diarrhea and are making vigorous efforts for their control, and yet, to those interested in this field of medicine, these diseases seem to continue unabated. However, the reasons appear to be quite obvious. The average layman takes his vacation with little or no thought to the hygienic or sanitary conditions he is to encounter or with a feeling of helplessness over their control; he may not have the protection of a well-organized city health department; boiling the drinking water does not solve all the problems of infectious diarrhea; and dirty eating utensils, infected foodhandlers, unsanitary privies, and fly prevalence are very important but not so recognized by many travellers. Furthermore, the family physician sometimes is beset with the difficulty of differentiating the causes of the diarrhea without bacteriological help. He is often troubled by the reluctance of the patient or family to have the case reported, to have feces cultured, or even to carry out simple isolation procedures. Health departments are hampered by failure to report cases of diarrheal diseases and by lack of personnel to inspect summer hotels, camps, and restaurants. Many resort areas have seasonal overloads and water supplies and sewage disposal systems that may be adequate for the normal low population level become inadequate.

The enormity of the problem may be ascertained from recent figures.[3] In 1951, more than 72 million Americans took 25 million vacation trips, and it was estimated that the tourist pleasure industry involved an annual expenditure of 7 to 10 billion dollars. There are 42,800,000 passenger cars, and trailer coach manufacturers produced 67,335 trailers in 1951. In the same year the National Park System had 36,679,316 visitors. Today there are more than 30,000 motels and 15,000 hotels in the United States, all of which encourages Americans to travel.

Modern advances in the control of diarrheal disorders have been due to a correlation of increased knowledge of the clinical, pathological, and bacteriological aspects. In the Shigella and Salmonella diseases early and repeated sigmoidoscopic studies have revealed as much or more than examinations at necropsy. For example, it has been demonstrated that patients with Shigella disease (bacillary dysentery) due to contact often have no clinical complaints yet have the characteristic one, two, or three stage pathological changes in the colon. The practical implication is that the feces of all persons who have had contact with Shigella disease must be cultured whether the persons have symptoms or not.

The diarrheal disease known as chronic ulcerative colitis remains one of the most difficult problems in medicine. Much of the literature appears to be controversial. There is no doubt, however, that in some instances the disease follows acute bacillary dysentery,[1] as shown by serial sigmoidoscopic studies carried out in the same person from the initial acute specific phase to the fully developed chronic form. Even a possibility of lessening the frequency of this one disease by preventing bacillary dysentery and other acute infectious diarrheas warrants careful study of the control of diarrheal disorders.

AMERICAN LEGION PHYSICIANS

Members of the American Medical Association who are planning to attend the American Legion Convention in New York City are invited to visit the rooms that will be reserved for the Association at the Statler Hotel from Aug. 24 to 27. Several individuals from the headquarters staff will be present to receive A. M. A. members.

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[5] Wald, G., and Brown, P. K.: The Role of Sulfhydryl Groups in the Bleaching and Synthesis of Rhodopsin, J. Gen. Physiol. **35**: 797, 1952.

[1] Felsen, J.: Bacillary Dysentery, Colitis and Enteritis, Philadelphia, W. B. Saunders Company, 1945.

[2] Permanent copies are in the Film Repository of the Wistar Institute for approved films in anatomy and biology. The film is also available for loan without cost from The Dysentery Registry, The Bronx Hospital, New York, N. Y.

[3] Biemiller, C. L.: Our Wonderful Restlessness, Holiday **12**: 46 (July) 1952.

MEDICAL NEWS

ALABAMA

Diagnostic Poliomyelitis Screening.—Through its consulting committee, the Montgomery County Health Department is prepared to render community service in the event of a poliomyelitis epidemic. The committee is composed of pediatricians, pediatric surgeons, and an orthopedist appointed by the county health department each year to serve during the poliomyelitis season. Two physicians, including one member of the consultant group, are required to see each patient under observation. Consultant service is without charge, regardless of the patient's ability to pay. Confirmed poliomyelitis cases must be reported to the county health department; however, physicians are asked to report suspected cases so that the health department may be alerted for possible emergency care of such cases. Except in extreme cases with paralytic or bulbar involvement, hospitalization of poliomyelitis and suspect cases is discouraged. A physical therapist employed by the county health department and paid by poliomyelitis funds provides home care to patients in Montgomery County and seven adjacent counties.

CONNECTICUT

Clinical Congress at New Haven.—The Connecticut Clinical Congress of the Connecticut State Medical Society and the Yale University School of Medicine will be held at New Haven Hospital and the Yale School of Medicine, New Haven, Sept. 17 and 18. Three sessions will be held simultaneously in three different meeting places, giving a broad selection of topics. Discussions on Wednesday include diabetic coma, arthritis, toxemias of pregnancy, gout, hyperthyroidism, arteriosclerosis, causes of sudden death, and new therapeutic agents in tuberculosis and hypertension. At 3:30 p. m. there will be a pediatric seminar on "Abdominal Pain in Infancy and Childhood," "Anemias in Childhood," and "Renal Diseases in Children." Other presentations will include "Progress Report on ACTH and Cortisone," "Clinical Application of Radioactive Isotopes in Malignancy," and "Indications for and Contraindications to Blood Transfusions." Thursday sessions will be devoted to surgery, including acute head injuries, severe burns, fractures, carcinoma of the breast and lung, and surgery of the hand, heart, and genitourinary, gastrointestinal, and biliary tracts.

Registration for members of the society will be $3 and for nonmembers $4. Medical students, interns, and residents will be the guests of the congress, if properly certified. Inquiries should be addressed to Dr. Creighton Barker, Executive Secretary, Connecticut State Medical Society, 160 St. Ronan St., New Haven.

DISTRICT OF COLUMBIA

University News.—At the commencement of Georgetown University, June 9, Rev. Paul A. McNally, S.J., Ph.D., dean of Georgetown's School of Medicine and director of its Medical Center, received the degree of honorary doctor of science "in recognition of his administrative as well as scientific ability . . ."

Hunter Award Established.—The Medical Society of the George Washington University, Washington, D. C., has established the Oscar B. Hunter Memorial Fund, in memory of the late eminent pathologist, who, at the time of his death, was vice-president of the American Medical Association. The trust fund will permit a cash award of $100 each year to the university's senior medical student of most notable scholarship in pathology. The first Oscar Benwood Hunter Award in Pathology was recently made at precommencement ceremonies to Allan Hugh Stewart of Mount Shasta City, Calif.

ILLINOIS

Fifty Year Club.—The Madison County Medical Society recently gave a dinner honoring Drs. Alfred E. Everett of Granite City and Mather Pfeiffenberger of Alton, at which they received the 50 year club insignia of the Illinois State Medical Society. Dr. Groves B. Smith, Godfrey, was the toastmaster, and Dr. Edwin S. Hamilton, Kankakee, a trustee of the American Medical Association, was the principal speaker.

Mortality from Communicable Diseases.—A decline of about 7% in mortality from the common communicable diseases was reported in Illinois in 1951. Of nine communicable infections, only three, measles, meningitis, and influenza, were responsible for higher mortality during the year. The number of deaths from measles increased from 8 in 1950 to 33 in 1951. Mortality from meningitis increased from 49 deaths in 1950 to 58 in 1951. Poliomyelitis deaths dropped from 130 in 1950 to 103 in 1951; syphilis from 558 to 490; tuberculosis from 2,215 to 1,890; pneumonia from 2,548 to 2,542; and whooping cough from 22 to 15. A single death from diphtheria was reported for each of the last two years.

Chicago

Society News.—At the annual meeting of the Chicago Gynecological Society, June 20, Dr. Edward M. Dorr was elected president; Dr. Henry Buxbaum, president-elect; Dr. Robert M. Grier, Evanston, Ill., vice-president; Dr. Edwin J. DeCosta, secretary; Dr. Harry Boysen, treasurer; Dr. John R. Wolff, pathologist; and Dr. Janet E. Towne, editor.

Medical Tour for Business Executives.—Research directed toward expansion of life expectancy for persons over 60 was recently demonstrated to Chicago businessmen at the University of Chicago under the sponsorship of 35 Chicago businessmen who form the university's Council on Medical and Biological Research. Key projects in various stages of development, aimed at improving treatment of cancer, degenerative diseases, heart ailments, and high blood pressure, were observed in the laboratories.

Dr. Lowell T. Coggeshall, dean of the division of biological sciences of the university, stated that two hospitals, where research will be concentrated on diseases of the aged, were being completed in the university's medical center at a cost of $4,500,000. Other scientists who spoke during the tours included the following: Dr. Dwight E. Clark, "Hyperthyroidism"; Dr. Charles B. Huggins, "Adrenalectomy"; Dr. Cyrus E. Rubin, "Early Diagnosis of Gastrointestinal Cancer"; Dr. John R. Lindsay, "Causes of Deafness"; James R. Blayney, D.D.S., "Dental Research and Degenerative Diseases"; and Ward C. Halstead, Ph.D., "Psychological Effects of Brain Injuries and Differential Aging."

KANSAS

Dr. Menninger's Ninetieth Birthday.—Dr. Charles F. Menninger, founder of the Menninger Clinic and chairman of the Board of Trustees of the Menninger Foundation, observed his 90th birthday on July 11 in Topeka. Dr. Menninger is a former president of the Shawnee County Medical Society and has served as president of the Topeka Board of Health. He recently presided at a meeting of the foundation's board in Chicago.

MASSACHUSETTS

Dr. O'Hara Resigns as Dean.—Dr. Dwight O'Hara has resigned as dean of Tufts College Medical School, Boston, and will become chief of the professional services at the Veterans Administration Clinics in Boston. He will, however, retain his position as professor of preventive and industrial medicine at

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Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

Tufts. Dr. O'Hara served as vice-dean from 1936 to 1941 and acting dean until 1945, when he was appointed dean of the medical school. He is a past president of the Massachusetts Medical Society.

Personal.—Dr. William A. Hinton has retired as chief of the department of clinical laboratories of the Boston Dispensary after 36 years of service. He will continue to serve in a consulting capacity and will also retain his position as one of the directors in the Institute of Laboratories of the Massachusetts Department of Public Health. Dr. Mario Stefanini, associate research professor in medicine at Tufts College Medical School, Boston, has been appointed acting chief of the clinical laboratories in the Boston Dispensary to succeed Dr. Hinton. Dr. Stefanini is affiliated with the New England Center Hospital and is associate director with Dr. William Dameshek of the blood bank and the hematology research laboratory.——Dr. Alexander J. A. Campbell, clinical professor of surgery at Tufts College Medical School, Boston, has been awarded an honorary doctor of laws degree by his alma mater, St. Francis Xavier University, Antigonish, Nova Scotia.——Dr. Maurice B. Strauss, clinical associate in medicine, Harvard Medical School, Boston, has been appointed professor of clinical medicine at the Boston University School of Medicine.

MICHIGAN

Services Donated to Children's Camp.—Replying to a request from the Burns Attendance Center of the Detroit Public Schools, for free services of physicians to examine about 75 children recommended for the Free Press Fresh Air Camp, seven physicians recently donated their services.

Dr. Eisman Retires.—Dr. Clarence H. Eisman has retired as diagnostician for the Detroit Department of Health after 45 years of service, during most of which he was also in private practice. Since 1947 he has been medical co-ordinator for the Michigan Crippled Children's Commission.

MINNESOTA

State Association Honors Dr. Condit.—Dr. William H. Condit, Minneapolis, was given the Minnesota State Medical Association's distinguished service award, at the recent annual banquet held in Minneapolis. The citation pointed out that "his has been an immeasurable contribution and an individual mark of proud achievement in Minnesota's medical progress." Dr. Condit has served the association for 30 years as councilor, first vice-president, and treasurer.

MONTANA

Surgeons to Meet in Billings.—The Montana Chapter of the American College of Surgeons will hold its annual meeting Aug. 23 at the Northern Hotel in Billings under the presidency of Dr. Raymond E. Benson, Billings.

The following program will begin at 9 a. m.:

Alfred M. Lueck, Livingston, Traumatic Surgical Emergencies in So-Called Minor Surgery.
Sidney C. Pratt, Miles City, Multiple Myeloma.
Neil M. Leitch, Kalispell, Patent Urachus.
Roger Anderson, Seattle, Backache: Practical Methods of Diagnosis and Treatment, and Simple Methods of Treating Difficult Fractures.
Charles W. McLaughlin, Omaha, Emergency Abdominal Surgery in Infancy and Childhood, and The Management of Acute Intestinal Obstruction.
Robert S. McCleery, Great Falls. Technic and Results of a Modified McVay Herniorrhaphy.
Alexander C. Johnson, Great Falls, The Paroxysmal Disorders of the Cranial Nerves.

At 7 p. m. there will be a reception, followed by a banquet for all members and guests. Rev. Father Norbert C. Hoff of Butte will present an address entitled "Tracks." All Montana physicians are cordially invited to attend this conference.

NEVADA

State Medical and Surgical Meeting.—The Nevada State Medical Association and the Reno Surgical Society will hold a joint annual meeting at the Riverside Hotel in Reno, Aug. 20-23. Dr. J. Vernon Cantlon of Reno is president of the state association, and Dr. Kenneth F. MacLean is president of the surgical society. Guests of honor and their first presentations are as follows:

Bernard J. Hanley, Los Angeles, Total Abdominal Hysterectomy for Benign Uterine Disease.
Frederick A. Coller, Ann Arbor, Mich., Cancer of the Colon.
Verne R. Mason, Los Angeles, Eosinophilia.
Charles F. McCuskey, Los Angeles, Changing Concepts in Anesthesia.
Richard P. Middleton, Salt Lake City, Management of Ureteral Calculi.
Alton Ochsner, New Orleans, Early Diagnosis and Treatment of Cancer of the Stomach.
Max Samter, Chicago, Has ACTH Changed Clinical Medicine?
Philip D. Wilson, New York, Treatment of Low Back Pain and Sciatica.

In addition to their lectures, the guest speakers will conduct informal discussions during the luncheon meetings. The Woman's Auxiliary to the Nevada State Medical Association will hold its annual meeting Aug. 21-22.

NEW JERSEY

New Employee Dispensary.—RCA Victor recently dedicated a new $50,000 dispensary for employees of its Camden plant and offices. The new dispensary is air-conditioned and has soundproof ceilings. It is open 18 hours each workday, with a plant physician and five nurses in attendance. Treatment is provided in cases where illness or injury is incurred by employees in the performance of their duties. In cases of illness or injury not incurred in the line of duty, the dispensary gives emergency treatment if required and then refers patients to their own physicians or to hospitals.

Harrison S. Martland Award.—The Essex County Pathological and Anatomical Society offers a prize of $250 under the annual Harrison S. Martland Award for the best unpublished paper on pathology or anatomy. The paper need not be based on original research but may be confined to a review and reinterpretation. The essayist will be known to the committee of judges by number only. Eligibility is limited to resident physicians and interns in the hospitals of New Jersey. Contributions must not exceed 5,000 words. Five copies of each manuscript must be mailed not later than Oct. 15 to Dr. Murray W. Shulman, Newark City Hospital, Newark.

NEW YORK

Study of Convicted Sex Offenders.—Dr. Bernard C. Glueck Jr., Ossining, has been appointed director of the state's study of convicted sex offenders, which has been under way for several years at Sing Sing Prison under the auspices of the New York State Psychiatric Institute. Dr. Glueck, who has been psychiatrist at Sing Sing since 1949, succeeds Dr. David Abrahamsen, who resigned. Dr. Glueck served in the Army Air Force in World War II as flight surgeon.

Dr. Dellaporta to Head Eye Bank.—Dr. Angelos Dellaporta of the University Eye Clinic of Vienna has arrived in Buffalo to head the clinical and ophthalmologic research program of the Buffalo Eye Bank & Research Society, Inc., an organization founded by the Lions Clubs of Western New York, which receives financial assistance from the Lions Blind Seal Committee, Lions Clubs, the general public, and the Community Chest.

Dr. Dellaporta will also lecture in ophthalmology and teach laboratory methods and ophthalmologic and pathological research methods at the University of Buffalo School of Medicine, which cooperated with the Eye Bank in bringing him to America.

New York City

Community Health Control Grant.—Community Research Associates, Inc., has been awarded $550,000 by the Grant Foundation for continuation of basic scientific research to aid the control of health and welfare problems. The projects will involve prevention and control of serious consequences of chronic disease and disability, behavior and emotional disorders, and chronic indigency and dependency. The first year will be devoted to selection and negotiation with cities suitable for experimentation. It is planned that the other four years will see the establishment of simultaneous local projects, the working out of classifications and the control procedures, analysis of findings, and preparation of reports.

NORTH CAROLINA

Postgraduate Symposium.—The New Hanover County Medical Society Symposium will be held in Lumina Ballroom, Wrightsville Beach, Aug. 22. The speakers will include:

H. Hudnall Ware Jr., professor of obstetrics, Medical College of Virginia, Richmond, Toxemia of Pregnancy.
Virgil P. W. Sydenstricker, professor of medicine, Medical College of Georgia, Augusta, Collagen Diseases.
Eugene P. Pendergrass, professor of radiology, University of Pennsylvania School of Medicine, Philadelphia, Cancer of the Lung with Comment Concerning Some of the Difficulties of Early Diagnosis.
Joseph Stokes Jr., professor of pediatrics, University of Pennsylvania School of Medicine, Philadelphia, Recent Studies in Certain Viral Diseases.
Robert L. Sanders, professor of surgery, University of Tennessee College of Medicine, Memphis, Surgical Lesions of the Colon.

The meeting will include a social hour and barbecue dinner in the evening.

TENNESSEE

Aid for Polio Patients.—According to policies established by the Nashville Pediatric Society, only paralytic polio cases in the community are recommended for hospital care. Hospitalization for patients with mild paralysis, however, is usually based on individual need. Suspect cases are referred to the diagnostic service of Vanderbilt University Hospital, which is the referral center for Nashville and the larger area including middle Tennessee, Southern Kentucky, and Northern Alabama. Any physician may refer his patients to the diagnostic clinic for consultation, blood counts, spinal fluid studies, and any other laboratory tests that are needed. A moderate fee is charged for the service. Patients who show no evidence of paralysis are referred back to their physicians for home care.

TEXAS

Postgraduate Conferences.—The Dallas Southern Clinical Society announces the following postgraduate conferences to be held in Room 433, Medical Arts Building:

Sept. 8-11, Cardiology, Tinsley R. Harrison, Alabama, and Don W. Chapman, Houston.
Sept. 29-Oct. 1, Pediatrics and Pediatric Dermatology, Gilbert B. Forbes, Dallas, and J. Lamar Callaway, Durham, N. C.
Nov. 3-5, Obstetrics-Gynecology, Albert W. Diddle, Knoxville, Tenn.

GENERAL

Society News.—At the annual meeting of the New England Society of Anesthesiologists held in Boston, May 13, the follow- g officers were elected for 1952-1953: president, Dr. Henry K. cker, Boston; vice-president, Dr. Stevens J. Martin, Hartford, ı.; and secretary-treasurer, Dr. Francis J. Audin, Boston.

Maxillofacial Surgeons to Meet in San Diego.—The American Society of Maxillofacial Surgeons will hold its annual meeting in San Diego, Calif., Sept. 10-13, at the Hotel del Coronado, under the presidency of Dr. Reed O. Dingman, Ann Arbor, Mich. By invitation the following collaborators will hold a cleft palate symposium on Saturday from 10:30 a. m. to 12 noon.

Herbert A. Ecker, Williamsport, Pa., Management of Premaxilla and Prolabium in Cleft Palate Patients.
Monroe K. Ruch, Los Angeles, Experiences with the Pharyngeal Flap Operation.
Benjamin F. Edwards, Santa Monica, Calif., Evaluation of Cleft Palate Surgery.

Other speakers by invitation include:

Thomas Giannini, San Diego, Calif., Total Nasal Reconstruction.
Michel Dechaume, Paris, France, Myeloplaxes Tumors of Maxillary Bones.
Clarence E. Rees and Maurice J. Brown, San Diego, Calif., Thyroglossal Duct Cysts.
Clyde Litton, Charleston, W. Va., Infantile Cortical Hyperostosis.
Harold I. Harris, Los Angeles, Congenital Unilateral Absence of the Ramus of the Mandible with Suggested Treatment in Young Children.
Alfred A. deLorimier, San Francisco, Arthropathies in the Temporomandibular Joint.
William S. Kiskadden and Sanford R. Dietrich, Los Angeles, and Clifford Barber, Glendale, Calif., Preliminary Review of Treatment of Micrognathia.
Paul A. Shea, San Diego, Calif., Electromyograph as an Aid in Diagnosis and Treatment of Maxillofacial Disease.
David M. Mayer and Wilson A. Swanker, New York, Use of Gelatinous Bone in Facial Defects.

American Society of Human Genetics.—The annual meeting of this society will be held September 8-10 in Martha Van Rensselaer Hall, Cornell University, Ithaca, N. Y., under the presidency of Dr. Franz J. Kallmann of New York. On Monday afternoon there will be a symposium on "The Genetics of Various Constitutional Defects." On Tuesday afternoon a symposium will deal with "Light from Animal Experimentation on Human Heredity." Wednesday morning there will be a symposium on "Counselling Clinics in Human Heredity" and at 11 o'clock a symposium on "Nomenclature in Human Genetics." Wednesday afternoon is left unscheduled so that members may visit laboratories on the campus or attend the symposium on biochemical evolution sponsored by the Society for the Study of Evolution and the American Society of Naturalists.

The annual dinner will be held at 6:15 p. m. in the Terrace Room of the Willard Straight Hotel, at which time the presidential address on "Human Genetics as a Science, as a Profession, and as a Social-Minded Trend of Orientation" will be delivered. Since only 100 persons can be admitted, early reservations may be made through the office of the New York State Psychiatric Institute, 722 W. 168th St., New York 32, before Sept. 3 or may be purchased at the registration desk before noon Sept. 8.

Prevalence of Poliomyelitis.—According to the National Office of Vital Statistics, the following number of reported cases of poliomyelitis occurred in the United States and its territories and possessions in the weeks ended as indicated:

Area	July 26, 1952	July 19, 1952	July 28, 1951
New England States			
Maine	3	1	2
New Hampshire	1	..	1
Vermont	2	1	1
Massachusetts	8	3	22
Rhode Island	..	..	..
Connecticut	15	6	6
Middle Atlantic States			
New York	76	68	50
New Jersey	10	16	9
Pennsylvania	24	18	17
East North Central States			
Ohio	179	69	31
Indiana	34	19	6
Illinois	68	46	58
Michigan	79	56	58
Wisconsin	33	26	35
West North Central States			
Minnesota	38	27	20
Iowa	116	82	13
Missouri	33	13	9
North Dakota	1	..	4
South Dakota	15	15	2
Nebraska	72	55	8
Kansas	24	37	24
South Atlantic States			
Delaware	4	3	3
Maryland	3	7	2
District of Columbia	..	1	5
Virginia	15	17	5
West Virginia	32	22	3
North Carolina	15	14	15
South Carolina	..	1	8
Georgia	18	25	42
Florida	20	22	14
East South Central States			
Kentucky	51	73	7
Tennessee	15	24	41
Alabama	12	20	41
Mississippi	54	51	27
West South Central States			
Arkansas	16	14	21
Louisiana	39	83	31
Oklahoma	65	46	38
Texas	259	243	91
Mountain States			
Montana	8	6	..
Idaho	16	4	3
Wyoming	2	1	4
Colorado	31	10	62
New Mexico	22	19	2
Arizona	7	8	7
Utah	6	4	13
Nevada	..	..	8
Pacific States			
Washington	36	38	9
Oregon	14	19	7
California	75	59	101
Territories and Possessions			
Alaska	..	..	..
Hawaii	10	12	1
Puerto Rico	1	1	1
Total	1,677	1,405	991

FOREIGN

Congress on History of Medicine.—The Congress of the International Society for the History of Medicine will convene at Nice, Cannes, and Monaco, Sept. 8-14. The program includes discussion of the precursors, medical relations between the West and the Orient, from empiricism to experimental medicine in the Mediterranean Basin, influence of the schools of Salerno and Montpellier, medical iconography in the 16th century, efforts made in various countries in favor of the teaching of the history of medicine, and the Jean Cantacuzene Commission on medical folklore: prehistoric elements in medical folklore.

Congress on Medical Records.—The First International Congress on Medical Records will be held in London, Sept. 8-12. Sir John Charles, M.D., F.R.C.P., chief medical officer of the Ministry of Health, will deliver the address of welcome. On Tuesday Dr. P. McKinlay, F.R.S., medical statistician, department of health for Scotland, will speak on "Measurement of Morbidity." Friday morning will be devoted to a symposium on "Reporting Systems." Dr. William P. D. Logan, D.P.H., chief medical statistician, general register office, will open the discussion with a presentation on "Cancer Records." Other speakers will talk on records concerned with mental health, tuberculosis, and public health; general practitioners' records; and discharge studies.

International Congress of Neuropathology.—The First International Congress of Neuropathology will be held in Rome, Italy, Sept. 8-13, under the presidency of Dr. Mario Gozzano, professor of neurology and psychiatry at the University of Rome.

Topics for discussion will include the histopathology of demyelinating diseases, cerebral vascular disease, schizophrenia, mental deficiencies, and senility. Physicians from the United States who are scheduled to present papers are Drs. Joseph H. Globus, New York; Jost J. Michelsen, Boston; Karl T. Neubuerger, Denver; W. L. Bruetsch, Indianapolis; Clemens E. Benda, Arlington, Mass.; and George A. Jervis, Thiells, N. Y. Dr. George B. Hassin, Chicago, is an honorary president of the congress.

MEETINGS

American Medical Association Public Relations Institute, Edgewater Beach Hotel, Chicago, Sept. 4-5. Mr. Leo E. Brown, 535 N. Dearborn St., Chicago 10, Director.

Alaska Territorial Medical Association, Anchorage, Aug. 21-23. Dr. William P. Blanton, P. O. Box 2569, Juneau, Secretary.

American Academy for Cerebral Palsy, Durham, N. C., Oct. 2-4. Dr. Meyer A. Perlstein, 4743 North Drake Ave., Chicago 25, Secretary.

American Association of Blood Banks, Hotel Schroeder, Milwaukee, Oct. 9-11. Miss Marjorie Saunders, 3500 Gaston Avenue, Dallas 1, Texas, Secretary.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, The Homestead, Hot Springs, Va., Sept. 4-6. Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Texas, Secretary.

American College of Surgeons,Waldorf-Astoria, New York, Sept. 22-26. Dr. Michael L. Mason, 40 East Erie St., Chicago 11, Secretary.

American Congress of Physical Medicine, Roosevelt Hotel, New York, Aug. 25-29. Dr. Frances Baker, One Tilton Ave., San Mateo, Calif., Secretary.

American Hospital Association, Philadelphia, Sept. 15-18. Mr. *George* Bugbee, 18 East Division St., Chicago 10, Executive Director

American Medical Writers' Association, Jefferson Hotel, St. Louis, Oct. 1. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

American Physiological Socety, New Orleans, Sept. 4-6. Dr. E. F. Adolph, University of Rochester School of Medicine and Dentistry, Rochester, N. Y., Secretary.

American Roentgen Ray Society, Shamrock Hotel, Houston, Texas, Sept. 23-26. Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Secretary.

American Society of Maxillofacial Surgeons, Hotel del Coronado, San Diego, Calif., Sept. 10-13. Dr. Casper M. Epsteen, 25 East Washington St., Chicago 2, Secretary.

Annual Gulf Coast Regional Conference on Industrial Health, Rice Hotel, Houston, Tex., Sept. 24-27. Dr. W. H. Hamrick, P. O. Box 2371, Houston, Tex., Chairman.

Biological Photographic Association, Hotel New Yorker, New York, Sept. 10-12. Mr. Lloyd E. Varden, Pavelle Color, Inc., 533 W. 57th St., New York 19, Secretary.

Colorado State Medical Society, Stanley Hotel, Estes Park, Sept. 9-12. Mr. Harvey T. Sethman, 835 Republic Bldg., Denver 2, Executive Secretary.

Constantinian Society, Lake Placid Club, Lake Placid, N. Y., Oct. 1-4. Dr. C. F. Shook, P. O. Box 1035-36, Toledo 1, Ohio, Secretary.

Delaware, Medical Society of, Rehoboth, Sept. 8-10. Dr. Andrew M. Gehret, 822 North American Bldg., Wilmington, Secretary.

District of Columbia, Medical Society of the, Hotel Statler, Washington, Sept. 29-Oct. 1. Mr. Theodore Wiprud, 1718 M St. N.W., Washington, Secretary.

Kansas City Southwest Clinical Society, Kansas City, Mo., Oct. 6-9. Dr. Galen M. Tice, 630 Shukert Bldg., Kansas City, Kansas, Secretary.

Kentucky State Medical Association, Brown Hotel, Louisville, Oct. 7-9. Dr. Bruce Underwood, 620 S. Third St., Louisville 2, Secretary.

Michigan State Medical Society, Book-Cadillac Hotel, Detroit, Sept. 24-26. Dr. L. Fernald Foster, 606 Townsend St., Lansing 15, Secretary.

Mississippi Valley Medical Society, Jefferson Hotel, St. Louis, Oct. 1-3. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

Montana Medical Association, Florence Hotel, Missoula, Sept. 18-21. Mr. L. R. Hegland, 104 North Broadway, Billings, Executive Secretary.

Neurosurgical Society of America, The Cloister, Sea Island, Ga., Sept. 18-20. Dr. C. D. Hawkes, 22 North Manassas St., Memphis 5, Tenn., Secretary.

Nevada State Medical Association, Reno, Aug. 21-23. Dr. William A. O'Brien III, 505 Chestnut St., Reno, Secretary.

New Hampshire Medical Society, Mt. Washington Hotel, Bretton Woods, Sept. 7-9. Dr. Deering G. Smith, 44 Chester St., Nashua, Secretary.

North Pacific Pediatric Society, Challenger Inn, Sun Valley, Idaho, Sept. 19-20. Dr. S. Gorham Babson, 1107 S.W. 13th Ave., Portland 5, Ore., Secretary.

North Pacific Society of Internal Medicine, Sun Valley, Idaho, Sept. 19-20. Dr. Robert L. King, 1115 Terry Ave., Seattle 1, Wash., Secretary.

North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, Texas, Sept. 17. Dr. E. Aubrey Cox, Hamilton Bldg., Wichita Falls, Texas, Chairman.

Oregon State Medical Society, Portland, Oct. 8-11. Dr. Robert F. Miller, 833 S.W. 11th Ave., Portland 5, Secretary.

Pacific Dermatologic Association, Palace Hotel, San Francisco, Aug. 29-30. Dr. Ervin H. Epstein, 447 29th St., Oakland, Calif., Secretary.

Pennsylvania, Medical Society of the State of, Bellevue-Stratford Hotel, Philadelphia, Sept. 28-Oct. 2. Dr. Walter F. Donaldson, 500 Penn Ave., Pittsburgh 22, Secretary.

Piedmont Proctologic Society, Savannah Beach, Ga., Aug. 23. Dr. B. Richard Jackson, 224 Hillsboro St., Raleigh, N. C., Secretary.

Reno Surgical Society Conference, Reno, Nevada, Aug. 21-23. Dr. Olin C. Moulton, 130 N. Virginia St., Reno, Nevada, Chairman.

Tri-State Medical Society of Texas, Louisiana and Arkansas, Texarkana, Texas, Oct. 2-3. Dr. Albert M. Hand, 619 Main St., Texarkana, Texas, Secretary.

U. S. Chapter, International College of Surgeons, Conrad Hilton Hotel, Chicago, Sept. 2-5. Dr. Arnold S. Jackson, 1516 Lake Shore Drive, Chicago, Executive Secretary.

Utah State Medical Association, Salt Lake City, Sept. 4-6. Dr. T. C. Weggeland, 42 S. Fifth East St., Salt Lake City, Secretary.

Vermont State Medical Society, Mt. Washington Hotel, Bretton Woods, N. H., Sept. 7-9. Dr. James P. Hammond, 128 Merchants Row, Rutland, Secretary.

Virginia, Medical Society of, Jefferson Hotel, Richmond, Sept. 28-Oct. 1. Mr. Robert L. Howard, 1105 West Franklin St., Richmond, Executive Secretary.

Washington State Medical Association, Olympic Hotel, Seattle, Sept. 13-17. Dr. Bruce Zimmerman, 338 White-Henry-Stuart Bldg., Seattle, *Secretary.*

Wisconsin, State Medical Society of, Hotel Schroeder, Milwaukee, Oct. 6-8. Mr. Charles H. Crownhart, 704 E. Gorham St., Madison, Secretary.

INTERNATIONAL

Aero Medical Association Interim Meeting, Paris, France, Sept. 26-28. Dr. Armand Robert, 2 Rue Marbeuf, Paris 8e, France, Secretary.

American Congress of Industrial Medicine, Rio de Janeiro, Brazil, Sept. 20-28. Prof. Dr. Jose Pedro Reggi, Calle Arenales No. 981 Buenos Aires, Argentina, Director General.

Australasian Medical Congress, Melbourne Victoria, Australia, Aug. 22-30. Dr. C. H. Dickson, Medical Society Hall, 426 Albert St., East Melbourne, Victoria, Australia, General Secretary.

Congress of International Institute of Embryology, University of Utrecht, Zoological Laboratory, Utrecht, Netherlands, Aug. 27-31. Dr. M. W. Woerdeman, Amsterdam, Holland, President.

European Congress of Cardiology. University of London, Bloomsbury, W.C.1, England, Sept. 10-12. Dr. K. Shirley Smith, 35 Wimpole St., London, W.1, England, Secretary.

European Society of Cardiovascular Surgery, Strasbourg, France, Oct. 3-4. Sir James Learmonth, Department of Surgery, University New Buildings, Edinburgh 8, Scotland.

Interamerican Cardiological Congress, Buenos Aires, Argentina, Aug. 31-Sept. 7. Dr. Blas Moia, Larrea 1132, Buenos Aires, Argentina, Secretary-General.

Inter-American Congress of Public Health, Havana, Cuba. Sept. 26-Oct. 1. Pan-American Sanitary Bureau, 2001 Connecticut Ave., Washington, D. C.

Inter-American Congress of Radiology, Mexico City, Mexico, Nov. 2-8. Dr. Guido Torres Martinez, Marsella No. 11, Mexico, D.F., Secretary-General.

International Congress of Anthropology and Ethnology, Vienna, Austria, Sept. 1-8. For information write: Secretaries, Royal Anthropological Institute, 52 Upper Bedford Place, London, W.C.1, England.

International Congress on Diseases of the Chest, Rio de Janeiro, Brazil, Aug. 24-30. Mr. Murray Kornfeld, 112 East Chestnut St., Chicago 11, Illinois, Executive Secretary.

International Congress of Haematology, Mar del Plata, Argentina, Sept. 21-26. Dr. Carlos Reussi, Anchorena 1710, Buenos Aires, Argentina, Secretary.

International Congress of the History of Medicine, Nice-Cannes, France, and Monaco, Sept. 8-14. Dr. F. A. Sondervorst, 124 Avenue des Allies, Louvain, Belgium, Secretary-General.

International Congress on Hydatid Disease, Santiago, Chile, Nov. 21-24. Organizing Committee, P. O. Box 9183, Santiago, Chile.

International Congress of Internal Medicine, Friends House, London, N.W.1, Eng. Sept. 15-18. Sir Harold Boldero, 12 Pall Mall East, London, S.W.1, England, Secretary.

International Congress of Medical Press, Venice, Italy, Oct. 4-5. Dr. Bruno de Biasio, S. Marco 4646, Venice, Italy, Secretary.

International Congress on Medical Records, London, England, Sept. 7-12. Miss Gwen Perkins, 120 C Street S.E., Washington, D. C., Chairman.

International Congress of Military Medicine and Pharmacy, Montevideo, Uruguay, March 1-7, 1953. Dirreccion General del Servico de Sanitad Militar, 8 de Octubre y Mariano Moreno, Montevideo, Uruguay, Secretariat of the Congress.

International Congress on Neuropathology, Rome, Italy, Sept. 8-13. Dr. Armando Ferraro, 722 W. 168th St., New York, N. Y., U. S. A., Secretary General.

International Congress of Otorhinolaryngology, Amsterdam, Netherlands, June 8-15, 1953. Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam, Netherlands, Secretary.

International Congress of Radiology, Copenhagen, Denmark, July 19-25, 1953. Professor Flemming Norgaard, 10 Oster Voldgade, Copenhagen K., Denmark, Secretary General.

International Congress of Therapeutics, Madrid, Spain, Oct. 16-18. Professor Armijo, Instituto de Farmacologia, Cuidad Universitaria, Madrid, Spain, Secretary.

International Congress of Urology, The Waldorf-Astoria, New York, N. Y., U. S. A., Sept. 15-18. Dr. John A. Taylor, 2 East 54th St., New York, N. Y., U. S. A., Secretary-General.

International Hospital Congress, London, England, May 25-30, 1953. Capt. J. E. Stone, 10 Old Jewry, London, EC2, England, Hon. Secretary.

International Study Conference on Child Welfare, Bombay, India, Dec. 5-12. For information write: All India Save the Children Committee, 5 Carmichael Road, Bombay, India.

International Union Against Tuberculosis, Rio de Janeiro, Brazil, Aug. 24-27. Prof. Etienne Bernard, 66 Blvd. St. Michel, Paris 6e, France, Secretary-General.

Medical Women's International Association, Vichy, France, Sept. 12-16. Dr. Margaret M. Wurts, 27 East Wellesley Road, Montclair, N. J., U. S. A., National Secretary.

Neuroradiological Symposium, Stockholm, Sweden, Sept. 17-20. Docent Ake Lindbom, Symposium Neuroradiologicum, Serafimerlasarettet, Stockholm K., Sweden, Secretary.

Pan American Congress of the Medical Press, Buenos Aires, Argentine, July 12-16, 1953. Secretaria del Congress, 763 Uriburu, Buenos Aires, Argentine.

World Congress of Medicine, Montecatini, Italy, Oct. 16-18. Secretariat: Corso Trieste, 65, Rome, Italy.

World Federation of Mental Health, Brussels, Belgium, Aug. 25-Sept. 1. Dr. J. R. Rees, 19 Manchester St., London, W.1, England, Secretary.

World Medical Association, Athens, Greece, Oct. 12-16. Dr. Louis H. Bauer, 2 East 103d St., New York 29, N. Y., Secretary General.

EXAMINATIONS AND LICENSURE

BOARDS OF MEDICAL EXAMINERS

Alabama: *Examination.* Montgomery, June 23-25. Sec., Dr. D. G. Gill, 537 Dexter Ave., Montgomery.

Alaska:* *Examination.* Juneau. On application in other towns where there are board members. *Reciprocity.* On application. Sec., Dr. W. M. Whitehead, Box 140, Juneau.

Arkansas:* *Examination.* Little Rock, Nov. 6-7. Sec., Dr. Joe Verser, Harrisburg.

California: *Written.* Los Angeles, Aug. 18-21; *Sacramento,* Oct. 20-23. Application must be received at least two weeks prior to date of examination. *Oral for Reciprocity Applicants.* Los Angeles, Aug. 16; San Francisco, Nov. 8. Applicants are requested not to arrange to come to an oral examination until they receive notice from the Credentials Committee. *Oral and Clinical for Foreign Medical School Graduates.* Los Angeles, Aug. 17; San Francisco, Nov. 9. Sec., Dr. Frederick N. Scatena, 1020 N St., *Sacramento.*

Colorado:* *Reciprocity.* Denver, Oct. 14. Exec. Sec., Mrs. B. H. Hudgens, 831 Republic Bldg., Denver.

District of Columbia:* *Reciprocity.* Washington, Sept. 8. Sec., Dr. Daniel L. Seckinger, 4130 E. Municipal Bldg., Washington.

Florida:* *Examination.* Jacksonville, Nov. 23-25. Sec., Dr. Homer L. Pearson, 701 Dupont Bldg., Miami 32.

Georgia: *Reciprocity.* Atlanta, Oct. 14-15. Sec., Mr. R. C. Coleman, 111 State Capitol, Atlanta 3.

Guam: The Commission on Licensure will meet whenever a candidate appears or submits his credentials. Ex. Sec., Dr. Austin W. Matthis, Agana.

Illinois: *Examination.* Chicago, Sept. 23-25. Supt. of Regis., Mr. Charles F. Kervin, Capitol Bldg., Springfield.

Kansas: Topeka, Dec. 10-11. Sec., Dr. O. W. Davidson, 864 New Brotherhood Bldg., Kansas City.

Maryland: *Examination.* Baltimore, Dec. 9-12. Sec., Dr. Lewis P. Gundry, 1215 Cathedral St., Baltimore 1.

Michigan:* *Examination.* Lansing, Oct. 8-10. Sec., Dr. J. Earl McIntyre, 202-4 Hollister Bldg., Lansing.

Minnesota:* Minneapolis, Oct. 21-23. Sec., Dr. J. F. Du Bois, 230 Lowry Medical Arts Bldg., St. Paul 2.

Mississippi: *Reciprocity.* Jackson, December. Asst. Sec., Dr. R. N. Whitfield, State Board of Health, Jackson 113.

Montana: Helena, Oct. 6-8. Sec., Dr. S. A. Cooney, 214 Power Block, Helena.

Nebraska:* *Examination.* Omaha, June 1953. Director, Mr. Husted K. Watson, Room 1009 State Capitol Bldg., Lincoln 9.

New Hampshire: Concord, Sept. 10. Sec., Dr. John S. Wheeler, 107 State House, Concord.

New Jersey: Examination. Trenton, Oct. 21-24. Sec., Dr. E. S. Hallinger, 28 W. State St., Trenton.

North Carolina: *Reciprocity.* Durham, Oct. 13. Sec., Dr. Joseph J. Combs, 419 Professional Bldg., Raleigh.

Ohio: *Reciprocity. Columbus, Oct. 7. Sec.,* Dr. H. M. Platter, 21 W. Broad St., Columbus.

South Carolina: *Reciprocity.* Columbia, Sept. 3. Sec., Mr. N. B. Heyward, 1329 Blanding St., Columbia.

Tennessee:* *Examination.* Memphis, Oct. 1-2. Sec., Dr. H. W. Qualls, 1635 Exchange Bldg., Memphis.

Virginia: *Examination.* Richmond, Dec. 11-13. *Reciprocity.* Richmond, Dec. 10. Sec., Dr. K. D. Graves, 631 First St., S.W., Roanoke, Va.

West Virginia: Charleston, Oct. 13-14. Sec., Dr. N. H. Dyer, *State* Office Bldg #3, Charleston 5.

Wyoming: Cheyenne, Oct. 6. Sec., Dr. Franklin D. Yoder, *State Office* Bldg., Cheyenne.

Virgin Islands: *Examination.* St. Thomas, Nov. 12-13. Sec., Dr. Earle M. Rice, St. Thomas.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

Alaska: *Examination.* On application. Juneau or other towns in Territory as decided by Board. *Reciprocity.* On application. Sec., Dr. C. Earl Albrecht, Box 1931, Juneau.

Arizona: *Examination.* Tucson, Sept. 16. Sec., Mr. Herbert D. Rhodes, Room 423, Liberal Arts Bldg., *University of Arizona,* Tucson.

Arkansas: *Examination. Little Rock, Oct. 1-2. Sec., Mr. Louis E.* Gebauer, 1002 *Donaghey Bldg.,* Little Rock.

Colorado: *Examination.* Denver, Sept. 10-11. Sec., Dr. Esther B. Starks, 1459 Ogden St., Denver 18.

Connecticut: New Haven, Oct. 11. Address: State Board of Healing Arts, 110 Whitney Ave., New Haven 10.

District of Columbia: *Examination.* Washington, Oct. 20-21. Sec., Dr. Daniel L. Seckinger, 4130 E. Municipal Bldg., Washington.

Florida: *Examination.* Gainesville, Oct. 25. Sec., Mr. M. W. Emmel, Box 340, University of Florida, Gainesville.

Michigan: *Examination.* Detroit and Ann Arbor, Oct. 10-11. Sec., Mrs. Anne Baker, 423 W. Michigan Ave., Lansing.

Minnesota: *Examination.* Minneapolis, Oct. 7-8. Sec., Mr. Raymond N. Bieter, 105 Millard Hall, University of Minnesota, Minneapolis.

Nebraska: *Examination.* Omaha, Oct. 7-8. Director, *Bureau of Examining* Boards, Mr. Husted K. Watson, 1009 State Capitol Bldg., Lincoln.

New Mexico: *Examination.* Santa Fe, Sept. 21. Sec., Mrs. Marguerite Cantrell, Box 1592, Santa Fe.

Oklahoma: *Examination.* Oklahoma City, Sept. 13. Sec., Dr. Clinton Gallaher, 813 Braniff Bldg., *Oklahoma City.*

Oregon: *Examination.* Portland, Sept. 6 and Dec. 6. Sec., Dr. Charles D. Byrne, State Board of Higher Education, University of Oregon, Eugene.

South Dakota: *Examination.* Vermillion, *Dec. 5-6. Sec.,* Dr. Gregg M. Evans, 310 E. 15th St., *Yankton.*

Tennessee: *Examination.* Memphis, Sept. 24-25. Sec., Dr. O. W. Hyman, 874 Union Ave., Memphis 3.

Texas: *Examination.* Austin, October. Sec., Brother Raphael Wilson, 407 Perry-Brooks Bldg., Austin.

Wisconsin: *Examination.* Madison, *Sept. 13, Milwaukee,* Dec. 6. Sec., Mr. W. H. Barber, 621 Ransom St., *Ripon.*

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* Basic Science Certificate required.

DEATHS

Greene, Edgar Hill ⊕ Atlanta, Ga.; born in Shellman, Ga., Jan. 22, 1889; Emory University School of Medicine, Atlanta, 1915; assistant professor of clinical gynecology at his alma mater; past president of the Medical Association of Georgia; past president and, from 1932 to 1939, a member of the board of trustees of the Fulton County Medical Society; one of the founders of the Southeastern Surgical Congress; member of the Southern Medical Association; fellow of the American College of Surgeons; for many years president of the Atlanta Graduate Chapter of Phi Delta Theta; member and past president of the state board of medical examiners; served in France with the American Expeditionary Forces during World War I, for which service he was decorated by the French government; a major in the Medical Officers Reserve Corps and the Georgia National Guard; retired from the service as a lieutenant colonel; as chairman of the American Preparedness Committee of the Medical Association of Georgia in 1940 headed the procurement and assignment service for physicians, dentists, and veterinarians, and later chairman of a similar committee for the Fourth Service Command; for his service was granted a presidential citation; for many years a trustee of the Good Samaritan Clinic; on the staffs of the Crawford W. Long, Grady Memorial, Emory University, Georgia Baptist, St. Joseph's, and Piedmont hospitals; died May 30, aged 63, of carcinomatosis.

Ringer, Paul Henry ⊕ New York; born in New York Nov. 6, 1881; Columbia University College of Physicians and Surgeons, New York, 1904; past president, vice-president and secretary of the Buncombe County (N. C.) Medical Society; past president of the Medical Society of the State of North Carolina, Southern Tuberculosis Conference, and the Southern Medical Association; formerly vice-president of the National Tuberculosis Association; member of the American College of Chest Physicians and past president of its Southern chapter, American Trudeau Society, Medical Society of the State of North Carolina, and the American Clinical and Climatological Association; fellow of the American College of Physicians; past president of the North Carolina State Board of Medical Examiners; specialist certified by the American Board of Internal Medicine; served overseas during World War I; formerly practiced in Asheville, N. C., where he was affiliated with the Mission Hospital; author of "Clinical Medicine for Nurses"; served on the editorial board of the *North Carolina Medical Journal,* and was its chairman from 1944 to 1950; died May 9, aged 71.

Lavan, John Leonard ⊕ Eloise, Mich.; born in Grand Rapids, Mich., Oct. 28, 1890; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1914; formerly major league baseball player; an officer in the Navy during World Wars I and II; formerly secretary and member of the advisory council of the Michigan Department of Health, and vice-president of the Michigan Public Health Association; at one time epidemiologist with the Kansas City, Mo., health department; served as health commissioner of Toledo and director of health service in Toledo schools, district health officer for the New York City Health Department, director of the department of public health and welfare of Kalamazoo, and commissioner of public health of Grand Rapids; formerly director of scientific research for the National Foundation for Infantile Paralysis; director of social hygiene for the Wayne County Health Department; died in Harper Hospital, Detroit, May 29, aged 61.

Scobee, Richard Gordon ⊕ St. Louis; born in Sherman, Texas, Nov. 23, 1914; University of Texas School of Medicine, Galveston, 1939; assistant professor of ophthalmology at Washington University School of Medicine; member of the American Academy of Ophthalmology and Otolaryngology and the Association for Research of Ophthalmology; fellow of the American College of Surgeons; specialist certified by the American Board of Ophthalmology; member of the Advisory Committee on Ophthalmic Devices, Council on Physical Medicine and Rehabilitation, American Medical Association; affiliated with St. Louis City, Barnes, Washington University, and St. Louis Children's hospitals; consulting ophthalmologist, Percy Jones General Hospital in Battle Creek, Mich.; served during World War II; died June 22, aged 37.

Williamson, Carl Sneed ⊕ Arroyo Grande, Calif.; born in Huntsdale, Mo., Dec. 11, 1896; University of Pennsylvania School of Medicine, Philadelphia, 1920; formerly professor of surgery at University of Arkansas School of Medicine, Little Rock; specialist certified by the American Board of Surgery; member of the Western Surgical Association and the Alumni Association of the Mayo Foundation; fellow of the American College of Surgeons; at one time fellow in surgery at Mayo Foundation in Rochester, Minn.; served during World War II; formerly affiliated with Atchison, Topeka and Santa Fe Railway Hospital and Veterans Administration Hospital in Albuquerque, N. M.; died June 2, aged 55.

Thomas, Harry George ⊕ Pawling, N. Y.; born in Hollansburg, Ohio, May 30, 1869; Medical Department of Omaha University, 1896; served overseas with the American Expeditionary Forces during World War I; civilian surgeon at the Navy's New Jersey munitions depot during World War II; at one time held a teaching post at the American University at Beirut in Syria; for many years practiced in Asbury Park, N. J., where he was member and president of the board of education; father of Lowell Thomas, well-known author and radio news commentator; died in New York May 6, aged 82, of fracture of the hip following a fall.

Kice, Luther Holden ⊕ Garden City, N. Y.; Jefferson Medical College of Philadelphia, 1908; an officer in the medical corps of the regular army; served during World War I; formerly medical examiner for the Civil Aeronautics Administration; member of the American Academy of Dermatology and Syphilology; a charter member of the Aero Medical Association; affiliated with Nassau Hospital in Mineola, Central Islip (N. Y.) State Hospital, Meadowbrook Hospital in Hempstead, and Mercy Hospital in Rockville Centre; died May 31, aged 67, of cardiac decompensation and cerebral vascular accident.

Adler, Henry I., Chicago; Maryland Medical College, Baltimore, 1904; found dead May 24, aged 72.

Allen, Leroy Kimble ⊕ Roseville, Mich.; Detroit College of Medicine and Surgery, 1921; health officer of the village of Roseville; on the staff of the Saratoga General Hospital in Detroit, where he died May 21, aged 64, of pneumothorax.

Allen, William Gray ⊕ Evanston, Ill.; Northwestern University Medical School, Chicago, 1896; an Associate Fellow of the American Medical Association; at one time professor of anatomy at Chicago College of Medicine and Surgery; formerly affiliated with Illinois Masonic and Henrotin hospitals in Chicago and St. Francis Hospital; died June 20, aged 78, of Hodgkin's disease.

Angle, George Burton, Waymart, Pa.; Jefferson Medical College of Philadelphia, 1901; at one time a member of the bureau of health of the Philippine Islands; formerly on the faculty of his alma mater; formerly affiliated with Fairview State Hospital; died in the Wayne County Memorial Hospital in Honesdale May 27, aged 80.

Archambeau, Lester Milward, Farmville, Va.; Howard University College of Medicine, Washington, D. C., 1931; died March 16, aged 49.

Atkinson, Bolling Jones, Emporia, Va.; Medical College of Virginia, Richmond, 1923; died April 15, aged 52, of coronary thrombosis.

Ayers, Willis Thereau, Columbus, Ga.; Meharry Medical College, Nashville, Tenn., 1920; affiliated with City Hospital; died May 1, aged 62, of hypertension and cardiovascular disease.

⊕ Indicates Member of the American Medical Association.

Barnett, Abner Jones ⊕ Jackson, Miss.; University of Tennessee College of Medicine, Memphis, 1917; affiliated with Veterans Administration Hospital; died June 22, aged 60, of heart disease.

Beard, Robert Samuel, Huntsville, Ala.; Meharry Medical College, Nashville, Tenn., 1922; died March 1, aged 70, of coronary occlusion.

Berry, Gilbert LeRoy ⊕ Lawton, Okla.; State University of Iowa College of Medicine, Iowa City, 1923; served during World War I; affiliated with Southwestern Clinic Hospital; died May 27, aged 59, of coronary occlusion.

Black, Vinton Green, Noblesville, Ind.; Physio-Medical College of Indiana, Indianapolis, 1909; served during World War I; died May 28, aged 77.

Bohm, John Ernst, San Francisco; Cooper Medical College, San Francisco, 1912; affiliated with Southern Pacific and St. Francis hospitals; died May 9, aged 65, of carcinomatosis.

Burbank, Lester Warren ⊕ Cabot, Vt.; University of Vermont College of Medicine, Burlington, 1896; past president of the Vermont State Medical Society; died March 11, aged 85, of carcinoma of the stomach.

Burke, Matt Otey ⊕ Richmond, Va.; Medical Department of Tulane University of Louisiana, New Orleans, 1891; Associate Fellow of the American Medical Association; died April 23, aged 85, of coronary occlusion.

Burton, Claude Naylor ⊕ Asheville, N. C.; University of Cincinnati College of Medicine, 1937; member of the South Atlantic Association of Obstetricians and Gynecologists; affiliated with St. Joseph's Hospital and the Memorial Mission Hospital, where he died April 21, aged 44, of cerebral hemorrhage.

Campbell, Leo Kempf ⊕ Chicago; Rush Medical College, Chicago, 1927; clinical professor of medicine (Rush), University of Illinois College of Medicine; affiliated with Presbyterian Hospital, where he died June 13, aged 52, of heart disease.

Cooney, Thomas Edwin ⊕ New Preston, Conn.; Long Island College Hospital, Brooklyn, 1913; member of the Medical Society of the State of New York; died in Rocky Hill March 28, aged 60, of cerebral abscesses following infection of cerebral infarcts.

Coughlin, John Francis ⊕ San Marino, Calif.; Baltimore Medical College, 1908; fellow of the American College of Surgeons; member of the Idaho State Medical Association; for many years practiced in Twin Falls, Idaho, where he was affiliated with Twin Falls County General Hospital; died in St. Luke's Hospital, Pasadena, June 11, aged 68, of myocardial infarction.

Cramp, George Walter ⊕ Brooklyn; Long Island College Hospital, Brooklyn, 1923; specialist certified by the American Board of Radiology; member of the Radiological Society of North America and the American College of Radiology; fellow of the American College of Physicians; served during World War II; formerly on the staff of the Methodist Hospital and consulting roentgenologist, St. Luke's Hospital; Newburgh; died May 20, aged 56.

Dames, Alphonse Ferdinand, St. Louis; Washington University School of Medicine, St. Louis, 1906; formerly on the faculty of his alma mater; died in St. John's Hospital June 3, aged 75, of coronary thrombosis.

Daniels, Charles Edward, Melbourne, Fla.; Rush Medical College, Chicago, 1902; died March 29, aged 77, of coronary occlusion.

Edge, Ora Walter, Miami, Fla.; Hospital College of Medicine, Louisville, 1907; died recently, aged 71, of coronary occlusion and hypertension.

Edmundson, Frank, Clearwater, Fla.; Hahnemann Medical College and Hospital of Philadelphia, 1909; died March 27, aged 64, of coronary thrombosis.

Fernbach, Joseph George ⊕ Lancaster, N. Y.; University of Buffalo School of Medicine, 1915; died April 19, aged 61, of carcinoma of the colon.

Flynn, Charles Bernard, Yonkers, N. Y.; Georgetown University School of Medicine, Washington, D. C., 1903; formerly director of communicable diseases, city health bureau; on the courtesy staff, St. John's Hospital, Yonkers General Hospital, and St. Joseph's Hospital, where he died June 6, aged 72, of carcinoma of the left lung.

Flynn, George Clifford ⊕ Springfield, Mass.; Tufts College Medical School, Boston, 1924; physician for the city schools; on the staff of Mercy Hospital; served during World War I; died in New England Deaconess Hospital, Boston, May 9, aged 52, of cirrhosis of the liver, diabetes mellitus, and chronic glomerulonephritis.

Foster, Fred P. ⊕ Chicago; Chicago College of Medicine and Surgery, 1909; formerly professor of anatomy at Bennett Medical College and professor of physiology at Loyola University School of Medicine; died May 17, aged 93, of organic heart disease.

Freligh, Clarence Neil ⊕ Waucoma, Iowa; Northwestern University Medical School, Chicago, 1902; died in St. Joseph's Hospital, New Hampton, May 14, aged 74, of primary carcinoma of the liver.

Friedman, Alvin Burton ⊕ Cleveland; St. Louis University School of Medicine, 1942; specialist certified by the American Board of Pediatrics; served during World War II; died in Mount Sinai Hospital May 28, aged 36, of subarachnoid hemorrhage.

Gill, Arthur Francis, Banning, Calif.; University Medical College of Kansas City, Mo., 1901; served during World War I; died April 2, aged 74, of carcinoma of the pancreas.

Gille, Hugo ⊕ Jersey City, N. J.; Jefferson Medical College of Philadelphia, 1902; also a graduate in pharmacy; an Associate Fellow of the American Medical Association; formerly member of the city board of health; at one time on the staffs of the North Hudson Hospital in Weehawken, and Christ Hospital; died May 9, aged 74, of cardiac failure and hypertension.

Gipson, Arthur Albert, Clarksdale, Miss.; Meharry Medical College, Nashville, Tenn., 1915; died May 5, aged 63.

Gjvestvang, George Martin, Ola, Ark.; Kansas City (Mo.) College of Medicine and Surgery, 1917; died May 5, aged 62, of a heart attack.

Glaser, Robert Edward ⊕ Brookville, Ind.; St. Louis University School of Medicine, 1936; served during World War II; affiliated with Margaret Mary Hospital in Batesville and Fayette Memorial Hospital in Connersville; died May 19, aged 41, of accidental carbon monoxide poisoning.

Goodpastor, William H. ⊕ Pittsburgh; Western Pennsylvania Medical College, Pittsburgh, 1907; for many years president of the school board; school physician; affiliated with Presbyterian Hospital; died in Wooster, Ohio, May 17, aged 76, of cerebral hemorrhage.

Gramling, Arthur Burt, Attalla, Ala.; Maryland Medical College Baltimore, 1904; died March 23, aged 71, of bronchopneumonia and hypertensive heart disease.

Grant, Emmet E. ⊕ Seattle; the Hahnemann Medical Colleg and Hospital, Chicago, 1904; died in Virginia Mason Hospita May 12, aged 76.

Green, John Summerfield Jr. ⊕ Towson, Md.; Johns Hopkin University School of Medicine, Baltimore, 1918; past preside of the Baltimore County Medical Society; affiliated with Unic Memorial Hospital, where he died May 26, aged 59, of a cerebr vascular accident.

Greene, Edward Chace ⊕ Colonel, U. S. Army, retired, Wint Park, Fla.; University of Michigan Department of Medicine a Surgery, Ann Arbor, 1904; entered the medical corps of t U. S. Army as a major in September, 1920; member of the N England Society of Psychiatry; formerly medical director of t American Airlines, Inc.; died in the Walter Reed Gene Hospital, Washington, D. C., April 16, aged 76.

Gunderson, Anna Hussey, Lockport, Ill.; Chicago Medi School, 1923; died June 15, aged 69.

Hall, Joe E. ⊕ Maryville, Tenn.; Chattanooga Medical College, 1910; died April 4, aged 64, of a heart attack.

Halsted, Frederick Smith ⊕ Denver; St. Louis University School of Medicine, 1913; member of the American Academy of Ophthalmology and Otolaryngology; affiliated with St. Joseph's and Children's hospitals; died May 1, aged 63, of acute coronary occlusion.

Handel, William Harold, Buffalo; University of Buffalo School of Medicine, 1926; member of the Industrial Medical Association; served during World War I; county medical director; clinical assistant in surgery, Millard Fillmore Hospital, where he died May 9, aged 52, of glioblastoma multiforme.

Hargrove, Reuben Morgan ⊕ Houston, Texas; University of Texas School of Medicine, Galveston, 1912; at one time on the faculty of his alma mater and the University of Oklahoma, Norman; certified by the National Board of Medical Examiners; specialist certified by the American Board of Otolaryngology; served during World War I; fellow of the American College of Surgeons; affiliated with Memorial Hospital, St. Joseph Infirmary, Southern Pacific Hospital, and Hermann Hospital, where he died May 6, aged 68, of coronary occlusion.

Harper, Alexander W. ⊕ Flint, Mich.; Saginaw (Mich.) Valley Medical College, 1903; affiliated with Hurley Hospital and St. Joseph Hospital, where he died May 3, aged 74, of carcinoma of the liver.

Harper, Henry Winston Jr. ⊕ Fort Worth, Texas; Washington University School of Medicine, St. Louis, 1922; fellow of the American College of Surgeons; affiliated with All Saints, Harris, Pennsylvania, and St. Joseph's hospitals; died May 9, aged 55.

Heetland, Louis Herman ⊕ Sibley, Iowa; Michigan College of Medicine and Surgery, Detroit, 1895; died in Sheldon Memorial Hospital, Sheldon, April 20, aged 84, of diabetes mellitus and uremia.

Hendrickson, Berent ⊕ Ringwood, Ill.; Chicago College of Medicine and Surgery, 1911; died in Wonder Lake, April 17, aged 72, of myocardial degeneration.

Hilsabeck, Lester Clyde, Gretna, Neb.; John A. Creighton Medical College, Omaha, 1910; died in Omaha May 15, aged 70.

Holman, Henry Delavan ⊕ Mason City, Iowa; State University of Iowa College of Homeopathic Medicine, Iowa City, 1903; died May 21, aged 76.

Johns, Myles Wendel ⊕ Utica, N. Y.; New York Homeopathic Medical College and Hospital, New York, 1903; specialist certified by the American Board of Radiology; member of the Radiological Society of North America and the American College of Radiology; served during World War I; affiliated with Rose Hospital in Rome, Oneida County Hospital, and the Utica Memorial Hospital, where he died June 4, aged 73, of hypertension and cardiovascular disease.

Johnson, Allen Huggins ⊕ Hemingway, S. C.; Medical College of the State of South Carolina, Charleston, 1934; past president of the Williamsburg County Medical Society; medical superintendent and chief of the Johnson Memorial Hospital; president of the Pee Dee Telephone Company; director of the Anderson State Bank; died May 13, aged 41, of acute coronary occlusion.

Johnson, William Paul, Barnesville, Ohio; Starling-Ohio Medical College, Columbus, 1912; formerly county coroner; affiliated with Barnesville Hospital; died May 26, aged 63, of coronary thrombosis.

Kahn, Maximilian ⊕ Cleveland; Julius-Maximilians-Universität Medizinische Fakultät, Würzburg, Bavaria, Germany, 1896; a charter member of the Cleveland Academy of Medicine; served on the staff of Mount Sinai Hospital, where he died June 2, aged 79, of fractured hip.

Kaplan, Jacob Copel ⊕ Cincinnati; Tufts College Medical School, Boston, 1919; member of the Massachusetts Medical Society; formerly affiliated with the Veterans Administration Hospital in Lexington; died in Jewish Hospital May 12, aged 54, of myocardial infarction and coronary occlusion.

Kautz, Frank Alexander Sebastian, Cincinnati; Miami Medical College, Cincinnati, 1898; specialist certified by the American Board of Obstetrics and Gynecology; formerly member of the board of education; affiliated with Cincinnati General Hospital, Good Samaritan Hospital, and the Jewish Hospital, where he died May 12, aged 76, of coronary occlusion and diabetes mellitus.

Moore, Johnson McDowell, Penney Farms, Fla.; Atlanta Medical College, 1894; Bellevue Hospital Medical College, New York, 1895; died recently, aged 84, of cerebral arteriosclerosis.

Moore, William Raymond, Vernon, Texas; Vanderbilt University School of Medicine, Nashville, Tenn., 1914; died May 22, aged 64, of cirrhosis of the liver.

Morgenroth, Frank Charles ⊕ Los Angeles; Chicago College of Medicine and Surgery, 1916; died May 12, aged 63, of cerebral hemorrhage.

Morse, Waldron Lewis ⊕ Springvale, Maine; Yale University School of Medicine, New Haven, Conn., 1933; certified by the National Board of Medical Examiners; past president of the York County Medical Society; a lieutenant in the U. S. Army for three years; on the staff of the Henrietta D. Goodall Memorial Hospital in Sanford; died May 8, aged 43.

Mowry, Jesse E. ⊕ Providence, R. I.; Harvard Medical School, Boston, 1891; member of the House of Delegates, 1916-1917, American Medical Association, of which he had been an Associate Fellow; past president of the Providence Medical Association; for many years treasurer of the Rhode Island Medical Society; on consulting staff, Rhode Island Hospital, where he died May 3, aged 87.

Moyer, Donald Grosch ⊕ Wyomissing, Pa.; Jefferson Medical College of Philadelphia, 1917; served during World Wars I and II; for many years secretary of the board of health; chief of pediatrics, St. Joseph's Hospital in Reading; died May 6, aged 59, of myocardial infarction.

Nathanson, Morris Henry ⊕ Los Angeles; University of Minnesota Medical School, Minneapolis, 1919; associate clinical professor of medicine, University of Southern California School of Medicine; at one time assistant professor of medicine at his alma mater; specialist certified by the American Board of Internal Medicine; fellow of the American College of Physicians; affiliated with Cedars of Lebanon and Los Angeles County hospitals; died April 24, aged 59, of coronary thrombosis.

Oldani, John P., Detroit; Detroit College of Medicine, 1908; died recently, aged 67, of bilateral bronchopneumonia and arteriosclerotic heart disease.

Parnell, Carter C. ⊕ Sledge, Miss.; Missouri Medical College, St. Louis, 1899; died May 18, aged 75, of a heart attack.

Parrella, Dominick ⊕ Brooklyn; Eclectic Medical College, Cincinnati, 1922; affiliated with Caledonian Hospital; died June 6, aged 57.

Payne, Mosby Hale ⊕ New York; Jefferson Medical College of Philadelphia, 1918; died recently, aged 60.

Pernoud, Flavius Gentry ⊕ St. Louis; Washington University School of Medicine, St. Louis, 1907; served during Mexican border campaign; senior instructor in surgery at St. Louis University School of Medicine; formerly instructor in surgery at his alma mater; on the staffs of the Alexian Brothers' Hospital, St. Mary's Group of Hospitals, Lutheran Hospital, and St. Anthony's Hospital, where he died June 9, aged 67, of coronary occlusion.

Person, Hector McLean, Goldsboro, N. C.; North Carolina Medical College, Charlotte, 1913; served during World War I; died in the Wayne Memorial Hospital June 9, aged 68.

Pirosh, Sigmar, Los Angeles; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1905; died April 20, aged 60, of coronary occlusion.

Purves, George Kern, Wichita, Kan.; Northwestern University Medical School, Chicago, 1900; served during World War I; on the staff of Veterans Administration Hospital, where he died June 7, aged 77, of carcinoma of the prostate.

GOVERNMENT SERVICES

ARMY

Seven New Permanent Army Hospitals.—Construction of the first of seven new permanent hospitals planned for Army posts in this country will begin early next year. The hospitals represent the first step in the Army's program for transferring patients from wooden, cantonment type structures built during World War II to modern multistory structures. Hospitals to be constructed at Fort Benning, Ga., Fort Bragg, N. C., Fort Knox, Ky., and Fort Riley, Kan., are for 500 beds on a 1,000 bed chassis. A new installation at Fort Belvoir, Va., will provide 250 beds initially, expansible to 500; while Fort Monmouth, N. J., will receive a 200 bed facility constructed on a 300 bed chassis. The largest structure will be erected at Fort Dix, N. J., and will furnish 750 beds, expansible to 1,000 beds. The Surgeon General stated that the new facilities incorporate the findings of extensive research in hospital management conducted by the Army Medical Service for the past three years at Valley Forge Army Hospital, Phoenixville, Pa., and at other major hospitals. These new features are expected to economize in the use of nurses, ward technicians, and other scarce personnel. Wards for the critically ill, operating rooms, and clinic facilities will be air-conditioned. The operating rooms will also be equipped to eliminate the danger of anesthetic explosions. All units are expected to be completed in from two to three years.

Dr. Bayne-Jones Named Research Director.—The Department of the Army announces that Dr. Stanhope Bayne-Jones has assumed his duties as civilian technical director of Army Medical Research, and will serve in the office of the Army Surgeon General, succeeding Dr. Francis G. Blake, who died last January. Dr. Bayne-Jones will be responsible for insuring close coordination between the Army's Medical Research effort and similar programs sponsored by the other armed forces and civilian federal agencies.

Since 1947, Dr. Bayne-Jones has been president of the Joint Administration Board of New York Hospital-Cornell Medical Center in New York City. During World War II, he served in the Surgeon General's Office as deputy chief of the preventive medicine service, and as administrator of the Army Epidemiological Board and director of the U. S. Typhus Commission. He was born in New Orleans, received the Bachelor of Arts degree from Yale University, and Doctor of Medicine degree from Johns Hopkins University. During World War I, he served in France and Germany with the British and American expeditionary forces. Among his decorations are the Distinguished Service Medal, the Silver Star with two oak leaf clusters, the Army Commendation Ribbon, and the U. S. Typhus Commission Medal. A former chairman of the Division of Medical Sciences of the National Research Council, Dr. Bayne-Jones has also been president of both the American Association of Immunologists and the Society of American Bacteriologists.

NAVY

Course in Radioactive Isotopes.—About 100 officers of the Navy Medical Department, Air Force, Royal Canadian Army and Navy, U. S. Public Health Service, and Civilian Scientists attended a five day course of instruction, which convened Aug. 4, in medical aspects of special weapons and radioactive isotopes at the Naval Medical School, Bethesda, Md. The training included lectures and demonstrations in nuclear physics; medical problems of radiological safety; operation of blood banks in time of major disaster; radiological defense, disaster planning, and medical aspects of chemical and special weapon warfare; and the medical aspects of civil defense.

Some of the lecturers were Dr. Howard Andrews, Chief, Section on Nuclear Radiation Biology, National Institute of Health; R. E. Lapp, Nuclear Science Service, and Carl Z. Morgan, Director, Health Physics Division, Oak Ridge National Laboratory; Lawrence W. Tuttle, Associate in Radiation Biology, University of Rochester School of Medicine and Dentistry; Herbert Friedman, Head of Electron Optic Branch, Naval Research Laboratory; Murray M. Copeland, Director of Oncology, Georgetown University Medical Center; S. T. Gibson, Associate Medical Director, National Blood Program, American Red Cross; and Douglas W. Overton, International Relations Office, Department of State. The course is conducted primarily for the benefit of inactive reserve medical and dental officers of the Navy and key civilian scientists. Certificates of attendance are issued to each person attending the course. These professional men are qualified to train others and to organize civil defense programs to reduce the destruction caused by this type of warfare.

DEPARTMENT OF DEFENSE

More Physicians to Be Inducted During September.—The Department of Defense has requested the Selective Service System to deliver to armed forces induction stations 355 doctors of medicine and 90 doctors of dentistry during the month of September. The armed forces will assign 180 physicians to the Army and 175 to the Air Force. All of the dentists will be assigned to the Air Force. The September call for physicians and dentists brings to a total of 1,522 the number of physicians and to 650 the number of dentists requested since July, 1951.

PUBLIC HEALTH SERVICE

Training for Foreign Health Workers.—The first health personnel from Iraq, Afghanistan, and Israel to come to the United States for intensive training programs arranged by the Division of International Health of the Public Health Service arrived in June. In addition trainees from six other countries arrived to begin their studies, which are sponsored by federal technical aid programs to foreign countries.

Aid for Employees with Emotional Disorders.—A program to help persons with emotional disorders to get and hold self-supporting jobs has been undertaken by the National Institute of Mental Health and the Office of Vocational Rehabilitation of the Federal Security Agency. As a first step, a two week training course for counselors from state vocational rehabilitation agencies has been begun at San Jose State College, San Jose, Calif., on July 21. Twenty counselors from 12 states are taking the course. At least two additional courses are planned, one at Columbia University, New York, and the other at the Menninger Foundation in Topeka, Kan. The courses are aided by funds made available through NIMH grants to these training centers. Dr. Richard H. Williams of the institute staff will participate in the three courses. Ruth Tiedman, professor of psychology at San Jose State, will be the director of the first course, with Dr. Maurice Grossman, of the Palo Alto Veterans Administration Hospital, and Dr. Henry C. Schumacher, mental health consultant for the Public Health Service at San Francisco, as instructors.

Objectives of the San Jose program are to provide counselors with information regarding human behavior and personality disorders that will help improve vocational rehabilitation programs; to develop more favorable attitudes toward the counseling, training, and placement of clients with neuropsychiatric disabilities; and to promote the use of community resources and consultative services in planning and promoting vocational rehabilitation. Counselors will be given an opportunity to observe and practice, under supervision, the skills and techniques needed in the vocational rehabilitation of emotionally disturbed clients. States sending counselors to the San Jose course are New Mexico, Louisiana, Texas, Idaho, Utah, Washington, Oregon, Colorado, Montana, California, Arizona, and Wyoming.

FOREIGN LETTERS

ARGENTINA

Blood Transfusion Services.—Many hospitals and maternity institutions in Buenos Aires have their own departments for blood transfusion and determination of blood groups. There is an important well-organized central Municipal Service of Blood Transfusion and Plasmotherapy, which is directed by Dr. E. Sammartino. It cooperates with organizations in 11 municipal hospitals of Buenos Aires. The Municipal School of Transfusion for Physicians and Nurses and the Association of Voluntary Blood Donors is also under the direction of Dr. Sammartino. In 1951 the National Institute of Hemotherapy, under the Ministry of Public Health, was responsible for 9,737 blood transfusions, 45,174 blood group determinations, and the production of 95,486 liters of plasma. The Army Hospital also provides important services. Whole blood has been collected mostly in Buenos Aires through an intense program of propaganda and many voluntary blood donors connected with the Institute of Hemotherapy. Plasma is used less than blood, and no other blood substitutes are needed owing to the numerous contributions of whole blood from voluntary sources.

Public Health Achievements.—At the end of the presidential period 1945-1951 the Minister of Public Health published statistical information on progress seen in the last six years. The number of beds in hospital wards increased from 66,000 to 114,609 (73% increase). The specialized institutes increased from 5 to 55. The annual expenses of the Ministry increased from 65 to 276 million pesos. The general mortality rate decreased from 14 to 8.6 per hundred inhabitants. Tuberculosis mortality decreased from 73 to 48 per hundred thousand inhabitants (a 34% decrease). The cases of malaria decreased after the DDT prophylactic campaign from 86,076 to 1,177. The cases of venereal disease decreased 58%. In leprosy 6,032 subjects affected are under sanitary control and treatment. Dr. Ramón Carrillo has been reappointed Minister of Public Health for the second presidential period of Gen. J. D. Perón (1952-1958).

ITALY

Physiopathology of Pulmonary Arterial Circulation.—Professor Condorelli, chairman of the medical clinics at the University of Catania, who is succeeding 70-year-old Prof. Nicola Pende to the chair of medical pathology at the University of Rome, reported in his opening address his studies on pulmonary arterial circulation. The number of persons with pulmonary hypertension who seek the general practitioner's help increases every day. Often, because these patients feel a sensation of precordial distress after strenuous physical exercise, they are confused with patients who have angina pectoris. Administration of drugs indicated for patients with angina pectoris, such as amyl nitrite by inhalation, may aggravate the symptoms. Aside from the fact that patients with pulmonary hypertension present a clinical picture different from that of patients with arterial hypertension of the greater circulation, the drugs that are useful for the latter, such as nicotinic acid, are harmful for persons with hypertension of the pulmonary circulation.

To say that a person with hypertension of the "lesser circulation" or pulmonary circulation does not have a genuine disease of the vessels but has chronic bronchitis, asthma, bronchiectasis, or lesions of the mitral valve is not a sufficient explanation for the fact that drugs that are effective on a pronounced arterial pressure of the systemic circulation do not have any effect on the pulmonary circulation. There is also in the smaller circulation hypertension, i. e., hypertension of the pulmonary artery caused by the effort that venous blood must overcome, through the pulmonary artery and its ramifications, to win the resistance of a lung that, because of previous and present respiratory illness, is not too elastic. We must admit, therefore, that there is no interdependency between the systemic and the pulmonary circulation. In fact, many persons in whom pressure is high when measured with the oscillometer show a normal pulmonary pressure if measured with Hamilton's tensiometer. On the other hand, many patients with pulmonary hypertension have normal or even below normal arterial pressure.

To confirm the existence of this autonomy of functions between the greater and smaller circulation there was need of an irrefutable proof of the different physiological conditions in the two circulatory systems. Condorelli and his co-workers Francaviglia and Truchetti reported on this subject. By means of an instrument that Condorelli made, he proved that, even though stimulation (compression) of the reflexogenic zone of the carotid sinus (Pagano's reflexogenic zone) causes a pronounced drop of the pressure of the systemic circulation, it does not change the pressure of the pulmonary artery. A certain drop of the pulmonary circulation is noticeable only as a consequence of the accompanying bradycardia. By stimulating the chemoreceptors of the greater circulation with nitrites (intravenous injections of sodium nitrite or inhalation of amyl nitrite) and causing an abrupt drop in the tone of the systemic circulation (hypotension), the pulmonary tensiogram shows only insignificant changes, which, at any rate, are attributable to the induced tachycardia. It is like saying that the stimulations that are received by the chemoreceptor centers of the greater circulation are spread only to the vasomotor fibers of the greater circulation and not to the ramifications of the lesser circulation or pulmonary circulation.

Condorelli showed that nicotinic acid causes capillary and precapillary hyperemia, acceleration of the velocity of circulation with increased use of oxygen, transitory increase of arterial pressure followed by a decrease, and the subsidence of eventual angiospasms because of the decreased pressure. From this rises the therapeutic indication of nicotinic acid against angiospastic syndromes caused by hemicrania and cerebral spasms with paretic manifestations. But on the pulmonary circulation, as Condorelli demonstrated, nicotinic acid has the opposite effect, in that it causes a pronounced increase of the pulmonary arterial pressure. The same is true of intravenous administration of aminophylline. This drug, while causing an abrupt decrease of the arterial pressure of the greater circulation, does not have any hypotensive effect on the smaller circulation.

On the contrary, epinephrine and arterenol increase the pressure of the pulmonary circulation. In this way hypertension due to pheochromocytoma proves to be much different from common arterial hypertension. While in the common hypertension of the greater circulation there is no involvement of the pulmonary pressure, in hypertension caused by pheochromocytoma there is simultaneous pressure increase of the two circulations: the smaller and the greater. This may be due to the susceptibility that the pulmonary artery has for the epinephrine and arterenol, which are abundantly produced by the adrenal gland affected with pheochromocytoma. This susceptibility to epinephrine and arterenol suggests the presence of sympathetic fibers in the nervous network of the pulmonary artery.

Acetylcholine and analogous preparations that have great effect on the vagus nerve (on dilatation of the arterial vessels and drop in pressure) do not have any effect on pulmonary hypertension. Moreover, if the effect of acetylcholine is potentiated with a previous injection of neostigmine, a combination that in patients with arterial hypertension brings about a real drop of pressure, there is no effect on the pulmonary pressure. This explains the uselessness of drugs that are believed to have an hypotensive effect on pulmonary hypertension. Finally, if, in a dog that is breathing in a closed circuit, hypertension is caused to rise gradually to a level of 20 mm. Hg (about 26 cm. of water), no "blocking" phenomenon is seen. While in the in-

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The items in these letters are contributed by regular correspondents in the various foreign countries.

spirating pulmonary veins the pressure drops to below zero, the pressure of the pulmonary artery increases very slightly: the systolic from 30-32 to 40-42, the diastolic from 10-20 to 30. The arterial pressure of the greater circulation, after a mild initial deflection, returns to its initial levels. If, however, the nervous synapses are blocked with curare and the pulmonary vessels are, therefore, deprived of the possibilities of reacting with their own autonomous mechanisms, even a respiratory hypertension of only 5 mm. Hg in addition to a very mild increase of the pulmonary tension causes, by means of the compression exerted on the capillaries and the pulmonary vein, a true "blocking" that manifests itself with a drop in the arterial pressure of the greater circulation.

In other words, the smaller circulation has its own neuroregulation system, independent from that of the greater circulation. Its autonomy, the study of which will open new therapeutic ways, is demonstrated by the varying behavior towards drugs that are used to check arterial hypertension.

LONDON

Agreement on Wage Plan.—At the Conference of Local Medical Committees, the delegates who represent the 20,000 general practitioners in the National Health Service endorsed a revised plan for distributing the enlarged central pool from which physicians are paid. The plan was formulated by a Working Party composed of British Medical Association and Ministry of Health representatives. The Working Party was set up after the adjudication award of Justice Danckwerts last March, under which four years' arrears of nearly £27 million are owed to the physicians. The award was subject to a satisfactory agreement being reached on distribution of the pool.

The conference will also assist any group of practitioners that does not benefit, when they expected to do so under the Working Party's terms of reference, by internal adjustment. These recommendations will now be referred to the Minister of Health, who will take steps to inform Parliament before the supplementary estimate is submitted in July. If this estimate is approved, it should be possible for payment of the arrears to start before the end of the year.

The terms of reference of the Working Party were as follows: "To secure an equitable distribution of the central pool based upon the recommendations of the Spens Committee, the object being to enable the best possible medical service to be available to the public, and to safeguard the standard of Medical Service by discouraging unduly large lists; at the same time, to bring about a relative improvement in the position of those practitioners least favorably placed under the present plan of distribution, to make it easier for new doctors to enter practice, and to stimulate group practice."

The main recommendations of the Working Party are as follows: (1) reduction in the maximum number of a practitioner's patients (*a*) from 4,000 to 3,500 for a private practitioner, (*b*) from 5,000 to 4,500 for a member of a partnership, provided the average for the partnership is not above 4,000, and (*c*) from 2,400 to 2,000 for each permanent assistant employed; (2) practice areas to be reclassified in three categories: restricted areas, doubtful areas, and "designated" areas, in which new entrants to the service would be eligible to apply for initial practice allowance; (3) new recommendations for the capitation fee including: (*a*) a basic fee of 17 s. (about $2.40) per patient, (*b*) an additional "loading" fee of 10 s. ($1.40) for every patient within the patient load range of 501 to 1,500, and (*c*) a final settlement, as a percentage of previous payments under *a* and *b* above, raising the income per patient from 17 s. in a load of 500 patients to a maximum of 23⅔ s. ($3.31) in a patient load of 1,500, and lowering the income as the load grows to its maximum of 4,000, to nearly £1 ($2.76); (4) a system whereby partnerships can apply to executive councils to be paid in what both partners consider to be the most financially advantageous division of the patients between them, thereby stimulating the formation of partnerships, and (5) a sum of money (about £100,000, or $280,000) to be set aside to stimulate the formation of group practices.

Infectious Hepatitis.—The *British Medical Journal* reported the Harveian Lecture by Sir John McNee, Negius Professor of Medicine in Glasgow University, in which he discussed infectious hepatitis. This disease was formerly called catarrhal jaundice, but now, according to the newer nomenclature, it is called viral hepatitis. In recent years two other forms of hepatitis have become recognized, namely homologous serum jaundice and syringe-transmitted hepatitis.

Infectious hepatitis is known best in its sporadic form but has been observed to spread widely in pandemic form. Usually regarded as benign, it can be highly malignant when epidemic, as revealed by its occurrence in Scandinavia, where there were severe and fatal infections in adults as well as children. It can be compared with spirochetal jaundice (Weil's disease) a rat-borne infection due to a spirochete and well known in military campaigns. During World War I there was no outbreak of infectious hepatitis in France. In World War II, the two types of infection, namely serum jaundice and the syringe-transmitted variety, became enormously important, both in the armed forces and among war workers at home. The pandemic was at its height between 1942 and 1944 and then declined. American soldiers were inoculated with a yellow fever vaccine containing human serum, and, of 28,505 in whom jaundice developed, 62 died. The incubation period is about three months. The virus of acute infectious hepatitis is probably most commonly transmitted by the feces and often by a contaminated water supply. The virus of the homologous serum variety depends almost exclusively on direct injection of blood or serum for transmission.

The two viruses would appear to be distinct because, although they cause diseases that are clinically identical, they have different incubation periods, but McNee gives evidence suggesting that one is a variant of the other. The findings of the Medical Research Council present strong evidence for considering the two viruses as distinct. First, the incubation period of the naturally acquired disease is between 20 and 35 days, but that of the parenterally acquired variety falls between 60 and 120 days. Although the clinical pictures and the histological condition of the liver in the two diseases are similar, there is evidence that a person can be immune to the homologous virus and not to the heterologous virus. In addition, there is a well-established difference in the routes by which artificial transmission can be achieved. Whereas in homologous serum jaundice successful transmission has been obtained only by parenteral administration of serum, infectious hepatitis can also be transmitted by spraying the nasopharynx with infected serum. The virus causing infectious hepatitis has also been conclusively demonstrated in the feces, whereas no evidence of fecal virus has been obtained in patients with homologous serum jaundice. Finally, although in the majority of cases the clinical picture of the two conditions is indistinguishable, many observers have noted a tendency for the homologous serum variety to be severer and more protracted.

The infectious period of both types of the disease appears to be remarkably short, but experimental proof of this is lacking. In an induced case of infectious hepatitis, virus has been detected 3 days before appearance of the first symptom and as late as 8 days after the onset of symptoms, but not 31 days after the onset of symptoms. A chronic carrier state with infectious hepatitis virus in the blood stream does not seem to occur. The history of homologous serum jaundice also shows quite clearly that donors of icterogenic plasma exist in whom no association with patients with infectious hepatitis appears probable. The origin of the icterogenic agent in their blood is therefore completely obscure. There is no evidence at present for McNee's suggestion that the agent arises from ordinary infectious hepatitis virus by adaptation from prolonged sojourn in the human blood. Even if this were so, the stability shown by the two viruses today is such as to justify their classification as distinct varieties despite any possible common origin. In view of the fact that the virus is not yet under laboratory control and cannot be cultured, no means of immunization or other form of prevention is yet possible. Attempts are being made to neutralize the virus in serum by exposure to ultraviolet light, and human serum that contains antibodies can be administered in the form of immune globulin with some measure of success as a preventive after blood transfusion.

College of General Practice.—The establishment of a British College of General Practice was first discussed 100 years ago and has been suggested from time to time ever since, but agreement has never been reached on many important aspects of its objectives and organization. Now a steering committee is engaged in collecting and sifting evidence in preparation of a comprehensive report, which should lead to a plan for the implementation of the college. There would, at present, appear to be no serious opposition to the formation of such an organization.

PARIS

Death of Prof. Henri Hartmann.—Prof. Henri Hartmann, one of the greatest French surgeons, a contemporary of Cunéo and Lecène, died in an accident at the age of 90. He was a pupil of Terrier, an intern at the Bichat Hospital, and later an assistant to Professors Cunéo and Gosset. After Pasteur's memorable communication to the Academy of Medicine in 1878, he was the first to apply aseptic methods at the Bichat Hospital. In 1898, he presented to the Academy of Medicine the first two cases of lasting recovery following operation for cancer of the pylorus. In 1902, he reported a 30% survival rate for cancer of the stomach and a 40% rate for cancer of the rectum operated on according to his method. He also evolved a new method, known under his name, for operating on cancer of the rectosigmoid area through the abdominal route. He is the promoter of the sign of "the distended intestinal loop" in intestinal obstruction. He was a wonderful teacher, preferring clinical teaching at the patient's bedside. He published extensively on gastrointestinal surgery, surgery of genitourinary organs (translated into German), and on operative gynecology (translated into English). He also wrote nine volumes on surgical aspects of clinical anatomy, the main basis of the last one, published in 1951, being composed of records he kept of his surgical patients. He was vice-president of the French League Against Cancer and president of the Association for the Development of Medical Relations with Foreign Countries. In 1926, he presided over the International Congress of Surgery at Warsaw. He was a member of the National Academy of Medicine and Surgery and also a member of 30 foreign scientific societies.

Death of Dr. Jules Tinel.—Dr. Jules Tinel, who died recently at 72, was the son of a well-known professor of anatomy and surgeon. His maternal grandfather was also a professor and surgeon, and his son, too, is a physician. In 1906, Tinel became an intern successively in the services of Professors Siredey, Troisier, Dejerine (Salpétrière Hospital), Landouzy (Laennec Hospital), and Netter (Trousseau Hospital). In 1910, he presented his thesis on "Radiculitis and Tabes," which attracted widespread attention. In 1915-1919 he was chief of the Neuro-Psychiatry Center at Le Mans, and, in 1922, he was nominated Doctor of the Paris Hospitals. From 1936 to 1940, he was chief of the Laboratory of Beaujon, from which he was expelled by the Nazis who took possession of the hospital. He then became chief of the laboratory of the Boucicaut Hospital, which he reorganized. For years he has been general secretary to the Society for Psychiatry. During the occupation he took an active part in the resistance movement. His scientific writings are numerous and deal with neurology, psychiatry, and general pathology. One book on his personal psychiatric experiences, begun while he was imprisoned at Bordeaux, remains unfinished. He described the "tingling sensation," which bears his name, in the regeneration of nerves. He was an ingenious scientist and a first-class technician, who was able to prepare most of his own laboratory instruments. Notwithstanding a severe disease, handicapping him for years, he never ceased his scientific work.

SWITZERLAND

New Antituberculosis Drugs.—After the publication, by Selikoff and Robitzek of New York, of the first results obtained with the derivatives of isonicotinic acid hydrazide, Swiss physicians were surprised to learn that these products were already obtainable without medical prescription in drug stores. The first results obtained after experimentation and trials of isonicotinic acid hydrazide in Switzerland were reported at the meeting of the Swiss Association Against Tuberculosis, held on April 26 in Lausanne. B. Fust, A. Studer, and E. Boehni, of the Hoffmann-La Roche firm in Basel, have shown that the action of isonicotinic acid hydrazide (called "rimifon" in Switzerland) varies markedly, according to the strain of tubercle bacillus, and that the bovine strain is less sensitive than the human strain. J. Morin, head physician of a sanatorium in Leysin, reported the first observations of patients treated with "rimifon." Of 73 cases, 68 had cavitary pulmonary tuberculosis with demonstrable tubercle bacilli and 5 had tuberculous meningitis. Clinically, he observed a feeling of well-being in the patients, weight gain, and, sometimes, a fall in the sedimentation rate. Bacteriologically, 50% of the patients became negative for tubercle bacilli in four to eight weeks after the beginning of treatment. Radiologically, he noted that 20 patients improved, 1 became worse, and 45 remained the same. The patients with tuberculous meningitis improved, but it is necessary to wait some months to give judgment. Less optimistic were E. Tanner, J. Wanner, and Z. Ramer, from the sanatorium of Davos. They also noted improvement in the general health, but they did not observe improvement of the radiological image; chiefly, there was no obliteration of the cavities. On the other side, they frequently observed an exudative or toxic reaction, with an increase of expectoration, increased findings on auscultation, and an elevated sedimentation rate. There exists often a paradoxical relationship between the radiological and the bacteriological picture. The radiological picture shows a worsening when the bacteriological analysis suggests improvement. Tanner reported the case of a patient whose bacilli became a thousand times less sensitive to "rimifon" after only 15 days of treatment. Düggeli compared the efficiency of streptomycin combined with *p*-aminosalicylic acid and "rimifon." It seems that "rimifon" has a tendency to liquefy caseous material, which explains the appearance of cavities sometimes observed in the course of treatment. During the discussion that followed these four presentations, Schoch remarked on the efficacy of "rimifon" in the treatment of cutaneous tuberculosis, of erythema induratum, and in generalized sarcoidosis. Baroni studied the influence of "rimifon" on the metabolism and on the immunologic mechanisms, through the Middlebrook-Dubos agglutination reaction. The levels of agglutination vary rapidly as soon as therapy is undertaken and follow a characteristic curve. It is still impossible to have a definitive opinion of the value of "rimifon" and similar products. As yet, nothing justifies the abandonment of streptomycin and *p*-aminosalicylic acid. But this matter of the new antituberculous drugs has revealed the lack of good distribution of medical information. At present, physicians are informed through the general press and the advertisements of the pharmaceutical industry. The Trudeau Society promptly published an explanation and cautioned against overenthusiasm for "rimifon."

During this meeting, the controversial problem of pulmonary excision for tuberculosis was discussed. Professor Decker, head of the university surgical clinic of Lausanne, underlined the great differences in opinion regarding this problem throughout the world. He made a distinction between absolute and facultative indications. A destroyed lung, bronchial stenosis, and rapidly extending cavities can be considered as absolute indications. More and more, extrapleural pneumothorax and thoracoplasty should be abandoned. Professor Brunner, disciple of Sauerbruch, head of the surgical clinic of Zurich, recalled the success obtained with reversible collapsotherapy. One should thus first try an intrapleural or extrapleural pneumothorax. As for thoracoplasty, when it gives bad results, it should be replaced by excision. As much as possible, one should operate on patients who are still responsive to streptomycin, i. e., those who have received less than 60 gm. of this antibiotic. Most of the complications occur in patients whose tubercle bacilli have become resistant. During the discussion, M. Sommer, who had returned from the United States, said that he believes that the discrediting of pneumothorax and the great number of excisions performed in the United States comes from a poor knowledge of the application of pneumothorax.

CORRESPONDENCE

PRESENT STATUS OF CERTIFICATION IN AVIATION MEDICINE

To the Editor:—At the request of the Interim Board on Aviation Medicine and with the encouragement of the Advisory Board for Medical Specialties, the American Board of Preventive Medicine and Public Health has given consideration over the past 18 months to the possibility of association of preventive medicine and aviation medicine as a specialty certifying board. After numerous joint conferences of the members of the Interim Board in Aviation Medicine and the American Board of Preventive Medicine and Public Health, a proposed affiliation was developed. Under the terms of this proposal, the American Board of Preventive Medicine and Public Health would become the American Board of Preventive Medicine, sponsored, in addition to the sponsorship of the original board, by the appropriate professional associations in aviation medicine. Under this reorganization, it was proposed that candidates might be examined and certified in either (*a*) public health or (*b*) aviation medicine, the standards for examination and certification in public health to remain essentially as they are at present, with the standards for examination and certification in aviation medicine to be such as to meet the basic standards established by the Advisory Board for Medical Specialties. The proposed special training to be required in aviation medicine for eligibility for examination were as follows:

(1) successful completion of two academic years of graduate study in (*a*) the elements of preventive medicine, (*b*) the basic sciences applicable to aviation medicine, and (*c*) aviation medicine, at least one academic year of which study to be pursued in a school, college, or university accredited for the purpose of such graduate study by the Council on Medical Education and Hospitals of the American Medical Association or by the American Public Health Association;

(2) at least one year of residency training in aviation medicine approved by the Council on Medical Education and Hospitals and the American Board of Preventive Medicine; and

(3) a minimum of six years of academic training, residency training, or practical experience in aviation medicine subsequent to the intern year, which includes 1 and 2 above.

This proposal was reviewed by the Advisory Board for Medical Specialties at its meeting on Feb. 10, 1952. After hearing the proposal as presented by the representatives of the American Board of Preventive Medicine and by the Interim Board of Aviation Medicine, the Advisory Board for the Medical Specialties voted to disapprove the proposal at the present time for the following reasons:

1. There are at the present time only two training centers where acceptable training in aviation medicine can be obtained, both of which are military. There are no civilian training centers in aviation medicine at this time.

2. The specialized training requirements as previously outlined were not sufficiently defined nor was there adequate assurance of qualified supervision.

3. There was objection to the restriction of applicants for examination to members of a single medical group such as the Aero Medical Association.

After further discussion, it was moved, duly seconded, and unanimously voted that the Advisory Board for Medical Specialties approve in principle the proposal made by the American Board of Preventive Medicine and Public Health and the Interim Board of Aviation Medicine but that no certificates be issued in aviation medicine until such time as the previously outlined deficiencies in the training program in aviation medicine are adequately corrected and brought back to the Advisory Board for Medical Specialties for approval.

In accordance with this action, the American Board of Preventive Medicine and Public Health amended its articles of incorporation and by-laws, changing the name of that board to the American Board of Preventive Medicine and provision was made for additions to the board membership representing aviation medicine. Otis O. Benson Jr. was elected vice chairman of the Board of Preventive Medicine, representing aviation medicine, and Jan H. Tillisch was elected to membership on the board and was named chairman of the Standards and Examination Committee for Aviation Medicine. Since the February meeting, the Interim Board of Aviation Medicine has received proposals from three universities for the development of specialized training programs in aviation medicine. Plans and facilities for both military and civilian residency training programs have been developed. It is hoped that an application for approval of these residency training programs can be submitted to the Council on Medical Education and Hospitals of the American Medical Association in the near future.

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TETANUS FOLLOWING NASAL INJURY

To the Editor:—In The Journal for June 14, page 660, Dr. R. B. Tudor reported a case of tetanus resulting from a foreign body in the nose, and stated that "Only one other case was found in the literature in which tetanus unquestionably resulted from a nasal injury." Dr. Tudor's case reminded me of a patient who died from tetanus following a nasal injury whom I treated more than 25 years ago, a case which, even at this late date, is perhaps worth adding as a third case to the literature. My own recollection of the case has been reinforced by the mother's.

On Feb. 22, 1927, an 18-month-old male child forced a metal button mold into one nostril. The mother heard him cry and removed the piece of metal soon after it had been inserted. Slight bleeding showed that the mucous membrane had been injured. At 6 a. m. on Feb. 28, six days later, the mother found that he could not open his mouth when she offered him a bottle of milk. At 9 a. m. he had a convulsion. When it was over he seemed better for a few hours and was able to swallow a little food. About noon, however, he had another convulsion, followed by a series of convulsions. His muscles became increasingly rigid. After the second convulsion he was given a large dose of tetanus antitoxin at home and was then taken to the City Memorial Hospital in Winston-Salem, where he was given more antitoxin, together with symptomatic treatment. He rapidly grew worse, however, and died the next day, March 1.

Although cultures from the nose were not made, it is difficult to conceive of any organism other than Clostridium tetani that could have given rise to such symptoms. No evidence of any other infection was found, and there was not the slightest break in the skin anywhere else on the child's body. There seems no reason to doubt that this was a fatal case of tetanus and that the point of entry was the injured nasal mucosa.

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RIGHT RECTUS RIGIDITY

To the Editor:—I wish to take exception to a statement in The Journal of May 3. On page 29, under the heading "Right Rectus Rigidity," the author states, "We know that it is quite impossible to contract one rectus muscle without contracting the other." While on duty at Valley Forge General Hospital in 1945 in the plastic surgery department, I had frequent occasion to observe a patient who delighted in demonstrating his corporeal achievements. He could, at will, alternately contract and relax first one and then the other rectus abdominis so that the surface of his abdomen gave the impression of a revolving door.

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BUREAU OF INVESTIGATION

THE WESTERN MEDICAL CORPORATION MAIL-ORDER "TREATMENT" FOR EPILEPSY

Many physicians have wondered recently what is behind a new type of form that epileptic patients have requested them to sign. It calls for a statement by the physician that the holder thereof is epileptic. It is a new twist in the *modus operandi* of the Western Medical Corporation. It results from the appearance of a representative of the firm before a Senate subcommittee wherein he indicated that the firm would require henceforth a physician's diagnosis of epilepsy before it did business with epileptic "patients."

The concern's headquarters are in Chicago. It dispenses, by mail, phenobarbital and other anticonvulsant drugs, together with vitamins, minerals, and laxatives. The price is now $6 for a 30-day supply. Dr. Harry L. James is the "chief-of-staff."

All of this comes about because of a provision in proposed legislation that would have required personal supervision of a physician before prescription drugs could be shipped in interstate commerce. The provision was stricken from the Durham-Humphrey bill in the Senate Subcommittee on Health of the Committee on Labor and Public Welfare. Representatives of the firm complained to committee members that they would be put out of business and that their customers (they call them "patients") would suffer from having their source of medicines cut off. They got the "patients" to write and wire the subcommittee members that this was so. The result was satisfactory to the firm and confusing, apparently, to some senators. The only evidence received came from the firm and its deluded "patients." Some members of the committee apparently felt that a great service was being denied deserving epileptic voters.

Even after the provision was dropped, Senator Magnuson of Washington obtained unanimous consent to have entered in the Congressional Record [1] a letter from Senator James E. Murray of Montana to the Federal Security Administrator, Mr. Oscar Ewing. Mr. Ewing's department includes, among other agencies, the Food and Drug Administration. The senators feared, apparently, that the Food and Drug Administration was going to do, by regulation, what could not now be done directly by law.

Senator Murray's letter contained a most apt observation in the second and third paragraphs of his item numbered 1. He wrote:

> It is my understanding that if a physician regularly engages in actions actually inimical to the health of his patients, he would be violating both a cardinal principle of the code of medical ethics and the laws governing medical licensure in most of our States.
>
> If this is true and if the physicians wórking for the Western Medical Corp. are in fact endangering the health of their patients by prescribing phenobarbital on the basis of mailed questionnaires, then I must ask why is this regulation needed? Why have not those Western Medical Corp. physicians, who are members of the Illinois Medical Society, been charged with violating medical ethics and, if they are in fact endangering their patients by their practices, expelled from the Society? Surely their fellow doctors know better than we in Washington what practices are bad for patients and most certainly they have obligated themselves to take action against unethical practitioners. Furthermore, why haven't the licenses of all the physicians involved been revoked by the Illinois Board of Medical Examiners if, in fact, these physicians are engaging in practices harmful to their patients? Since neither of these actions has been taken, I can only conclude that the practices followed by the physicians of the Western Medical Corp. are not considered harmful by their fellow practitioners in Illinois. If they are not, then there is no reason for the proposed regulation.

Senator Murray and the members of the subcommittee should not be confused by the testimony of Robert J. Creevy, the vice-president of the firm, which implied that their "staff physicians" were not members of the Illinois State Medical Society by their own choice. The members of the staff, other than Dr. J. J. Hennessy, were well aware of their own ineligibility to membership. Nonetheless, Creevy testified that staff members had never been told that they could not be members of the state society. There is no indication that any of them ever made application.

Dr. Hennessy was then a member of the Chicago Medical Society. The society admitted him to membership when he was engaged in the practice of medicine in Blue Island, Ill., a suburb. He did not inform the society of his present employment. When his membership came up for discussion in the Senate subcommittee, however, it was promptly determined. Dr. Hennessy was expelled.

It is true, however, that no medical society has ever taken official action to urge institution of proceedings to have the licenses of Dr. James and his staff revoked. In good conscience, there is no question but that the senator has a point here. If local medical societies were more aggressive in matters of this kind, they could not later be accused of condoning bad or illegal practice of medicine.

It should be observed, however, that membership in a medical society is one thing and the license to practice medicine another. The state has a medical board that issues licenses and that has defined statutory powers in the matter of suspension and revocation. It is not a matter of record that the board of medical examiners of Illinois has ever questioned the license status of these men.

There is in Illinois (and possibly in Montana) provision for admittance to the practice of law on examination by a board appointed by the state supreme court. The admittance to practice on examination and showing of good moral character is done in the form of a motion to include on the roll of attorneys of the supreme court the name of the successful candidate. If in his career he commits an act that is unethical, he may be brought before a grievance committee of his local bar association. That grievance committee may revoke or suspend his right or "license" to practice law, subject only to appeal to the state supreme court.

It may be argued fairly, therefore, that if the local medical society was directly chargeable with responsibility for the ethical and legal conduct of those admitted to the practice of medicine, it could well be censured for its apparent condonation of what appears to be an unethical and illegal practice of medicine. Neither the Chicago Medical Society nor the Illinois State Medical Society has such powers. Their grievance committees function solely to hear and to determine complaints of unethical conduct of their members. The local medical grievance committee may, on hearing, and for cause, recommend to the society suspension or expulsion of a member. He may appeal to the state committee and to the American Medical Association's Judicial Council. His license to practice medicine is not affected.

Actually, the status of the Western Medical Corporation and its staff physicians can be determined on a state level.

This business has continued now for approximately 30 years. Originally it was the "Western Medical Association." In 1933, however, there was an incorporation of the business under the laws of Delaware. The corporation applied for and received a certificate of authority to transact business in Illinois. A document on file in the office of the Secretary of State of Illinois reads in part:

> Now therefore, I, Edward J. Hughes, Secretary of State of the State of Illinois, by virtue of the powers and duties vested in me by law, do hereby certify that the said Western Medical Corporation is from the date hereof duly authorized for a term of 99 years to transact the following business in this state, namely:
>
> To conduct and carry on a general mail order business. To acquire, establish, maintain and conduct in all its branches a general mercantile business and in connection therewith to carry on all or any of the trades, businesses, undertakings or functions that in any manner are subsidiary, incidental and contributory thereto.
>
> To compound, manufacture, buy, sell, import, export and generally deal in, either at wholesale or retail, in all kinds of drugs, druggist's, sundries, specialties and supplies and pharmaceutical, medical, surgical, hospital, invalid, optical and all other preparations, supplies, specialties, instruments, appliances scientific and other apparatus, machinery and proprietary articles of all kinds.
>
> To engage in any other manufacturing, mercantile and trading business.

At the hearing held last fall before the Subcommittee on Health of the Committee on Labor and Public Welfare of the Senate, Mr. Creevy testified that the firm at that time had 21,000 active "patients." Testimony was offered to the effect that the principal anticonvulsant dispensed is phenobarbital. In some instances bromide preparations are substituted. Dosages average 3 to 4½ grains (0.2 to 0.28 gm.) of phenobarbital per day, with a maximum of approximately 8 or 9 grains (0.51 to 0.57 gm.). Treatments are mailed on the basis of a 30 day supply. Psychological advice was also mentioned in testimony as a part of the package sent to patients. A uniform price of $5 (now $6) is charged, although in cases of need a reduction in price is made.

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[1] Congressional Record: Senate, May 5, 1952, pp. 4855-4856.

There was also testimony that the business employs 105 people and is conducted by mail. These 105 people therefore must handle an average of 1,000 "patients" a day, on the basis of a five day week. There are four doctors of medicine on the staff. Just how they could give any personal attention to 1,000 "patients" in a day's time rather taxes the imagination. Now there might well be a corps of clerical help. If the clerk thinks a case needs expert attention, one of the physicians might be called on to venture an opinion. It is possible that the clerk might be giving "psychological advice." Droplets of psychological wisdom for epileptics could be "canned."

Further testimony was intended to deny that the firm made any diagnoses. For instance, Mr. Creevy stated:

> If, upon receipt of our reply and additional literature, a prospect wishes to take advantage of our method of treatment, he then, in accordance with our instructions, completes a lengthy case history form which is concerned only with the individual patient. On this form, the questions presented, if answered properly, give our physicians a thorough knowledge of the type and severity of the prospective patient's attacks. Two additional questions of extreme importance which appear on this form are:
>
> (a) Has your case been diagnosed as epilepsy by a physician?
> (b) What is the name and address of this physician?
>
> If the answer to the diagnosis question is 'No', we do not accept the case for treatment under any circumstances, even though the prospective patient may give a vivid description of seizures which are most likely epileptic. We do not attempt at any time to diagnose by mail.

The "history" blank covers two sides of a "legal"-sized form. A box cautions the applicant as follows:

> Answer the questions on BOTH SIDES. These are the very same questions that our physicians would ask if you came here personally. Write the answers to the questions in the convenience of your home and send them to us.

Questions cover age, status, physical items, identity of physician who diagnosed the case as epilepsy, number and severity of attacks, identity of current medication, presence of other diseases named, whether head injury has occurred, symptoms during attacks, and special questions for women and children.

In the opinion of the Bureau and of those physicians to whom the Bureau sent copies of the "patient's case history" blank, there is no other possible reason for such a blank than to enable the corporation (or its staff) to diagnose. As one physician remarked, if this is not diagnosing, then only one question should be asked, "Do you have epilepsy?" A prominent neurologist observed as follows:

> The 'Patient's Case History' . . . is inadequate for diagnosing epilepsy. Of course, the questionnaire states that patients will not be accepted for treatment who have not been diagnosed as epilepsy by a physician. Such a diagnosis, however, may or may not be correct and no reputable physician would accept a patient for treatment without himself examining and questioning the patient. Furthermore, the treatment of epilepsy varies according to the type of the symptomatology and there is a definite attempt in the questions to diagnose the type of attack. This corporation could not adequately treat patients with epilepsy.

Another wrote:

> . . . It is, of course, obvious from this material that patients are solicited to apply directly to this Corporation for treatment and that the data which they have requested from the patient on this form is for the purpose of arriving at some sort of a diagnosis. It must be obvious from this form alone that such diagnoses are made by mail in spite of the statements of the Corporation to the contrary.

A third replied:

> In reply to your questions as to whether the case history blank is actually diagnosis or an attempt thereat, there is no question in my mind whatsoever but that it is an attempt at diagnosis. For the numerous questions definitely attempt to determine the type of attack from which the patient suffers. In addition to that, it relates to the possible cause of such attacks.

All three agreed that the blank was totally inadequate to enable a physician to arrive at a proper diagnosis. Without personal examination of the patient, there could be no ruling out of such matters as brain tumor or the residual of trauma.

One may wonder why the vice-president of the Western Medical Corporation was careful to emphasize the fact that no diagnosing is done by the firm. There is no doubt that the corporation through its agents could not legally "diagnose." From another legal aspect, however, there appears to be a fiction indulged in by the corporation to suit its own purpose: If it is not found to be diagnosing, it should follow that it is not engaged in the corporate practice of medicine. It is only "dispensing" or "selling" drugs. This appears to be an effort at defense, a defense against a *quo warranto* proceedings with a view to judgment at ouster; in plain language, a court order to the firm to get out of business in Illinois. It so happens that there is legal precedent for such action.[2] The Illinois Attorney General's office might well inquire into the actual operation of the Western Medical Corporation. There appears to be an excellent case for such ouster proceedings. To say the least, there is great moral and ethical need for such ouster, if the best interests of epileptics are to be served. This holds true no matter where they may live.

If such action were successful, the persons who are listed on the firm's letterhead as "medical staff" or "chief-of-staff" should then find themselves in violation of that subsection of the Illinois Medical Practice Act that provides for revocation of license under such circumstances. Subsection 9 of Section 16 provides as one of the bases for revocation of license the following:

> Professional connection or association with or lending one's name to another for the illegal practice by another of the treatment of human ailments as a business or professional connection or association with any person, firm or corporation holding himself, themselves or itself out in any manner contrary to this Act.

The Bureau urges now that those in charge of administration and enforcement of the Illinois Medical Practice Act nullify the criticism of Senator Murray. Epileptics are not getting adequate or expert care. The mail-order practice of medicine can never be justified. The Bureau has called attention in the past to the reprehensible character of this promotion.[3] The Federal Trade Commission in 1943 exacted a promise from the firm to cease the misleading advertising practices then followed.

Along this line here is a revealing sidelight on the true character of this business. "Patients" are asked for the names and addresses of other epileptics. The firm, on receipt of such names, sends promotional literature, including a booklet it calls "Gratitude," to the prospective "patient."

"Gratitude" is a collection of testimonials of "grateful patients." It has been published in at least four editions (1936, 1938, 1943, and 1949). From edition to edition the pictures and letters of the same testimonial givers appear in several cases. The firm has modified and edited these testimonials from edition to edition. One wonders just whose testimonials they really are.

In spite of Mr. Creevy's testimony and pressure, which apparently convinced members of the Senate subcommittee, it seems preferable, in the light of modern scientific knowledge, to treat epileptics on the basis of individual care. This observation has been summed up rather pointedly in the following excerpt from a letter received from a staff member of a prominent eastern neurological institute. He has commented in part:

> Epilepsy is a condition which is highly individual with respect to causes and treatment. Seizures may be due to brain tumor which cannot be diagnosed by mail. The fact that a physician has examined the person and made a diagnosis of epilepsy does not mean that no neurosurgical treatment may be required. Among other things, the patient's doctor may not have divulged his fears.
>
> Medical treatment by the [Western Medical] Corporation is limited to phenobarbital, since the newer medicines require personal supervision because of the possibility of serious side effects such as allergic reactions or blood dyscrasia. Most patients require the use of dilantin, mesantoin, tridione or phenurone. However, patients taking the last three must have blood examinations at least monthly or personal observation in case of drug eruption. Obviously, such personal attention cannot be given by mail-order concerns. Consequently, these majority patients cannot be treated properly.

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[2] In the Kerner case (1936) the Illinois Supreme Court ousted the United Medical Service, which was incorporated and "practiced medicine." (200 N. E. 157).

[3] Western Medical Association: Another Phenobarbital (Luminal) Mail-Order Treatment for Epilepsy, The Propaganda for Reform, J. A. M. A. **78:** 296 (Jan. 28) 1922. Cass Treatment for Rheumatism: Another Piece of Chicago Mail-Order Quackery, The Propaganda for Reform, ibid. **88:** 189 (Jan. 15) 1927. The "Cass Treatment" Trickery, The Propaganda for Reform, ibid. **88:** 1983 (June 18) 1927. Van Ard Sanatorium: A Dangerous Piece of Mail-Order "Rheumatism Cure" Quackery, Bureau of Investigation, ibid. **94:** 1255 (April 19) 1930. The Western Medical Corporation: Selling Phenobarbital (Luminal) as a Mail-Order Treatment for Epilepsy, Bureau of Investigation, ibid. **101:** 463 (Aug. 5) 1933. Histeen, Another Addition to Asthma and Hay Fever Quackery, ibid. **101:** 1251 (Oct. 14) 1933. More Nostrums in Retrospect: Condensed Reports on "Patent Medicines" Previously Dealt with in Greater Detail, Bureau of Investigation, ibid. **105:** 1791 (Nov. 30) 1935. Stipulations: Agreements Between Federal Trade Commission and Promoters of Various Products, Bureau of Investigation, ibid. **125:** 1058 (Aug. 12) 1944. Western Medical Association Epilepsy Cure, Questions and Answers, Hygeia **3:** 59 (Jan.) 1925. Cramp, A. J.: Quack Epilepsy Cures, ibid. **6:** 263 (May) 1928.

BUREAU OF LEGAL MEDICINE AND LEGISLATION

MEDICOLEGAL ABSTRACTS

Basic Science Acts: Constitutionality of Arizona Basic Science Act.—An information was filed against the defendant, charging him with the unlicensed practice of chiropractic. Before making a decision, the trial judge certified certain constitutional questions to the Supreme Court of Arizona.

It was stipulated that the defendant took the basic science examination but failed to pass. He then took the examination given by the board of chiropractic examiners and passed but was not issued a license because he had failed to pass the basic science examination. Later the defendant commenced the practice of chiropractic without the proper certificate and license. The defendant moved to quash the information that had been filed against him on the ground that the basic science act of Arizona was unconstitutional.

In answering the first question certified to us, said the Arizona Supreme Court, we are confronted with the question of the unlawful delegation of legislative powers to the board of examiners. The court first said that the act itself defined the five basic sciences on which applicants are to be examined and that it did not delegate to the board of basic science examiners the responsibility of establishing the definition of the basic sciences, as the defendant contended. Nor, continued the court, does the act delegate legislative authority to the board, because no provision is made for examination of applicants or the grading of papers. The matter of selecting questions to be asked and the passing on or grading the papers is purely an administrative duty to be performed by the board. It is well established that the legislature may set a general standard for the guidance of a board, such as was done in the basic science law, and then leave the administrative details to the board. The Supreme Court therefor concluded that the basic science act was not unconstitutional as an unlawful delegation of legislative power to the board of examiners in the basic sciences.

The defendant next contended that the act violated the due process clauses of the Arizona and United States constitutions on the ground that the act designated no one to establish a definition of the terms "practice of healing" or "practicing healing" and that no provision was made for notifying applicants at such time as the term "basic science" was specifically defined. The defendant referred to the provisions of the act defining the above mentioned terms and including the phrase "and not otherwise specifically defined." Apparently, said the Supreme Court, the defendant had in mind that these provisions indicated that some independent definition would be made. This of course is erroneous as the phrase obviously means that the terms shall remain as defined by the provisions unless otherwise specifically defined. The defendant further complained that no technical qualifications were prescribed by the act for the members of the examining board and that the limitations imposed thereon tended to discriminate against chiropractic practice. The act provided that the board members be members of the faculty of the University of Arizona; this in itself, said the court, embodied certain qualifications. It cannot be said that every act establishing a board or committee that fails to specifically enumerate extensive and detailed qualifications for its members is for that reason violative of the constitutional due process clauses, in which a general qualification is established in the same act. The guarantee of due process is insured through provisions of the act providing for an appeal from the board's decision to the superior court. The court also found that limiting the appointments of members of the examining board to members of the faculty of the University of Arizona did not tend to discriminate against chiropractors. That was an additional regulating factor affecting all applicants equally.

In a Washington case involving an attack on the Washington basic science law, one of the questions raised was whether a person intending to practice as a chiropractor was deprived of due process because he was required to take an examination in the basic sciences. The Washington statute, said the Arizona Supreme Court, is identical with the Arizona statute except that in Washington chiropractors are not required to pass an examination in bacteriology. The Washington court held that the basic science law was not unconstitutional as a denial of liberty and due process of law. Furthermore, the Michigan Supreme Court upheld an act regulating the practice of the system of chiropractic and held that one desiring to practice that system was not deprived of equal protection of the laws by being required, as a condition for securing a license, to pass an examination in anatomy, histology, embryology, physiology, chemistry, bacteriology, pathology, diagnosis, hygiene, and public health, despite the fact that it was shown that such subjects were not taught in chiropractic schools.

Finally, the defendant contended that the act was ambiguous, indefinite, and a contradiction in terms. We are of the opinion, concluded the court, that the legislature sufficiently and adequately defined the terms used in the basic science act. The Supreme Court, therefore, held that the Arizona basic science act was not unconstitutional for any of the reasons raised by the defendant. *State v. Gee, 236 P. (2d) 1029 (Arizona 1951).*

Pharmacists: Sale of Sodium Amytal Without Prescription.—The plaintiff sued the defendant for actual damages alleged to have resulted from the sale by the defendant of "sodium amytal" without a physician's prescription. From a judgment in favor of the plaintiff, the defendant appealed to the Supreme Court of Oklahoma.

The defendant, Ralph Wootan, was the owner and operator of a drug store wherein he had in his employ the defendant McMillian, as a pharmacist. The plaintiff went to the defendant's drug store to get some medicine to relieve a headache. The defendant, McMillian, sold him eight capsules containing the drug "sodium amytal" and instructed the plaintiff to take them in the same manner as he would take anacin® tablets. The plaintiff took two of the capsules in the drug store; the next day he took four, two in the morning and two later. As a result, the plaintiff was unconscious for two days and was away from his work for about eight days. He was treated during this time by a physician who appeared as a witness at the trial. McMillian sold the medicine to the plaintiff without requiring a physician's prescription and in violation of the law.

Section 9, Title 23, of the Oklahoma statute provides: "In any action for the breach of an obligation not arising from contract, where the defendant has been guilty of oppression, fraud or malice, actual or presumed, the jury, in addition to the actual damages, may give damages for the sake of example, and by way of punishing the defendant." On the basis of this statute, the plaintiff sought to recover actual damages and also punitive damages against the defendant because of the fact that the drug was sold without a physician's prescription. Punitive damages were assessed by the trial court, and the defendant appealed on the ground that the trial court erred in giving an instruction to the jury. The instruction complained of was as follows: "You are instructed that our statutes provide that in any action for the breach of an obligation not arising from contract where it is shown that the defendant has been guilty of violation, of a positive criminal statute or of oppression, fraud or malice, actual or presumed, the jury, in addition to actual damages, may allow damages for the sake of example, and by way of punishing the defendant."

To entitle a plaintiff to recover exemplary damages in an action sounding in tort, said the Supreme Court, the proof must show some element of fraud, malice, or oppression. The act that constitutes the cause of action must be actuated by or must be the result of such gross negligence or such disregard of another's rights as is deemed equivalent to such intent. The instruction complained of in the case at bar, the court continued, had the effect of authorizing recovery of punitive damages if the defendant was merely guilty of violating a positive criminal statute. Such was beyond the scope and meaning of the statute and was in conflict with the conclusion of prior decisions of the Supreme Court of Oklahoma. Accordingly, the portion of the judgment of the trial court awarding punitive damages was reversed. *Wootan v. Shaw, 237 P. (2d) 442 (Oklahoma 1951).*

MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A.M.A. Am. J. Diseases Children, Chicago

83:573-718 (May) 1952

*Studies on Interstitial Giant Cell Pneumonia: I. Report of Five Cases with Autopsy. M. Wolman, G. Izak, E. Freund and Z. Shamir. —p. 573.
Incidence of Skin-Test Reactions Among Children in Pittsburgh District: I. Histoplasmin. A. M. Carpenter and P. D. Spino.—p. 589.
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Id.: III. Moniliin. A. M. Carpenter, J. C. Hadden and P. D. Spino. —p. 603.
Allergy to Viral and Rickettsial Vaccines: II. Allergic Reactions Encountered and Further Studies with Refined Influenza and Mumps Vaccines in Allergic Children. B. Ratner, S. Untracht and F. Hertzmark.—p. 608.
Water Metabolism in Water Intoxication: Review of Basic Concepts. J. L. Baskin, H. M. Keith and B. H. Scribner.—p. 618.
*Scarlet Fever: Evaluation of Continuous and Intermittent Penicillin Therapy. P. L. Mathieu Jr., B. J. Mathieu and E. J. West.—p. 628.
Acetic Acid Aerosol in Treatment of Purulent Bronchiectasis Due to Pseudomonas Aeruginosa. W. W. Currence.—p. 637.
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Circulation Times in Newborn Infants by Fluorescein Method. R. E. Lesser, R. Meyer and A. T. Henderson.—p. 645.
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Congenital Direct Communication Between Biliary System and Respiratory Tract. E. B. D. Neuhauser, M. Elkin and B. Landing.—p. 654.
Rectal Lesions of Lymphogranuloma Venereum in Childhood: Review of Literature and Report of Case in 10-Year-Old Boy with Rectal Stricture. L. Banov Jr.—p. 660.
Primary Malignant Tumor of Pancreas in 15-Month-Old Boy. R. O. Warthen, M. C. Sanford and E. C. Rice.—p. 663.

Interstitial Giant Cell Pneumonia.—The clinical and pathological changes in five infants between the ages of 3 weeks and 1 year with interstitial giant cell pneumonia are described. There was clinical evidence of bronchial involvement without consolidation. Three patients had diarrhea and were dehydrated, and two of them had evidence of nonpurulent pharyngitis. The temperature was either normal or subfebrile. The two patients who were treated with sulfonamide drugs, penicillin, and streptomycin did not respond to the treatment. All the patients died, and three of them suddenly, without premonitory symptoms. Necropsy was performed in all. Besides the usual findings of interstitial pneumonia, there was pathological evidence of generalized lymphatic tissue involvement. Epithelial giant cells containing cytoplasmic inclusion bodies were found in the alveoli and bronchi. Interstitial giant cell pneumonia is considered to be due to a virus. Such a view is supported by the presence of intracellular inclusion bodies, by the epithelial proliferation and metaplasia, by the interstitial type of the pneumonic process, and by the noncontributory bacteriological findings. In the authors' cases there was no clinical evidence of measles, nor were there changes in the lymphoid tissues similar to those described in measles. Interstitial giant cell pneumonia resembles the disease called virus pneumonia of infants, plasma cell pneumonia, or pneumonia of premature infants except for the presence of giant cells. It is suggested that interstitial giant cell pneumonia and virus pneumonia of infants represent a single disease entity caused by a virus.

Penicillin in Scarlet Fever.—Continuous and intermittent treatment with penicillin was given to 386 patients with scarlet fever. Of the 386 patients, 119 received 20,000 to 40,000 units of an aqueous solution of penicillin G intramuscularly every three hours for seven days. The other 267 patients were given 300,000 units of procaine penicillin intramuscularly every 24 hours for seven days. Results in the treatment of scarlet fever were equally good with both regimens. Ninety per cent of all patients were afebrile 72 hours after treatment with penicillin was started. More than 90% were free from Streptococcus pyogenes within 24 hours. The number of complications was reduced. In 8% of the patients nonsuppurative complications developed after treatment was discontinued. With penicillin administered every three hours suppurative complications occurred in 1.7% of the cases, and with penicillin administered every 24 hours in 0.8% of the cases. There were no late sequelae in any patient. The relative mildness of scarlet fever during the past years and the extreme sensitivity of the hemolytic Streptococcus organism to penicillin have combined to eliminate mortality due to this disease and to reduce the incidence of early and late complications.

A.M.A. Arch. Neurology and Psychiatry, Chicago

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Hypothalamic Syndromes Complicating Antirabies Vaccination. A. J. S. McFadzean.—p. 451.
*Trihexyphenidyl (Artane®), Stramonium, and Nonchemotherapy of Paralysis Agitans. B. Berkowitz and E. Alvermann.—p. 462.
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Cerebral Angiography with Iodopyracet Injection U.S.P. (Diodrast®): Its Dangers, Particularly in Hydrocephalic Infants. I. M. Tarlov and M. Rosenberg.—p. 496.
Effects of Frontal Lobotomy on Morphine-Abstinence Syndrome in Man: Experimental Study. A. Wikler, M. J. Pescor, E. P. Kalbaugh and R. J. Angelucci.—p. 510.
Effect of Epinephrine on Cortical and Basal Electroencephalograms and Eosinophile Count. L. A. Gottschalk.—p. 522.
Psychosomatic Aspects of Multiple Sclerosis. H. A. Teitelbaum, B. H. Hall and R. E. Phillips.—p. 535.
Expanding Concepts in Neurophysiology. M. A. B. Brazier.—p. 545.

Trihexyphenidyl (Artane®), Stramonium, and Nondrug Therapy of Paralysis Agitans.—Many patients with paralysis agitans (Parkinson's disease) will be benefited by a combination of drug therapy, physical therapy, and psychotherapy. Objective criteria for measuring changes during therapy were established by recording the patient's efficiency in performing 16 exercises designed to measure function. Subjective changes were evaluated by the patient's answers to 34 standard questions, asked each week. One group of 19 patients were given stramonium, and another of 17 patients were given trihexyphenidyl (artane®). Five patients added after the start of the experiment received no drug therapy during the initial period; later, two were given stramonium, and three trihexyphenidyl. The first period of treatment lasted 16 weeks; in the second period of 14 weeks, the drugs were changed so that the first group received trihexyphenidyl and the second stramonium. The patients were not told what drugs they were receiving, or that the drugs were changed. Trihexyphenidyl was not demonstrably superior to stramonium. Improvement ascribed to medication is often the result of physical therapy and psychotherapy. The patients in both groups and the five added later showed an objectively measurable improvement after 16 weeks; the mean level of efficiency in the objective tests was not greater

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Titles marked with an asterisk (*) are abstracted.

after 30 weeks than after 16. Mental activities, however, seemed to improve continuously with practice. Subjective evaluation may be misleading; objective tests often failed to substantiate changes reported by the patient.

A.M.A. Arch. Otolaryngology, Chicago

55:405-524 (April) 1952

SYMPOSIUM ON FACIAL PARALYSIS

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James Lawrence Little, a Forgotten Pioneer. J. J. Rainey.—p. 451.
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The Paranasal Sinuses. S. Salinger.—p. 489.

American Journal of Clinical Pathology, Baltimore

22:301-402 (April) 1952

*Acquired Hemolytic Anemia. A. S. Wiener, A. A. Samwick, M. Morrison and L. Loewe.—p. 301.
Preservation of Dried and Frozen Plasma Over 10-Year Period. M. M. Strumia, J. J. McGraw Jr. and G. E. Heggestad.—p. 313.
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*Renal Papillary Necrosis. A. Knutsen, E. R. Jennings, O. A. Brines and A. Axelrod.—p. 327.
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Determination of Concentration of Least Coagulable Protein (Huggins' Test): Study of Over 1,100 Consecutive Clinic Admissions. L. A. Schneider, J. L. Eisaman and R. E. Berger.—p. 345.
Adrenocortical Function in Portal Cirrhosis. A. J. Finestone and C. R. Shuman.—p. 348.
Atherosclerosis: In Vitro Study of Pathogenesis of Atherosclerosis. S. M. Evans, H. K. Ihrig, J. A. Means and others.—p. 354.
Simplified Index of Pathologic Diagnoses. E. Woll.—p. 364.

Acquired Hemolytic Anemia.—Three unusual examples of acquired hemolytic anemia are described, with special reference to the role of autoantibodies in the pathogenesis of the disease. The first patient was a woman with "coated" red cells but without demonstrable autoantibodies or isoantibodies in her serum; nevertheless she died following severe hemolytic reactions after transfusions of blood that was compatible in vitro. The second patient had thrombocytopenia in addition to anemia and icterus, and did not immediately improve following splenectomy. Hemolytic transfusion reactions were explained by sensitization to the Kell (or Si) factor, and avoided by transfusing Kell-negative blood, but the purpura did not improve. The patient's serum failed to clump her own unmodified red cells at body temperature, but autoagglutination occurred at body temperature and refrigerator temperature when the red cells were first treated with proteolytic enzymes. The patient showed clinical improvement when transfusions were withheld and corticotropin was administered. The blood count rose but the purpura persisted. Following discharge from the hospital the blood count and platelet count became normal. Possibly this improvement represents a delayed effect of the splenectomy. To date, the patient has remained well, even though the autoantibodies are still demonstrable in low titer, the cells have become coated again, and the sedimentation rate remains elevated. The third patient had hemolytic anemia as a complication of atypical pneumonia. The anemia was explained by the demonstration of potent cold autoagglutinins in the patient's serum (titer 1,000 units for untreated cells at refrigerator temperature, but scarcely any reaction at body temperature). The patient was given four transfusions of whole blood and recovered completely from the anemia. These observations illustrate the graver significance of autoantibodies, which act preferentially at body temperature even though of low titer, while the patient in case 3 shows that hemolytic anemia due to cold autoantibodies usually has a good prognosis even though the antibodies are of high titer.

Renal Papillary Necrosis.—Renal papillary necrosis has only recently become generally recognized as a distinct pathological entity that occurs almost exclusively in patients with diabetes mellitus or chronic obstruction of the urinary tract. Papillary necrosis, renal papillitis, necrotizing renal papillitis, medullary necrosis, necrotizing pyelonephritis, and papillitis renis necroticans are some of the synonyms for this term. Of the 16 cases described in this report, 14 were verified at necropsy, 1 by the examination of a surgically removed kidney, and 1 by the examination of a necrotic papilla removed cystoscopically. Eleven of the patients were diabetic and four of the remainder had urinary tract obstruction. Six patients exhibited an acute rather fulminating clinical course characterized by several renal or systemic infections with fever, pyuria, progressive azotemia, coma, and death within 17 days of the onset. Eight patients, five of whom are diabetic, had a subacute course that lasted from 30 to 100 days. The third clinical pattern was shown by two patients, both of whom had unilateral renal stone with severe colic and hematuria that were self-limited. The gross and histological changes in the kidneys do not support the hypothesis that there is a "reverse Trueta shunt" in the development of renal papillary necrosis. Thrombosis was rare. Arteriosclerosis, arteriolosclerosis, and bacterial infection were the commonest and most consistent findings in the kidneys, and it is assumed that the earlier onset and greater severity of such vascular changes in patients with diabetes may explain also the high incidence of renal papillary necrosis in diabetic patients. Renal ischemia from vascular disease seems to be the most important predisposing factor in the pathogenesis of the lesion in man. The normal relative ischemia of the renal papillae and subjacent pyramids apparently localizes the lesion to its characteristic site.

American Journal of Obstetrics & Gynecology, St. Louis

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Etiology of Pre-Eclampsia-Eclampsia: IV. Sodium Chloride Test for Diagnosis of Pre-Eclampsia. W. J. Dieckmann, R. E. Pottinger and L. M. Rynkiewicz.—p. 783.
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Upper Age Limit of Parturition: Review of Literature. J. W. Newell and J. Rock.—p. 875.
Fructose and Fructolysis in Human Semen. C. H. Birnberg, D. A. Sherber and R. L. Kurzrok.—p. 877.
Rate of Growth of Uterine Myomas. H. Speert.—p. 880.

Cancer of Cervix Diagnosed After Hysterectomy.—Carcinoma of the cervical stump is apparently declining owing to the increased use of total hysterectomy, but about 1% of the total hysterectomies performed for myoma or uterine prolapse at the woman's clinic of the New York Hospital between 1947 and

1951 resulted in the detection of hitherto unsuspected cervical cancers. Nineteen of these 21 patients had squamous cell carcinoma (9 in stage 0 and 10 in stage 1), while two had adenocarcinoma. Sixteen of the cancers had no physical signs, while five deserved further preliminary study, because of erosion, contact bleeding, or postmenopausal bleeding. Multiple biopsies of the squamocolumnar junction and curettage or aspiration smears of the endocervix would have resulted in preoperative diagnosis of the cancer. Nine of the cancers were in the endocervix and 10 at the squamocolumnar junction, while only 2 were on the exocervix. The cervical cancers that are most likely to be undiagnosed are (1) those of the endocervical canal, (2) squamocolumnar junction cancers, (3) very early cancers of the exocervix, and (4) cancer associated with descensus. None of the patients with stage 0 cancer were treated further. One-half of the patients with stage 1 cancer, and all of the patients with adenocarcinoma received irradiation. No additional surgery was done to remove tubes or ovaries. All patients are living and have no evidence of recurrence. Fourteen patients have been observed for over two years.

American Journal of Ophthalmology, Chicago

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Tuberculous Choroiditis Associated with Xanthoma Tuberosum. E. Rosen. —p. 573.
Cataracts and Retinopathy in Juvenile Diabetes. W. G. Marr.—p. 577.

Ocular Sarcoidosis.—Sarcoidosis is characterized by the presence in organs or tissue of epithelioid-cell tubercles, and refractile or apparently calcified bodies are frequently present in the giant cells of the tubercles. The characteristic lesions may be replaced by fibrosis, hyalinization, or both. Three cases of Boeck's sarcoid involving the uveal tract are presented. The first patient showed precipitates on the corneal endothelium, and had only enough vision to count fingers at 18 inches. Enlarged inguinal nodes were present, and a biopsy revealed sarcoid. The patient received a total of 360 mg. of corticotropin, parenterally administered, but there was no improvement in vision. The second patient had posterior keratic deposits, as well as a choroidal lesion in the left fundus that was thought to be sarcoid. Biopsy of an enlarged femoral node revealed lesions of Boeck's sarcoid. This patient received a course of old tuberculin, with no improvement. A total of 100 gm. of urethane was administered. Vision improved in the left but not in the right eye. The third patient had myocardial, pulmonary, and skin sarcoidosis in addition to the uveal disease. She received 240 mg. of corticotropin systemically and cortisone was applied locally. Improvement was marked. The final refraction gave vision of 20/70 in the left eye, which had been virtually blind; in the right eye vision improved from light perception to 20/30. It is felt that corticotropin and cortisone may be of some value in the treatment of sarcoidosis involving the ocular structures, and the results in case 3 warrant further trials with these hormones.

American Journal of Physiology, Baltimore

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Effect of Chronic Salt Depletion on Blood Pressure of Renal Hypertensive Dogs. J. Frieden, J. Stamler, W. Hwang and others.—p. 500.
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American Journal of Surgery, New York

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Vascular Tumors of Head and Neck. J. W. Hendrick.—p. 640.
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•Cancer of Rectum Diagnosed by Sponge Biopsy. S. A. Gladstone. —p. 664.
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Persistent Omphalomesenteric Duct: Incidence Relative to Meckel's Diverticulum. K. L. Brown and D. M. Glover.—p. 680.
Pre- and Postoperative Care for Anorectal Pathologic Conditions. F. H. Murray.—p. 686.
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Technic for Lumbar Sympathetic Ganglionectomy. J. L. Madden. —p. 695.

Cancer of Rectum Diagnosed by Sponge Biopsy.—Sponge biopsy is a surface biopsy in which tissue is collected with a sponge for microscopic examination. Its efficacy was first tried on 10 surgical specimens from patients with cancer of the colon. Pieces of sponge were firmly rubbed over the base and margins of the ulcerating lesions. Examination of the sponges and absorbed contents revealed the presence of diagnosable cancerous tissue in each instance. Then sponge biopsies from 52 patients with rectal lesions were studied, and 15 were diagnosed as positive for cancer. In 14 the diagnosis was confirmed by surgical biopsy or surgical specimen. In one case additional tissue for confirmation was not available, but subsequent laparotomy revealed an annular lesion of the colon with metastases to the liver. A single piece of tissue removed by surgical biopsy may miss the cancer, but this is not likely with sponge biopsy, which provides many small tissue particles from different areas of the ulcer base and margins. The prognosis of a cancer large enough to be recognizable clinically is likely to be poor, but total removal and complete cure may be possible with a small ulcer that can be identified as cancer only by microscopic examination.

Am. J. Tropical Medicine & Hygiene, Baltimore

1:177-360 (March) 1952. Partial Index

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Histopathology of Experimental Schistosomiasis: I. Hepatic Lesions in Mice Infected with S. Mansoni, S. Japonicum and S. Haematobium. H. E. Meleney, D. V. Moore, H. Most and B. H. Carney.—p. 263.
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Anesthesiology, New York

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Use of Pontocaine in Subposologic Quantities for Bronchoscopy and Bronchography. A. A. Carabelli.—p. 169.
Densities of Common Spinal Anesthetic Solutions at Body Temperature. H. Davis and W. R. King.—p. 184.
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Place of Leather-Soled Shoes in Prevention of Anesthetic Explosions. B. A. Greene.—p. 203.
*Use of Sciatic Nerve Block for Producing Vasodilatation of Lower Extremity and Comparative Study with Paravertebral Lumbar Sympathetic Ganglion Block. M. J. Marmer.—p. 207.

Sciatic Nerve Block in Therapy of Peripheral Vascular Diseases. —The sciatic nerve was blocked with 10 cc. of 1% procaine solution in 42 patients, and paravertebral lumbar sympathetic block was done in 11 patients with peripheral vascular diseases. Readings of the surface temperature of each toe, heel, and ankle showed that sciatic nerve block is as effective as lumbar sympathetic ganglion block, and sometimes a greater degree of vasodilatation is obtained. In the four patients in whom sympathetic block was performed on one side first and then sciatic block on the other, the rise in temperature of the foot was more rapid, more sustained, and greater with sciatic block. Temperature rises varied from 0.5 F to 22 F, and the more complete the block the greater the degree of vasodilatation. The temperature rises within 10 minutes after the block, reaches its maximum in 20 to 30 minutes, and remains elevated for at least one hour. With this method total paralysis of the foot persists for about 45 minutes, with no pain for the patient, who feels at ease and seems to enjoy the increased warmth of the foot. After the anesthesia wears off, the patient experiences euphoria. Two patients with intermittent claudication obtained relief, thus suggesting an increase of blood flow to the muscles. Sciatic nerve block is easier to perform and less painful than paravertebral lumbar sympathetic ganglion block, and complications are rarer. None was reported in these 42 patients, whereas in 1 of the 11 patients in whom sympathetic block was performed a cardiovascular accident resulted in right hemiplegia.

Annals of Allergy, Minneapolis

10:109-244 (March-April) 1952. Partial Index

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Hypoallergic Penicillin: IV. Chlor-Trimeton-Penicillin. S. W. Simon. —p. 187.
Treatment of Hay Fever by Intravenous Antihistamine. G. K. Spearman. —p. 192.

Circulation, New York

5:481-640 (April) 1952

Effect of Aging and of Development of Disease on Ballistocardiogram: Study of 80 Subjects, Originally Healthy, Followed from 10 to 14 Years. I. Starr and E. A. Hildreth.—p. 481.
Tissue Distribution and Excretion of Radioactive Digitoxin: Studies on Normal Rats and Cats, and Rats with Dietary-Induced Myocardial Lesions. C. S. Fischer, A. Sjoerdsma and R. Johnson.—p. 496.
Certain Mechanical Peculiarities of Human Cardiac Pump in Normal and Diseased States. G. E. Burch, C. T. Ray and J. A. Cronvich. —p. 504.
*Obesity, Fat Metabolism and Cardiovascular Disease. J. W. Gofman and H. B. Jones.—p. 514.
Studies in Experimental Pericardial Tamponade: Effects on Intravascular Pressures and Cardiac Output. J. Metcalfe, J. W. Woodbury, V. Richards and C. S. Burwell.—p. 518.
*Use of Combination of Anion and Cation Exchange Resins in Treatment of Edema and Ascites. B. L. Martz, K. G. Kohlstaedt and O. M. Helmer.—p. 524.
Studies of Plasma Quinidine Content: III. Value of Delayed Absorptive Coated Tablets in Oral Quinidine Therapy. J. J. Sampson, H. Foreman and B. C. Solomon.—p. 534.
Etiologic Concept of Atherosclerosis Based on Study of Intimal Alterations After Shock. O. J. Pollak.—p. 539.
Effects of Intravenous Procaine and Procaine Amide (Pronestyl) upon Ectopic Ventricular Tachycardia Accompanying Acute Myocardial Infarction. A. S. Harris, A. Estandía, T. J. Ford Jr. and others. —p. 551.
Electrocardiogram in Heart Disease Detection: Comparison of Multiple and Single Lead Procedures. T. R. Dawber, W. B. Kannel, D. E. Love and R. B. Streeper.—p. 559.
Vasodilator Effects of Substance Present in Normal Human Urine in Comparison with Effect of Methacholine and Sodium Nitrite: Statistical Analysis of Reliability of Method of Assay. H. D. Green, J. M. Little, C. G. Gaddy and others.—p. 567.
Effect of Sympathectomy for Essential Hypertension on Hallucal Circulation. M. Mendlowitz and A. S. W. Touroff.—p. 577.
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Observations Concerning Metabolism of Cholesterol in Hypo- and Hyperthyroid Rat. R. H. Rosenman, M. Friedman and S. O. Byers. —p. 589.
Tissue Clearance as Measure of Nutritive Blood Flow and Effect of Lumbar Sympathetic Block upon Such Measures in Calf Muscle. S. I. Rapaport, A. Saul, C. Hyman and M. E. Morton.—p. 594.
Abnormal Serum Lipid Pattern in Patients with Coronary Arteriosclerosis. A. Steiner, F. E. Kendall and J. A. L. Mathers.—p. 605.
Functional Importance of Coronary Collaterals. C. J. Wiggers.—p. 609.

Obesity, Fat Metabolism, and Cardiovascular Disease.—In previous reports concerning serum lipid transport via lipoproteins, Gofman and Jones established that certain classes of lipoproteins are closely associated with development of human atherosclerosis. Then they decided to determine whether obesity exerts pressure on irradiated mucosa. Teeth, irrespective of their condition, should be removed before irradiation unless they are adequately protected, the alveolar process should be prepared and the mucosa sutured. The mucosa should be completely healed before the treatment is started. The incidence of this complication is suggested by the fact that at a New York hospital 235 patients developed irradiation osteonecrosis among a total of 1,819 patients with intraoral cancer. In this paper Cook describes 11 cases of irradiation osteonecrosis. The therapeutic approach to this problem has varied from the recommendation to wait until sequestration to resection of the involved part.

New England Journal of Medicine, Boston

246:561-596 (April 10) 1952

Preventive Medicine in Obstetrics. D. Baird.—p. 561.
*Cortisone and ACTH as Adjunct to Surgery of Craniopharyngiomas. F. D. Ingraham, D. D. Matson and R. L. McLaurin.—p. 568.
Miliary Tuberculosis Developing During Pregnancy. R. H. Goodwin and M. D. Kenler.—p. 572.
Practical Aspects of Cancer Prevention. E. L. Wynder.—p. 573.
ACTH and Cortisone in Treatment of Sickle-Cell Anemia: Report of Case. M. Sass.—p. 583.

Cortisone and ACTH in Surgery of Craniopharyngiomas.—The complications in surgical treatment of craniopharyngioma result from the proximity of the lesion to the pituitary gland and the hypothalamus. Manipulation in this region is perilous, and the danger is increased because of depression of the pituitary-adrenocortical response to stress. Major complications include disturbances of vasomotor control, temperature regulation, and salt, water, and carbohydrate metabolism. Adrenal substitution therapy in the form of cortisone and corticotropin was first employed by Ingraham and associates as an adjunct to operation for pituitary adenoma. In this paper they record experiences with these drugs in four children who underwent operation for craniopharyngioma. One received corticotropin and three cortisone. The uncomplicated postoperative recovery of these four patients suggests that adrenal substitution therapy is a useful adjunct in the surgical management of craniopharyngiomas in children. The importance of replacement therapy may be even greater from adolescence to middle age, because endocrine deficiency is usually more pronounced in this age range. The relatively short period during which large doses of hormone (200 mg. of cortisone or 100 mg. of corticotropin per day) are administered to these patients precludes most complications that may follow long-term therapy. In the reported cases no untoward effects of the hormonal therapy were noted.

246:597-636 (April 17) 1952

Combined Effect of Cortisone and Partial Protein Depletion on Wound Healing. C. W. Findlay Jr. and E. L. Howes.—p. 597.
Urinary Colloids in Prevention of Kidney-Stone Formation. A. J. Butt and E. A. Hauser.—p. 604.
Contribution of Psychiatry to Medical Practice. R. W. Hyde.—p. 607.
Penicillin Sensitivity of Staphylococci. E. Reiss, E. J. Pulaski, W. H. Amspacher and A. A. Contreras.—p. 611.
Progress in Obstetrics. B. Tenney Jr.—p. 613.
Physicians in Art. S. C. Wiggin.—p. 620.

246:637-676 (April 24) 1952

*Replacement of Gastric and Intestinal Fluid Losses in Surgery: Preliminary Report. R. E. Cooke and L. G. Crowley.—p. 637.
*Paroxysmal Nocturnal Hemoglobinuria: Clinicopathological Correlation. R. R. Merliss.—p. 642.
*Hypertensive Toxemias of Pregnancy: Simplified Method of Management Using Purified Extract of Veratrum Viride. F. A. Finnerty Jr.—p. 646.
Radiation Therapy of Carcinoma of Cervix: Review of Fundamental Considerations. J. D. Reeves.—p. 652.
Pneumocholecystitis: Report of Case. W. G. Abel and L. M. Rousselot.—p. 662.

Replacement of Fluid Losses in Surgery.—Three general principles should control fluid therapy: replacement of deficits, maintenance (supplying water, electrolyte, protein, calories, and vitamins), and concurrent replacement of abnormal fluid losses. Deficits in body composition and renal adjustments can be avoided if volume-for-volume replacement of abnormal fluid losses is carried out with solutions similar in composition to those being lost, and at the rate at which the losses occur. A system of fluid therapy has been developed in the department of pediatrics of Yale University School of Medicine that is applicable to patients of all types and ages. Two solutions are used in replacing abnormal fluid losses; a "gastric replacement solution" for losses incurred through gastric suction or vomiting, and an "intestinal replacement solution" for losses incurred through intestinal suction and biliary or pancreatic drainage. Both solutions provide potassium; the gastric replacement solution provides an excess of chloride over sodium ions, preventing the development of metabolic extracellular alkalosis, which leads to severe intracellular potassium depletion, and the intestinal replacement solution provides sodium in excess of chloride, preventing the development of metabolic acidosis. These solutions, which are similar in composition to the secretions they replace, are usually given intravenously, but may be given subcutaneously without reaction. They are often mixed with other fluids used for maintenance therapy, and a 50% solution of dextrose is sometimes added when extra calories are needed. The rate at which they are given has never exceeded 500 cc. an hour. The patient's drainage losses and fluid intake are calculated, frequently at first, later at 12 hour intervals, and the appropriate amount of the proper type of solution is added to the other required fluids for the ensuing period. The solution to be used is determined by the drainage tube location and sometimes by the type of drainage. Examination of patients receiving fluids according to this system revealed no evidence of central nervous system stimulation or depression such as might result from an excess of ammonium ions; edema was not observed; there were no signs of toxicity; the electrocardiogram was not affected; and no urinary abnormalities were detected. All patients expressed a feeling of well-being, and many were intermittently ambulatory.

Paroxysmal Nocturnal Hemoglobinuria.—The hemolysis characteristic of paroxysmal nocturnal hemoglobinuria is related to the physiological drop in the pH of the serum during sleep. Anemia is usually intense, and is accompanied by reticulocytosis, icterus, and leukopenia. Splenomegaly and lymphadenopathy are commonly present. Life may be maintained by supportive measures, but the disease has never been known to disappear either spontaneously or as a result of treatment. Two cases, one of which ended in death, are reported; so rare is the condition that only 45 cases have previously been reported in the literature, and some of these are doubtful. The name "paroxysmal nocturnal hemoglobinuria" is misleading, first, because the hemoglobinuria is not necessarily nocturnal and, secondly, because it is not constant, long periods without it occasionally occurring. Its absence, however, does not mean that the disease is arrested; hemolysis may be active and yet not rapid enough to produce a hemoglobin serum level greater than the renal threshold for hemoglobin. Both the author's patients had an elevated icterus level, an abnormally high reticulocyte count, and a decreasing red cell count even during periods of anhemoglobinuria. Any elevation of temperature precipitates a hemolytic crisis and may endanger the patient's life. Transfusions are needed to raise the hemoglobin level, which falls alarmingly during febrile periods. Transfusions were sometimes accompanied by severe febrile reactions not due to blood incompatibility; when this occurred, hemolysis and hemoglobinuria were intense, but, when no complications accompanied the transfusion, the signs of hemolysis decreased and the urine often cleared completely. The higher the hemoglobin level was elevated the longer the absence of hemoglobinuria. Transfusions had more than a replacement value in these patients; they slowed or suspended the hemolytic process temporarily. Normal red cells cannot be hemolyzed by the disease, and this makes transfusions especially effective. No other treatment proved useful. Administration of riboflavin in one case and of acetylsalicylic acid (aspirin) in both provoked hemoglobinuria. Both patients had intra-abdominal pain, sometimes severe. Deposition of hemosiderin in the renal tubules only, as found at autopsy in the case of one patient, suggests that the actual breakdown of the red cells occurs in the epithelium of these tubules.

Hypertensive Toxemia of Pregnancy.—Aqueous injectable "vergitryl" (a purified extract of Veratrum viride containing known concentrations of the hypotensive ester alkaloids) was used as the principal therapeutic agent in 122 cases of hypertensive toxemia of pregnancy. The average effective intramuscular dose of "vergitryl" is 0.75 unit (0.5 cc.), and the following schedule was established for treating nonconvulsive toxemias, including postpartum preeclampsia: 0.75 unit (0.5 cc.) of "vergitryl" mixed with 1 cc. of 1% procaine solution (to minimize pain at the injection site) is given intramuscularly at once; blood pressure and pulse are recorded every half hour and additional "vergitryl," 0.75 unit intramuscularly, is given whenever the blood pressure exceeds 140/90 (once every hour if needed); if there is no hypotensive effect at the end of one hour, dosage is increased to 0.9 unit (6 cc.). If nausea or vomiting occurs, 50 mg. of pentobarbital sodium may be given intravenously. The patient's response determines the dosage interval; if 0.75 unit of "vergitryl" keeps the blood pressure below 140/90 for three hours, the dose may be repeated at three hour intervals. The amount and dosage interval vary with each patient and become evident as treatment proceeds. Intravenous administration is used in treating convulsive toxemia, which always constitutes an emergency. One physician records the blood pressure every minute while another gives a solution of 1.5 units (1 cc.) of "vergitryl" with 20 cc. of 5% dextrose in water at a rate of 1 cc. per minute until the first 20 mm. fall in systolic or 10 mm. fall in diastolic pressure occurs. The needle is left in the vein, but "vergitryl" administration is suspended, because the pressure frequently drops precipitously in the next one or two minutes. If it does not, "vergitryl" administration is continued at the rate of 1 cc. per minute until the first sign of hypotension appears. The needle is then replaced by a polyethylene catheter, through which all additional medications are given. Blood pressure recordings should be made at 15 minute intervals after the first hypotensive effect occurs, and additional "vergitryl" (half the previous effective dose) given whenever the pressure is 140/90 or more. The need for the drug occurs at definite intervals, which increase in length as the severity of the condition diminishes. Vomiting was the only toxic manifestation, occurring in 16% of the cases, and was easily controlled with moderate doses of pentobarbital sodium given intravenously. There were no maternal deaths and only two fetal deaths in this series.

New Orleans Medical and Surgical Journal

104:385-426 (April) 1952

Use of Streptokinase and Streptodornase in Pelvic Abscesses: Preliminary Report. C. G. Collins and G. W. Tucker.—p. 385.
Use of Cortisone in Pelvic Cellulitis: Preliminary Report. C. G. Collins, V. A. Davidson and N. M. Mathews.—p. 389.
Current Applications of Angiocardiography. H. J. Jacobs.—p. 394.
The Gynecologist Evaluates Lower Abdominal Pain. C. G. Johnson.—p. 399.
Suppurative Pericarditis with Report of Three Cases. C. V. Menendez.—p. 402.
Carcinoma of Thyroid. L. H. Strug.—p. 409.
Trachoma Therapy. J. W. Rosenthal.—p. 413.

North Carolina Medical Journal, Winston-Salem

13:109-164 (March) 1952

SYMPOSIUM ON PREMATURITY

Prophylactic Treatment of Prematurity. B. Carter, R. A. Ross and V. H. Turner.—p. 109.
Management of Labor and Delivery in Interest of the Premature Infant. D. L. Whitener.—p. 115.
Anesthesia for Premature Labor: Prevention of Anoxia. B. C. Ogle.—p. 119.
Hospital Management of Premature Infants. A. McBryde.—p. 123.
Evaluation of the North Carolina Premature Infant Care Program R. J. Murphy.—p. 125.

Ureterosigmoidostomy: Past and Present. H. H. Baird and H. W McKay.—p. 129.
Allergic Manifestations in Otolaryngology. J. F. McGowan.—p. 137.
Diseases of Larynx: Photographic Study. J. C. Peele.—p. 143.
Acute Interstitial Myocarditis Associated with Carcinoma of Body of Pancreas: Report of Case. J. O. Williams, R. S. Pollitzer and H .D. Green.—p. 147.

Northwest Medicine, Seattle

51:281-372 (April) 1952

Nasal Triad, Nasal Allergy and Deafness. A. R. Miller.—p. 302.
Intussusception in Adults. E. R. Sanderson.—p. 310.
Multiple Liver Abscess. J. H. Mills.—p. 315.
Unusual Skin Metastasis and Pulmonary Lymphangitic Metastasis. C. S. Campbell.—p. 316.
Obstructing Lesion of Colon Due to Non-Penetrating Trauma of Abdomen. G. E. Jones and J. W. Settle Jr.—p. 317.

Ohio State Medical Journal, Columbus

48:391-488 (May) 1952

Rehabilitation of the Hemiplegic. H. J. Bearzy.—p. 409.
Medical Aspect Aboard the Snow-Bound Streamliner, "City of San Francisco." W. H. Roehll.—p. 415.
*Goitrogenic Action of Antithyroid Drugs: Major Side Effect Causing Surgical Difficulty. C. B. DeCourcy.—p. 417.
Fixation of Depressed Sternal Fractures. M. Kazuk.—p. 419.
Adenomatous Polyps of Rectum. J. T. Crone Jr.—p. 422.
Unusual Case of Rupture of Uterus. H. Seidemann.—p. 425.
Health Aspects of Stream Pollution in Ohio. J. D. Porterfield.—p. 427.

Surgical Difficulties from Goitrogenic Action of Antithyroid Drugs.—DeCourcy believes that the goitrogenic action is the most serious side-effect of noniodinated antithyroid drugs. The gland, which may already be enlarged, grows still larger in most patients treated with propylthiouracil, methylthiouracil, or methimazole ("tapazole"). The gland becomes quite vascular, and the already great risk of hemorrhage during thyroidectomy is increased by the friability of such a gland. Consequently, the use of strong iodine (Lugol's) solution for 10 or 20 days in preoperative preparation has been indispensable to counteract partially the goitrogenic action of the antithyroid drugs, but it is not certain whether an antithyroid drug should be given with the iodine. DeCourcy stresses iodine's beneficial effects and his conviction that most patients with hyperthyroidism should receive only strong iodine solution in preparation for operation. In almost all hyperthyroid patients iodine administration induces such changes as the following: Tremor is diminished. Tachycardia and palpitation decrease as the heart beat becomes less forceful. Vasomotor instability is replaced by normal reactions. The tendency toward hemorrhage during or after operation vanishes. All these changes are accomplished within 10 days to 2 weeks. The patient is not weakened by prolonged operation or loss of blood and thyroidectomy becomes less hazardous, in contrast to the difficulties and risks of operation after the use of goitrogenic drug plus iodine.

Oklahoma State Medical Assn. J., Oklahoma City

45:125-162 (April) 1952

Present Day Methods Concerned with Relief of Intractable Pain. M. P. Capehart Jr.—p. 127.
Present Day Use of Antihistamines with Special Reference to Use of Chlorcyclizine Hydrochloride (Perazil). V. D. Cushing.—p. 131.
Socialism and Psychopathy. C. H. Campbell and H. G. Sleeper.—p. 135
The Good Country Doctor. K. E. Jones.—p. 139.

Pediatrics, Springfield, Ill.

9:387-518 (April) 1952

Effect of Apnea Neonatorum on Intellectual Development. G. L. Usdin and M. L. Weil.—p. 387.
Unipolar Lead Electrocardiography in Children, with Special Reference to Its Value in Congenital Heart Disease. L. G. Veasy and F. H. Adams.—p. 395.
Q-T Interval During Active and Inactive Rheumatic Fever. S. Finkel and J. S. Baldwin.—p. 410.
Sulfobromophthalein Sodium Excretion Test as Measure of Liver Function in Premature Infants. W. Obrinsky, M. L. Denley and R. W. Brauer.—p. 421.
Report of Two Years' Experience in Transportation of Premature Infants in New York City. H. M. Wallace, M. A. Losty and L. Baumgartner.—p. 439.
*Duplications of Alimentary Tract. R. E. Gross, G. W. Holcomb Jr. and S. Farber.—p. 449.
Studies on Abnormal Hemoglobins: IV. Persistence of Fetal Hemoglobin in Erythrocytes of Normal Children. A. I. Chernoff and K. Singer.—p. 469.
Adrenal Cortical Hyperfunction in Childhood: Report of Case with Adrenocortical Hyperplasia and Testicular Adrenal Rests. E. K. Rose, H. T. Enterline, J. E. Rhoads and E. Rose.—p. 475.
Effects of Early Feeding of Strained Meat to Prematurely Born Infants. M. B. Andelman, P. S. Gerald, A. C. Rambar and B. M. Kagan.—p. 485.

Duplications of Alimentary Tract.—The term "duplication of the alimentary tract" is preferred by the authors because it simplifies the classification of these malformations, which are found in many different locations. In the literature these anomalies are designated by such terms as "enterogenous cysts," "giant diverticula," "ileum duplex," "thoracic cysts of enteric origin," "bronchogenic cysts," and "unusual Meckel's diverticula," apparently because each was believed to be a separate entity. It is the authors' belief that all alimentary tract duplications have a similar origin, regardless of their location. Duplications are spherical or tubular structures that have a smooth muscle layer and are lined with mucous membrane; they are found at any level from the tongue to the anus, and usually are intimately attached to some portion of the alimentary tube. From 1928 through 1950, 67 patients with a duplication of some portion of the alimentary tract, including one with two lesions, were seen at Children's Hospital in Boston. Although 23 of these cases were reported previously, all of them are listed in this study. The fact that this many cases have been observed in one hospital seems to indicate that these abnormalities must occur often enough to warrant the attention of the pediatrician, roentgenologist, and surgeon so that they can be recognized and treated. They should not be confused with mesenteric lymphatic cysts or anomalies arising from a Meckel's diverticulum. Symptoms produced by these lesions usually vary according to their location, their size, and the type of fluid secreted by their mucosa. Duplications should almost invariably be removed, even though they are incidentally discovered and have not as yet given rise to symptoms. The mortality rate in cases treated prior to 1940 was high, but in the 20 cases treated in the last four years there have been no deaths.

Postgraduate Medicine, Minneapolis

11:267-370 (April) 1952

Follow-Up Report of a 5 Year Old Sexually Precocious Boy 20 Years After Removal of Malignant Adrenal Cortical Tumor. H. Lisser and L. Player.—p. 267.
Successful Surgical Removal of Adrenal Cortical Adenoma Causing Virilism for 30 Years. R. F. Escamilla and S. G. Johnson.—p. 272.
Adrenal Cortical Carcinoma with Excess Androgen Production in an Adult Man. W. J. Kerr and G. S. Gordan.—p. 278.
Successful Removal of Benign Androgenic Adrenal Tumor from a Diabetic Woman. C. I. Hamilton Jr. and H. C. Shepardson.—p. 284.
Pheochromocytoma: Report of Case with Symptoms Simulating Acute Anxiety Attacks. R. H. Crede and W. J. Kerr.—p. 288.
Renal Cyst Simulating Pheochromocytoma. J. C. Weaver, N. Kawata and F. Hinman Jr.—p. 294.
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Id.: II. Review of Cases Reported for 20 Year Period 1930-1949, Inclusive. E. Rapaport, M. B. Goldberg, G. S. Gordan and F. Hinman Jr. —p. 325.

Proc. Soc. Exper. Biol. & Med., Utica, N. Y.

79:555-740 (April) 1952. **Partial Index**

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Effects of Intravenous Administration of Dextran on Renal Function. J. W. Fleming, W. H. Cargill and W. L. Bloom.—p. 604.
*Studies on Platelets: IV. Thrombocytopenic Factor in Normal Human Blood, Plasma, or Serum. M. Stefanini and J. B. Chatterjea.—p. 623.
Influence of Hemagglutinating Viruses on Tumor Cell Suspensions: I. Growth Inhibition and Reversal of Effect. A. E. Moore and L. C. Diamond.—p. 663.
Genetic Modification of Response to Spleen Shielding in Irradiated Mice. H. S. Kaplan and J. Paull.—p. 670.
*Salicylates and Experimental Beryllium Poisoning. A. J. Finkel and M. R. White.—p. 672.
Further Studies on Relationship Between Human Serum Cholinesterase and Serum Albumin. M. G. Levine and A. A. Suran.—p. 686.
Eosinopenia Produced by ACTH in Patients with Schizophrenia. H. H. Hiatt, W. S. Rothwell and M. K. Horwitt.—p. 707.
Alterations in Circulating Platelets Following Administration of Adrenocorticotropic Hormone. J. Lauridsen, J. S. Belko and R. Warren. —p. 709.
Intravenous Infusions into Human Subjects of Fractionated Coconut Oil Emulsions. B. G. P. Shafiroff, J. H. Mulholland and H. C. Baron. —p. 721.

Thrombocytopenic Factor in Normal Human Blood.—Sensitivity to normal plasma manifested by symptoms that have been grouped under the definition of "plasma transfusion reaction" has been found to occur consistently in paroxysmal nocturnal hemoglobinuria and, occasionally, in some types of blood dyscrasia and extensive carcinomatosis. Most of these patients exhibit some degree of thrombocytopenia. Transitory thrombocytopenia was observed by Stefanini and Chatterjea to be one of the characteristic features of plasma transfusion reaction. Further studies of the specificity of this finding revealed that temporary but significant reduction in blood platelets usually followed transfusion of compatible blood, plasma, or serum of normal donors into normal recipients or nonthrombocytopenic patients. In almost every case no evidence of clinical reaction occurred at the time of the induced thrombocytopenia. Blood, plasma or serum were transfused into 36 compatible recipients with normal platelet count. The recipients had never been previously transfused. The donors with normal platelet level had been fasting for at least four hours prior to donation. Temporary but significant thrombocytopenia was noted in 32 of 36 persons receiving compatible blood, plasma, or serum from nonthrombocytopenic donors. No spontaneous bleeding manifestations, alterations of the various hemostatic mechanisms, changes in number or activity of the bone marrow megakaryocytes, alterations of function or morphology of the remaining platelets, agglutinating or lysing activity of the recipient's serum against normal platelets were noted. Heparinization of the recipient did not affect the thrombocytopenic effect of the transfusion of compatible plasma. The thrombocytopenic effect was apparently due to a component of plasma, stable at 56 C, not absorbed by Seitz filters nor by ion-exchange resins, but absorbed on $Ca_3(PO_4)_2$ gel, from which it could be eluted with sodium citrate solution. Work is in progress to establish the nature, the mechanism of action of this agent, and its relation to products of platelet destruction.

Salicylates and Experimental Beryllium Poisoning.—Acute beryllium poisoning in mice, produced by the intravenous injection of soluble beryllium salts, usually terminates fatally in three days. Death results from acute liver failure following hepatic focal and midzonal necrosis. Pathological changes have also been observed in the kidney and spleen. Recently, aurintricarboxylic acid, a substance of relatively low toxicity, was found to be an effective antidote when it was administered to mice up to eight hours after they had received LD_{95} amounts of beryllium sulfate intravenously. This protection was attributed to its chelating and lake-forming properties. The present study was undertaken to ascertain if the salicylic acid portion of the aurintricarboxylic acid molecule would be as effective in reversing experimental beryllium intoxication. It was found that intravenously or intraperitoneally administered sodium salicylate in doses of 600 mg. per kilogram of body weight is an effective antidote for acute beryllium poisoning in mice when given up to eight hours after intravenous administration of beryllium sulfate in LD_{95} amounts. Salicylate analogues without the ortho-hydroxy, carboxylic acid grouping were generally ineffective.

Psychosomatic Medicine, New York

14:71-148 (March-April) 1952

Differential Emphasis in Somatic Response to Stress: Experimental Study. J. I. Lacey and R. VanLehn.—p. 71.
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Psychiatric Observations in Patients Treated with Cortisone and ACTH. S. Brody.—p. 94.
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Effect of Incomplete Information on Estimating Prevalence of Disease. A. P. Iskrant and Q. R. Remein.—p. 384.
New Media for Differentiation of Enteric Bacteria. H. Colichon.—p. 401.

Review of Gastroenterology, New York

19:261-338 (April) 1952

SYMPOSIUM ON PEPTIC ULCER

Medical Aspects of Peptic Ulcer with Brief Clinical Reports on Banthine, Prantal and Uroanthelone (Kutrol). D. J. Sandweiss and M. H. Sugarman.—p. 271.
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Gynecic Endocrine Factors in Relation to Alimentary Tract Function and Abdominal Symptomatology. A. E. Rakoff.—p. 317.

Rocky Mountain Medical Journal, Denver

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Twenty-Five Dollars! J. D. McCarthy.—p. 419.
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Aortography in Roentgen Diagnosis. R. R. Lanier, R. V. Elliott and M. H. Levine.—p. 427.
Recent Trends in Esophageal Surgery. P. Thorek.—p. 435.
Simple Method for Piercing Ears. R. B. Patterson.—p. 440.

Surgery, Gynecology and Obstetrics, Chicago

94:513-640 (May) 1952

Hematologic Response of Pregnant Woman to Intravenous Saccharated Iron Oxide. D. B. Nicholson and N. S. Assali.—p. 513.
*Late Results After Total Gastrectomy. W. H. Re Mine and J. T. Priestley.—p. 519.
Ligation of Inferior Mesenteric Artery at Aorta in Resections for Carcinoma of Sigmoid and Rectum. R. S. Grinnell and R. B. Hiatt. —p. 526.
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Elimination of Untoward Radiation Sequelae in Treatment of Carcinoma of Uterine Cervix. J. F. Nolan and L. Du Sault.—p. 539.
*Treatment of Aneurysms with Fibroplastic Agents: Experimental and Clinical Studies with Use of Sodium Dicetyl Phosphate. J. K. Berman and J. E. Hull.—p. 543.
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Reconstruction of Tracheobronchial Tree with Resection of Lung, Carina, and Lower Trachea: Experimental Study. R. W. Ehrlich, R. P. Meyer, C. B. Taylor and others.—p. 570.
Wound Healing: Heat Turbidity Index and Methylene Blue Reduction Time of Plasma. R. W. Kapp and D. T. Shaw.—p. 577.
Consideration of Present Status of Simple Suture in Treatment of Acute Perforated Gastroduodenal Ulceration. S. Mage and B. A. Payson. —p. 581.
Effect of Intravenous Administration of Meperidine upon Circulation of Man and upon Circulatory Response to Tilt. B. D. King, J. D. Elder Jr. and R. D. Dripps.—p. 591.
Roentgen Therapy of Carcinoma of Larynx. L. H. Garland and M. A. Sisson.—p. 598.
Penetrating Wounds of Heart: Report of 81 Cases. A. De L. Maynard, J. W. V. Cordice Jr. and E. A. Naclerio.—p. 605.
Malignant Polyps of Rectum and Sigmoid: Therapy Based upon Pathologic Considerations. E. R. Fisher and R. B. Turnbull Jr. —p. 619.

Results of Total Gastrectomy.—All cases in which total gastrectomy was performed and in which esophageal and duodenal tissue were observed in the surgical specimen at the Mayo Clinic in the years 1917 to 1948 were reviewed. Total gastrectomy was performed for a malignant lesion in 170 cases and for a benign lesion in 15 cases. In most of the latter cases total gastrectomy was performed for a suspected malignant lesion. Gastrointestinal continuity was established after removal of the entire stomach by anastomosing the end of the esophagus and a loop of the jejunum in 165 of the 185 cases. Esophagoduodenostomy was performed in 15 of the cases, and some other type of anastomosis such as a Roux-Y procedure was employed in the remaining cases. A "diffuse" lesion involving virtually the entire stomach was present in 50% of the 170 malignant cases. Regional lymph nodes were involved in 78% of cases, and extension of the malignant process to neighboring structures was found in 38% of the cases. Grading of the malignant lesions after the method of Broders revealed that grade 4 lesions comprised 66% of the series, 22% were of grade 3, 9% of grade 2, and only 3% of grade 1. Among the 15 benign lesions were 13 inflammatory ulcers, 4 of which were multiple. The operative mortality of total gastrectomy has declined from about 60% in 1917 to about 13% in the last five years. The technique of total gastrectomy, provided it is properly chosen and executed, seems to have little influence on the operative mortality. The authors start giving nourishment by mouth within 48 hours after operation, and feel that in past years they increased the diet too slowly after operation. Six patients, each of whom was subjected to total gastrectomy for a benign lesion, were followed five years after operation. Four of these were alive. Of 50 patients who underwent total gastrectomy for a malignant lesion of the stomach and were followed for 5 years, 9 were alive. Half of the patients who survived total gastrectomy for five years were able to perform their usual work. Half of the remaining patients were able to work part time. All required treatment for anemia. Approximately two-thirds of the patients who survived five years regained their preoperative weight.

Treatment of Aneurysms with Fibroplastic Agents.—In a previous report Berman had demonstrated that a solution of dicetyl phosphate in olive oil produced extensive fibroplasia without necrosis or toxic effects. The present report is concerned with use of this compound in patients who had aneurysms of arteriosclerotic or syphilitic origin. The compound forms an easily injectible suspension, which does not require heating. Experiments on rats indicated that solutions up to 10% destroy almost no tissue cells, and induce fibroplasia slowly. Experiments on dogs revealed that a 2% aqueous solution of sodium dicetyl phosphate may be injected periarterially without toxic effects and with resultant fibroplasia. A 2% aqueous solution of sodium dicetyl phosphate was prepared and autoclaved at 240 degrees, with 15 lb. (6.8 kg.) pressure for 20 minutes. The injections were made through 21 gage needles. Six patients who did not have dissection of the aneurysm wall showed clinical improvement following perisaccular injection of dicetyl phosphate. The material is nontoxic, and does induce fibroplasia equal to that produced by cellophane® wrapping. Perhaps it will be most useful as an adjunct to other modes of treatment, such as partial ligation and the introduction of wire as advocated in abdominal aortic aneurysms by Blakemore; or in conjunction with cellophane® wrapping, which may be used on the "free" sides of the aneurysm, the injection being used posteriorly or at points of attachment where dissection is hazardous. Aneurysms that cannot be treated safely by combined methods may be reinforced by the fibroplasia that results from injection of sodium dicetyl phosphate.

Texas State Journal of Medicine, Fort Worth

48:197-248 (April) 1952

Carcinoma of Thyroid Gland; Pathologic Survey of 47 Cases. C. T. Ashworth.—p. 201.
Malignant Tumors of Ovary. C. D. Fitzwilliam.—p. 205.
Malignant Tumors of Corpus Uteri: With Review of 48 Cases. W. O. Russell, W. L. Greene and J. A. Wall.—p. 211.
Carcinoma of Cervix. G. Turner and H. F. Heslington.—p. 216.
Squamous Metaplasia of Cervix Uteri: Follow-Up Study to Determine Its Malignant Potentialities. F. M. Townsend and V. A. Stembridge. —p. 220.
Diagnosis of Kidney Tumors and Cysts. R. E. Cone.—p. 223.

Western J. Surg., Obst. & Gynecology, Portland, Ore.

60:141-192 (April) 1952

Evaluation of Operations Used for Carcinoma of Large Bowel. W. W. Babcock and H. E. Bacon.—p. 141.
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Use of Dermal Graft to Supply Defect in Diaphragm. D. Metheny, V. O. Lundmark and T. Morcom.—p. 156.
Strength of Quadriceps Function After Patellectomy. D. H. O'Donoghue, F. Tompkins and M. B. Hays.—p. 159.
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Recognition and Treatment of Aberrant Breast Tissue, with Report of Cases. J. M. Roberts.—p. 175.
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Annales de Dermatologie et de Syphiligraphie, Paris

79:137-256 (March-April) 1952. Partial Index

Excretion of Thallium Acetate; Action of Dimercaprol (BAL). S. Lapière and Demey-Ponsart.—p. 137.
*Triad of Urethritis-Conjunctivitis-Arthritis. G. Daguet.—p. 149.
Ultrasonics in Dermatology. J. Meyer.—p. 161.

Syndrome Characterized by Urethritis, Conjunctivitis, and Arthritis.—Daguet discusses the syndrome characterized by urethritis, conjunctivitis, and arthritis, which had been termed Reiter's disease, Fiessinger-Leroy's urethro-oculo-synovial syndrome, Waelsch's amicrobic urethritis, or pseudogonococcic enteritis. The variety of symptoms makes [illegible] Urethritis usually appears first, but may be preceded occasionally by an enteritic phase. Within 10 days conjunctivitis may be the predominant feature, continuing for two weeks on an average. If arthralgia develops early, polyarthritis may continue for several months. Emphasis is laid particularly on the associated local manifestations, such as late cutaneous lesions consisting of keratoderma, cornified cones, and urticarious erythema, mucosal lesions involving the internal surface of the cheeks, and digestive disturbances, which may be insignificant or of dysentery or colitis type. The etiology of the syndrome remains doubtful, but there is evidence that ultramicrobes, such as the Chlamydozoa oculogenitale, "L" organisms, and common bacteria in a submicroscopic phase may play a part. Antibiotic therapy is the method of choice. An extensive bibliography covering all the papers published on the subject from 1916 to 1951 is appended.

British Journal of Radiology, London

25:225-280 (May) 1952

Röntgenologic Aspects of Meningiomas. E. P. Pendergrass and C. R. Perryman.—p. 225.
Actions of Radiations on Living Cells, 1946 and After. L. H. Gray.—p. 235.
Radiology of Auditory Ossicles. E. Samuel and C. Theron.—p. 245.
Cystourethrography in Women. H. Roberts.—p. 253.
Plasma I^{131} Index for Assessing Thyroid Activity. H. Blondal.—p. 260.
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Charcot's Arthropathy of Spine, with Note on Case. H. W. Holland.—p. 267.
Note on Determination of Half-Value Layers of Soft X Rays. M. T. Morrison and G. W. Reed.—p. 270.
Incorporation of Methionine S^{35} into Proteins of Jensen Rat Sarcoma Cells After Irradiation of Tumour. B. E. Holmes and L. K. Mee.—p. 273.

British Journal of Tuberculosis, London

46:81-130 (April) 1952

Differential Diagnosis in Children of Pulmonary Manifestations of Primary Tuberculosis and Its Immediate Sequelae. L. G. Blair.—p. 83.
Diagnosis and Treatment of Bronchial Carcinoma in Presence of Active Pulmonary Tuberculosis: Report of 14 Cases. W. H. Helm and A. J. Moon.—p. 85.
Cancer of Lung: Laryngeal and Pharyngeal Syndrome as First Presenting Symptom. N. Asherson.—p. 95.
Observations of Bronchial Arteries in Lobar Pneumonia and Pulmonary Infarction. L. Cudkowicz.—p. 99.
*Pathogenesis of Tuberculosis of Female Genital Organs. S. G. Clayton.—p. 103.
Symptomless Circumscribed Intrathoracic Opacities: Analysis of 56 Consecutive Cases. G. Cruickshank.—p. 109.
Natural Resistance to Multiple Tuberculous Lesions: Report of Case. G. A. O'Malley.—p. 115.

Tuberculosis of Female Genitalia.—Endometrial biopsy revealed that about 5% of sterile women have tuberculous endometritis. Most of these women feel well and have no gynecological symptom except sterility. In one woman biopsy revealed tubercles in the endometrium, and injection of endometrial fragments caused tuberculosis in a guinea pig. A salpingogram showed that the left tube was blocked at the abdominal end. A radiograph of the chest showed no abnormality. She was treated with streptomycin and *p*-aminosalicylic acid. A year later she was well, and no tubercle bacilli could be grown. A second example shows that there may be no symptoms in more advanced cases of tuberculous salpingitis. This woman had had no significant illness. At laparotomy the left tube was found to be completely disorganized, and there was a small right pyosalpinx with tubercles on the adjacent peritoneum. Microscopic examination confirmed the diagnosis of tuberculosis. Recovery was uneventful after removal of the tubes. Such cases show that "silent" pelvic tuberculosis is an important problem. The author reviews some recently expressed opinions about the spread of tuberculosis to and from the pelvic organs, and includes observations based on 38 recent patients seen at his hospital, including 3 with active pulmonary tuberculosis and 6 with healed pulmonary lesions. If it is accepted that the usual site of initial infection of the genital organs is the tubal mucosa, it still remains to be explained how the tubercle bacilli get to these sites. As to whether the tube is infected from the peritoneum or the peritoneum from the tube, it is probable that both events occur. All evidence is against the possibility that infection ascends from the lower genital tract. Concurrent genital and urinary tuberculosis is uncommon in women, one investigator finding only 4 cases of urinary tuberculosis in 200 cases of tuberculous salpingitis. Among the cases studied for this paper no instance of urinary tuberculosis was found, although every patient with urinary symptoms was studied thoroughly. This rarity of association of genital and urinary tuberculosis is probably explained by the fact that in the female the urinary tract is less closely related to the genital organs than in the male. While studies of patients with infertility due to genital tuberculosis has shown that for the most part they remain in good health without any treatment, all patients for whom this diagnosis is made should be followed.

British Medical Journal, London

1:881-932 (April 26) 1952

*Surgical Treatment of Chronic Ulcerative Colitis. W. R. Gabriel.—p. 881.
Treatment of Septic Abortion. C. W. F. Burnett.—p. 886.
Post-Mortem Diagnosis of Air Embolism by Radiography. J. D. Taylor.—p. 890.
*Prolonged Anticoagulant Therapy with Heparin. A. Wynn, J. F. Goodwin and A. Birbeck.—p. 893.
Mitral Valve Disease in Light of Surgical Treatment. G. Bourne.—p. 896.
Chronic Mercury Poisoning in Latent Finger-Print Development. G. Forbes and J. White.—p. 899.
Pilonidal Sinus of Axilla. I. Aird.—p. 902.
Acrylic Replacement After Segmental Resection of Bone. J. M. P. Clark and A. C. Bingold.—p. 903.

1:933-986 (May 3) 1952

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Carcinoma of Oesophagus: Diagnosis and Surgical Treatment of 159 Cases. R. H. Franklin and J. J. Shipman.—p. 947.
Cancer of Hypopharynx and Its Surgical Treatment. R. W. Raven.—p. 951.
Note on Absolute Viability in Water of S. Typhi and Dysentery Bacilli. J. F. D. Shrewsbury and G. J. Barson.—p. 954.
Case of Myositis Ossificans Progressiva. R. H. Maudsley.—p. 954.
Case of Glandular Fever with Cerebral Complications. I. M. Librach.—p. 956.

Surgical Treatment of Chronic Ulcerative Colitis.—Failure of medical treatment, roentgenologic and sigmoidoscopic evidence of pseudopolyposis with the possibility of malignant change, and arthritis are the most important indications for surgical treatment of chronic ulcerative colitis. The best surgical technique is ileostomy and colectomy. A three stage operation for total colectomy is recommended. A terminal ileostomy, which is best performed through a short right transrectus incision, is followed by a subtotal colectomy three to four weeks after a satisfactory functioning ileostomy has been established, and this is followed by a total colectomy three to six months later. The autho operated on 68 patients with this disease during the last 1 years and used this three stage operation in 50 of them. Of th 50 who had ileostomy (mortality 6%), 32 subsequently ha subtotal colectomy (mortality 6.25%), and of these 21 had tot colectomy (no mortality). Although the condition of many these patients was serious when surgery was begun, the progre sive improvement in weight and well-being as the various stag were carried out was remarkable. Carcinoma developed after t operation in six patients in whom the disease had been prese for an average of 19 years. Colostomy in cases of segmen colitis is not indicated, since the disease will spread sooner later. Cecostomy is equally ineffective. One stage ileostomy and colectomy is indicated in patients with pseudopolyposis and in those in whom repeated blood transfusion cannot replace the persistent blood loss. The value of vagotomy as a surgical treatment for this disease is doubted. Ileo-anal anastomosis, appendicostomy, and other operations aiming to preserve the rectal function, although temporarily successful, may be easily followed by relapse and involvement of the remaining rectal segment.

Prolonged Heparin Anticoagulant Therapy.—Thirty-seven patients with thromboembolic diseases were treated with heparin for as long as five weeks. The medication was given intravenously to nine patients every four hours in doses of 50 to 75 mg. for 3 to 14 days by an indwelling needle with a Gorth adapter. Doses of 150 mg. in the form of a concentrated aqueous solution containing 250 mg. of heparin per milliliter were given intramuscularly every 12 hours for 4 to 42 days to 23 patients. Five patients received heparin by both routes. Coagulation times were estimated one hour and four hours after the intravenous and the intramuscular injections, respectively, and again before the next dose was given. In most patients of the group receiving heparin intravenously the maximum clotting times were prolonged temporarily to from 16 to 60 minutes. The minimum clotting time was almost normal at the end of four hours. Local infection around the indwelling needle limited the duration of therapy. In the patients who received heparin intramuscularly the maximum clotting time was reached in 3 to 4 hours and the minimum clotting time was maintained at approximately twice the preheparin level for 9 to 10 hours. Firm pressure applied locally after the injection reduced the tendency to bruising. Painful nodules and hematomas occurred mainly when the dose was given oftener than every 12 hours, when it exceeded 150 mg., when the minimum clotting time was maintained above 20 minutes, and when the patient had high venous pressure and loose inelastic connective tissues. In seven patients the therapy had to be discontinued. One patient died after recurrent myocardial infarction, and another had a mild recurrent infarction despite adequate prolongation of the clotting time. The drug rather than the method of administration may have been ineffective in these cases. The other 35 patients did not have further thromboembolic incidents. Since in acute phases of thromboembolic incidents intravenous injection of heparin is more effective than intramuscular injection, it is suggested that the medication be given intramuscularly only in a later stage of these diseases.

Canadian Medical Association Journal, Montreal

66:419-528 (May) 1952

General Practitioner and Undergraduate Medical Teaching. G. E. Hall.—p. 419.
Combined Antibiotic Therapy. H. Z. Pomerantz.—p. 424.
Hepatitis in Infectious Mononucleosis. A. K. Mathisen.—p. 426.
More Glomerular Changes in Diabetics. H. J. Barrie, C. L. Aszkanazy and G. W. Smith.—p. 428.
Study on Rupture of Collateral Ligaments of Knee Joint. W. W. D. Williams.—p. 432.
*Pulmonary Resection for Bronchiectasis. J. F. Paterson.—p. 433.
Acute Purulent Meningitis In Childhood. C. S. Anglin, G. A. McNaughton and N. Silverthorne.—p. 435.
Hereditary Transmission of Physical Defects in EMB Family. E. R. Selby.—p. 439.
Effect of Adrenal Steroids on Eosinophils. R. D. T. Cape, J. W. Thomas and R. A. Palmer.—p. 441.
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Late Results of Pneumothorax Therapy. J. H. Rubin and H. E. Burker.—p. 447.
Eczema of Hands. F. E. Cormia.—p. 451.
Pan-Hypopituitarism in Male Following Pituitary Apoplexy (Treated with ACTH and Cortisone). L. N. Roberts, W. M. Goldberg and H. Pickard.—p. 458.
Hexachlorophene (G 11) in Surgical Scrub. Brushless Surgical Wash. H. Cleland.—p. 462.
Clinical Use of Exfoliative Utero-Vaginal Cytology. A. A. Earn.—p. 465.
Economic Organization of Anaesthesia Service in Hospital. E. Allard.—p. 468.
Ulcerating Lesions of Stomach. A. W. Hardy.—p. 472.

Pulmonary Resection in Bronchiectasis.—Of 118 patients who underwent complete or partial lobectomy for bronchiectasis between 1941 and 1949, a total of 104 could be followed up. Of the 104, 58 were satisfied with the results, 24 were partly satisfied, and 22 were dissatisfied. Thirty-eight of the last two groups were investigated. Four are dissatisfied because they have pain in the chest beneath the scar, coming on usually after exertion. Three are dissatisfied because thoracoplasty has been necessary for empyema following operation. Two are dissatisfied because they have pulmonary tuberculosis. Sixteen patients still have bronchiectasis, including nine who have residual lingular bronchiectasis, following left lower lobectomy, that was present before operation. One patient has residual right middle lobe bronchiectasis following right lower lobectomy. One has residual bronchiectasis of the anterior segment of the right upper lobe following right middle and lower lobectomy. In four patients there is residual contralateral bronchiectasis. One patient has bronchiectasis of the posterior subsegment of the left apicoposterior segment. Fourteen patients complained before and after operation of recurring attacks of what they described as "chest colds." These were attacks of cough with mucoid sputum, with malaise, shortness of breath, and wheezing. The author believes that these 14 men had both bronchiectasis and recurrent bronchitis. Four still have both diseases, but 10 have only recurrent bronchitis. The points Paterson wishes to stress are: First, the importance of complete mapping of the bronchial tree before resection. Second, the importance of being certain that the lingular segment has been properly mapped and that it is free of disease before a left lower lobectomy alone is done. Third, when it is known that residual bronchiectasis will be present, the patient should be warned that the operation may not relieve his symptoms completely. Fourth, if the patient has both bronchiectasis and recurrent bronchitis, removal of the bronchiectatic segment may not cure the bronchitis. Despite occasional failures, resection is an excellent operation and is the only means of permanent cure.

Chirurg, Heidelberg

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W Type Hernia: Clinical and Prognostic Peculiarities. E. Melchior.—p. 198.
Treatment of Tetanus with Repository Tubocurarine. F. Stein.—p. 199.
Repository Curare in Therapy of Tetanus. S. Fackert,—p. 202.
*Therapy and Prophylaxis of Postoperative and Traumatic Circulatory Collapse and of Hypotension Associated with Spinal Anesthesia: Preliminary Report. J. Feuerstake and F. Koenig.—p. 205.

Therapy and Prophylaxis of Postoperative and Traumatic Circulatory Collapse.—L-arterenol by intravenous drip was employed successfully by the authors in treatment of 21 patients with postoperative and traumatic circulatory collapse. Six patients are reported on in detail. One 18-year-old patient had a comminuted fracture of the pelvis, one 54-year-old patient had been subjected to laminectomy for herniation of a nucleus pulposus, and four between the ages of 39 and 67 with arthritis deformans had been subjected to plastic operations on the head of the femur or on the acetabulum. Within two minutes after initiation of the intravenous drip of L-arterenol the blood pressure could be measured distinctly. Measurements of blood pressure and pulse were taken every three to five minutes. The blood pressure could be regulated by increasing or reducing the rate of flow of the drug solution. Graphic recordings of the blood pressure revealed rise of the systolic as well as of the diastolic blood pressure; consequently the mean pressure was increased. This is the most important indication of adequate peripheral blood perfusion. The rise of blood pressure was regularly associated with relative bradycardia, which may be interpreted as being due to central reflex regulation designed to diminish the load on the heart. Within a short time of application of the L-arterenol, respiration became quiet and deep. Hypotension during spinal anesthesia could be prevented by giving L-arterenol by intravenous drip at the onset of anesthesia. The therapeutic advantages of L-arterenol over epinephrine are its lower toxicity and its purely vasoconstrictor effect. Untoward side-effects were not observed.

Deutsche medizinische Wochenschrift, Stuttgart

77:573-604 (May 2) 1952. **Partial Index**

*Experimental Chemotherapy of Tuberculosis (Isonicotinic Acid Hydrazide). G. Domagk, H. A. Offe and W. Siefken.—p. 573.
*Treatment of Tuberculosis with Isonicotinic Acid Hydrazide. P. Klee.—p. 578.
Myalgia. K. Voit and A. Gamp.—p. 581.
Phlebography and Venous Pressure in Varicosities of the Lower Extremities. F. Kaindl, W. Lindemayr and B. Thurnher.—p. 587.

Chemotherapy of Tuberculosis.—After discussing the use of thiosemicarbazone (amithiozone), streptomycin, and *p*-aminosalicylic acid in the therapy of tuberculosis, Domagk and associates describe in vitro and animal experiments with isonicotinic acid hydrazide and its derivatives. The studies indicate that these drugs have a greater inhibiting effect on tubercle bacilli than *p*-aminosalicylic acid and streptomycin. They are about 100 times as potent as streptomycin and at least 10 times more effective than the most active thiosemicarbazones. Furthermore, the isonicotinic acid hydrazides have this effect even on strains of bacilli that have become resistent to *p*-aminosalicylic acid, streptomycin, and the thiosemicarbazones. Animal experiments also indicate that the hydrazide of isonicotinic acid is more effective in tuberculosis than any other drug.

Clinical Results of Use of Isonicotinic Acid Hydrazide in Tuberculosis.—Humans have great tolerance for isonicotinic acid hydrazide. The optimal daily dose is from 10 and 15 mg. per kilogram of body weight, that is, from 0.5 to 0.9 gm. daily for adults. This quantity is divided into four (usually 0.2 gm.) doses, which are given orally after meals. Variations of this dosage may be necessary, because of cumulative effects. Treatment was usually discontinued for 3 or 4 days following the first 10 days of treatment, when *p*-aminosalicylic acid may be given. In some cases, however, treatment with isonicotinic acid hydrazide was continued for three months without interruption. Dosage should be such that an effective concentration is attained in the blood. Although parenteral administration is not absolutely necessary, the drug is available in a 2% solution for intramuscular injection, and is tolerated without irritation. This 2% solution can also be given intrapleurally; in tuberculous empyema or secondary serous pleurisy 5 cc., equal to 100 mg., can be introduced. In tuberculous meningitis from 1 to 2.5 cc. of a 2% solution, which is equal to 20 to 50 mg., can be dissolved in 10 cc. of cerebrospinal fluid and injected intraspinally. No undesirable secondary effects were observed in adults with this form of intrathecal administration; in children further experiments are necessary. Inhalation of a 1% solution or introduction through Monaldi's tube into cavities and fistulas also proved valuable. Undesirable secondary effects are slight in view of the good tolerance to this drug. Of 126 patients in whom the drug was used, 61 had been treated for from six weeks to five months. More than two-thirds of these 61 patients showed rapid improvement and others remained about the same. Increase in appetite and in weight was observed in almost all patients. Forty-five of the 61 patients had fever at the onset of treatment. In 32 the fever subsided rapidly, but in the other 13 subfebrile temperatures persisted. The sputum became negative in 15 of 58 patients who had bacilli in the sputum at the beginning of treatment. Cavities, which were present in 40 patients, disappeared in four without local intervention or collapse therapy; in 18 considerable shrinkage of the cavities was noted. Roentgenoscopy revealed some regression of foci in 14 of the 61 cases. Isonicotinic acid hydrazide effected improvement in some patients who had not responded to streptomycin, *p*-aminosalicylic acid, and amithiozone. High bacteriostatic potency combined with good tolerance and low dosage are the advantages of this drug.

Giornale di Clinica Medica, Parma

33:105-228 (Feb.) 1952. **Partial Index**

*Tuberculosis of Lateral Cervical Lymph Nodes: Clinical and Pathogenic Study. B. Lorenzini and V. Maddaloni.—p. 105.
*Hepatosplenography with Ethyl Di-Iodostearate. A. Baronchelli and L. Rossi.—p. 150.

Pathogenesis of Tuberculosis of Cervical Lymph Node.—From 1945 to 1951 the authors studied 93 patients with tuberculosis of the lateral cervical lymph nodes in an attempt to determine whether this infection is lymphogenous or hematogenous in origin. Only six of these patients had a history of lesions of the skin and mucous membranes (conjunctivitis, otitis, or tonsillitis) that are important factors in lymphogenous infection of the cervical lymph nodes. Roentgenograms revealed an old tuberculous process in 64 patients, in 48 of whom it was localized in the respiratory system. At admission these patients were in good general condition, with a slightly increased erythrocyte sedimentation rate, mild hypochromic anemia, strong positive reaction to tuberculin, and mild elevation of temperature in the evening. Lymph node biopsy revealed caseous necrosis in some patients, productive processes, and exudative and inflammatory processes in others. Disseminated micronodular pulmonary tuberculosis of recent origin was present in 14 patients who had intermittent fever with profuse sweating, mild enlargement of the liver and spleen, great increase of the erythrocyte sedimentation rate, pronounced hypochromic anemia, and a negative or very weak reaction to tuberculin. Biopsy revealed discrete or conglomerate tubercles. There is no doubt about the hematogenous pathogenesis of the disease in these patients, but the authors suggest that the disease may have the same origin in the other patients. According to them, if there are no lesions in the skin and mucous membranes, the hematogenous diffusion of tuberculosis to the cervical lymph nodes should be considered in all instances. Clinical, bacteriological, radiological examinations, biopsy and allergy tests, and the history are of great importance.

Hepatosplenography with Ethyl Di-Iodostearate.—A suspension of ethyl di-iodostearate in glucose solution was used as a contrast medium in hepatosplenography. This is an isotonic colloidal suspension of an organic iodine preparation with particles having a maximum diameter of 2 μg. If the diameter of the particles were greater than that of blood capillaries, the substance would produce severe embolism. Since it is 10 to 100 times less, the solution easily traverses the small arteriovenous peripheral and pulmonary capillaries and is retained in the liver and spleen for as long as six days. It is very opaque, painless, easily tolerated, and quickly eliminated. The patient's sensitivity should be tested by intradermal or intravenous injection of 2 cc. of the preparation. The total doses used by the authors ranged from 20 to 35 cc. There were no signs of intolerance to the drug when it was injected into the brachial artery of a patient with swelling of the right forearm, into the subclavian artery of a patient with swelling in the right supraclavicular fossa, and into the dorsal vein of the foot of a patient with varices in the left leg. With 20 cc. the opacity of the spleen was moderate, with almost no visualization of the liver, whereas with 30 to 35 cc. the spleen was clearly visible even in the presence of shadows attributable to pathological processes. The opacity was proportional not only to the amount of preparation but also to the velocity with which it was injected. Opacity in the spleen occurs about three minutes after the injection, increases progressively, is greatest 3 to 4 hours later, and disappears completely after 24 hours. Opacity of the liver occurs after 2 hours, is greatest after 5 to 6 hours, and disappears within 24 hours. Sometimes a slight opacification of the kidney and a late opacification of the gallbladder were obtained.

Helvetica Paediatrica Acta, Basel

7:105-228 (April) 1952. **Partial Index**

*Effect of Virus of Rubeola on Human Fetus. G. Töndury.—p. 105.
Axial Transversal Stratigraphy (Cross-Section Tomography) in Study of Congenital Cardiac Defects. R. Bulgarelli.—p. 156.
Effect of Measles on Tuberculous Meningitis Treated with Streptomycin. A. Akkoyunlu.—p. 180.
Chronic Granulocytopenia During Childhood. M. R. Vrtilek.—p. 207.

Effect of Rubeola Virus on Human Fetus.—Töndury applies Bamatter's term "embryopathia rubeolaris" to the defects observed in fetuses and infants of mothers who had rubeola during pregnancy. Although the clinical aspects of embryopathia rubeolaris are well understood, little is known as yet about the processes taking place in the embryo. Töndury was able to make anatomic studies on six fetuses with the typical signs of embryopathia rubeolaris whose mothers had had rubeola between the 35th and 51st day of pregnancy. At the time of interruption of pregnancy or delivery the gestation periods ranged from 68 to 276 days. The eyes, the labyrinth, and the dental structures were studied histologically. The observations indicate that the rubeola virus circulates in the maternal blood and reaches the fetal circulation by ultrafiltration without damaging the chorionic epithelium. It does not produce a systemic disease in the fetus, but attacks and exerts destructive effects only on certain organs, the lens, the epithelium of the internal ear and the enamel organs, that is, it has a decided organ affinity. The destructive process is characterized by swelling and vacuolization and finally there is nuclear pyknosis and the cytoplasm disintegrates into droplets. These destructive processes do not appear simultaneously in all three organs. Whereas the lens fibers show degenerative changes as early as 17 days after the outbreak of the disease in the mother, the cells of the enamel organ and of the inner ear show such changes only 222 days after the maternal rubeola. The author believes that the virus attacks the epithelial cells of the lens, the auditory, and enamel organs during the early embryonic period and remains and multiplies in these tissues. Its effects become evident, however, only after the cells reach a stage of differentiation that is accompanied by great metabolic activity. In this respect the action of the rubeola virus is phase specific. It interferes with metabolic processes in the cell and finally produces degeneration of the cytoplasm.

Hoja Tisiológica, Montevideo

12:1-110 (March) 1952. Partial Index

Angiopneumography in Pleuropulmonary Tuberculosis. R. Rimini and A. Rodríguez.—p. 1.
Diagnosis, Clinical Forms, and Treatment of Thoracic Actinomycosis. R. J. Pintos Fuentes.—p. 61.
Two Years of Dispensary Practice in a Department in the Interior. H. G. Cardoso.—p. 69.
*Tuberculous Morbidity and Mortality in BCG Vaccinated and Control Groups. R. G. Bacigalupi.—p. 73.

Tuberculous Morbidity and Mortality in BCG-Vaccinated Children.—The clinical histories of 4,986 children treated at the hospital of Dr. Pedro Visca between October, 1943, and October, 1951, showed that 2,804 (56.2%) had received BCG vaccination at birth; most of these came from modest homes and had the same living conditions as others who had not been immunized. The remaining 2,182 (43.7%), consisting chiefly of children who had never been immunized, included an unknown proportion who had been BCG vaccinated; this improves the morbidity and mortality indexes of the control group. There were 26 cases of tuberculosis with 6 deaths in the first group, and 93 cases with 41 deaths in the control group. Analysis of the figures showed 16 cases of tracheobronchial adenopathy, 6 cases of pulmonary and pleuropulmonary tuberculosis, and 4 cases of tuberculous meningitis in the vaccinated group; the corresponding figures for the control group were 35, 11, and 37, with 10 cases of miliary tuberculosis. A striking fact was the absence of tuberculous meningitis among the vaccinated children from September, 1946, to October, 1951; during the same period there were 21 cases among the controls. Disseminated and meningeal infections occurred more than 12 times as often among the nonvaccinated children as among the vaccinated. The mortality figures were probably somewhat higher than the hospital statistics given, because certain children who were removed in serious condition cannot have survived long. BCG vaccination evidently confers protection during the first years of life when tuberculosis is most serious; periodic revaccination until the end of the second year is apparently needed to maintain immunity.

Klin. Monatsbl. für Augenheilkunde, Stuttgart

120:337-448 (No. 4) 1952. Partial Index

Intermittent Exophthalmus and Blindness. L. Högel.—p. 346.
*Eye Changes in Congenital Heart Disease. M. Burk and F. Northoff.—p. 351.
Eye Changes in Malnutrition. H. F. Piper.—p. 357.
Eye Lesions Caused by Caterpillar Hairs. H. Geserick.—p. 373.
Relation Between Myopia and Corneal Scars. K. Rumpelhardt.—p. 397.

Eye Changes in Congenital Heart Disease.—Ophthalmologic studies were made on 74 patients with congenital cardiac defects, at a time when the exact nature of the defect had not as yet been ascertained. Later it was found that 27 had the tetralogy of Fallot, 15 had Eisenmenger's complex, 11 had patent ductus arteriosus, and 21 had various atypical deformities. The children with patent ductus arteriosus without cyanosis, also had no eye ground changes or polycythemia. However, children with the tetralogy of Fallot or symptoms of congenital cyanotic heart disease had eye changes such as cyanosis of the conjunctivas and retinas, erythema of the optic papillae, tortuosity, and dilatation and darkening of the retinal vessels, particularly of the veins. The degree of eyeground changes corresponded to the severity of the cyanosis and polycythemia. They are regarded as manifestations of compensation. The chronic oxygen deficiency characteristic of this form of heart disease is present also in persons at high altitude, and similar changes are observed in their eyes before acclimatization has taken place.

Lancet, London

1:831-884 (April 26) 1952

After 56 Years. J. Learmonth.—p. 831.
*Coeliac Disease: Gastro-Intestinal Studies and Effect of Dietary Wheat Flour. C. M. Anderson, A. C. Frazer, J. M. French and others.—p. 836.
*Nephrosis Treated by Malaria. D. Gairdner.—p. 842.
Malarial Therapy in Lipoid Nephrosis. E. A. J. Byrne.—p. 844.
Management of Respiratory Infections in Patients Severely Paralyzed by Poliomyelitis. F. H. Stevenson.—p. 845.
Cortisone and A.C.T.H. in Exophthalmic Ophthalmoplegia. G. N. Chandler and S. J. Hartfall.—p. 847.
Effect of Splenectomy in Case of Congenital Porphyria. C. H. Gray.—p. 851.

Celiac Disease: Effect of Wheat Flour.—Ten children with celiac disease, in whom the diagnosis was established by clinical, biochemical and radiological evidence, were studied while they were on a normal hospital diet. As children with celiac disease apparently show the same radiological changes in the small intestine and the same biochemical abnormalities as adults with idiopathic steatorrhea, it seems likely that they have the same defect of absorption in the upper intestine. On the normal hospital diet there was an increase in fecal fat, apparently because of an absorption defect. In these children fat was normally digested and prepared for absorption by fine emulsification, but absorption of particulate fat was delayed and depressed, as shown by the chylomicrographs. The glucose tolerance curves were also compatible with an absorption defect, which may possibly be due to excessive mucus secretion or lack of intestinal motility, as suggested by the radiological findings. Observations of Dicke and the later studies of Weijers and van de Kamer of the Netherlands in 1950, on the beneficial effect of excluding wheat and rye flour from the diet of celiac children, induced the authors of this report to try a wheat free diet. Such a diet produced striking clinical improvement and increased the absorption of fat. The radiographic appearances of the small intestine and the absorption of dextrose and particulate fat also became more normal, suggesting that all absorption had improved. The effect of reintroducing wheat flour or wheat gluten demonstrated that wheat causes deterioration in celiac children. Wheat starch does not have the same action as wheat flour, showing that it is not the starch that is harmful; in fact, children receiving a relatively large amount of starch improved. In the diets prescribed by previous workers, who reported that starches had a deleterious effect in celiac disease, removal of starches was synonymous with removal of wheat flour. The present studies indicate that there is no need to restrict intake of carbohydrates or even fat; choice of food may be almost unlimited, provided that wheat and rye flour are excluded.

Nephrosis Treated by Malaria.—It is known that in nephrosis (type 2 nephritis of Ellis) a remission may sometimes be initiated by such factors as infections, particularly measles, infusions of plasma, and administration of urea, salts such as potassium citrate, or corticotropin. Sometimes the remissions provoked by these means have proved long-lasting and amounted to cures. Since malaria can be easily induced and easily controlled, four patients with the nephrotic syndrome were infected with malaria. The first three of the patients had the nephrotic syndrome in its pure form, with normal or nearly normal blood pressure, no nitrogen retention, and only a few red cells or casts in the urine. In case 4 there was nitrogen retention, and red cells and granular casts were always numerous in the urine; the histological features of the kidney in this case were those of Ellis type 1 nephritis. Malaria induced a striking remission in two of the first three patients, who were still well three years and one year later. In the third child malaria produced no remission, but the plasma cholesterol level fell temporarily from a high to normal level; this response was also seen in the child with a nephrotic syndrome due to a type 1 nephritis. The author suggests that the effect of malaria in nephrosis may be essentially the same as that of corticotropin. In any case, since nephrosis, in its earlier stages at least, may be reversible, the physician should try the agents known to have initiated remissions, and malaria seems worth using in this manner.

1:885-934 (May 3) 1952

Structural and Functional Plasticity of Influenza Virus. W. Smith. —p. 885.
Subdural Effusions in Purulent Meningitis. H. E. Jones.—p. 891.
Daily Urinary 17-Ketosteroid Output of African Negroes. N. A. Barnicot and D. Wolffson.—p. 893.
Functional Disorders of Small Bowel After Gastrectomy and Vagotomy: Correlation of Radiographic and Kymographic Studies. A. J. Glazebrook.—p. 895.
Disinfectant Barrier in Dressings Applied to Burns. E. J. L. Lowbury and A. M. Hood.—p. 899.
Osteomyelitis of Skull Complicating Acute Frontal Sinusitis: Report of Two Cases. W. L. Thompson, J. V. Clark and J. A. Simons.—p. 901.
*Rupture of Testicle. C. Dundon.—p. 903.

Rupture of Testicle.—A man, aged 49, was kicked in the scrotum. When he was seen eight hours later, the scrotum was grossly swollen, and a diagnosis of bilateral hematoceles with a superficial hematoma was made. He had passed urine since the accident, and tests for hematuria were negative. With the patient under anesthesia an incision was made over the left hematocele, and multiple clots were removed. There was a longitudinal rupture of the tunica albuginea, with protrusion of testicular tissue. The tunica was closed with interrupted catgut sutures. Another incision was made over the right hematocele. Many clots were removed, but the testicle presented only a minor contusion. A drain was inserted into each hematocele, and the wounds were closed with catgut sutures. Penicillin and sulfadimidine were given for seven days. Convalescence was uneventful. Seven months later the left testicle was normal. At the time of the operation, the author had thought that the testicle would atrophy. Now he believes that such a prognosis is unwarranted, and recommends exposure and examination of the testicle in every case in which a hematocele follows trauma. If the testicle is ruptured, suture of the tunica albuginea should be attempted instead of orchidectomy.

Medical Journal of Australia, Sydney

1:425-460 (March 29) 1952

Epidemiology of Infectious Diarrhoea. S. D. Rubbo.—p. 425.
Bacteriology of Infectious Diarrhoea. R. Mushin.—p. 428.
Effect of Pneumothorax on Pulmonary Vital Capacity. A. H. Campbell. —p. 432.
Kaposi's Varicelliform Eruption Due to Vaccinia Virus. M. H. Slonim. —p. 434.

1:461-496 (April 5) 1952

Centenaries of 51. H. Sutton.—p. 461.
Effect of Hyaluronidase on Absorption of Heparin. P. Hagen.—p. 471.
Incidence of Retrolental Fibroplasia in Premature Infants in Sydney. E. H. Hipsley.—p. 473.
*Pain in Cardiac Infarction. J. A. Forbes.—p. 475.
Tuberculosis Mortality Rates of Native-Born White Australians and Migrants, 1910-1912, 1920-1922, 1932-1934 and 1946-1948. P. D. Abbott. —p. 476.

Pain in Cardiac Infarction.—The reports of 167 consecutive cases of cardiac infarction discovered at postmortem examination were correlated with the clinical histories, which were examined particularly for an account of the pain that might have been associated with the occurrence of the infarct. The term "recent" is applied to infarcts in which fibrosis was not apparent on macroscopic examination, their maximum age being about three weeks. Circumscribed scars are classified as old infarcts. Thirty-nine per cent of the subjects had had recent infarcts only, while in 26% the infarcts were healed. In the remaining 35% of cases, old and recent infarcts were recognizable in the same heart. Pain of cardiac origin (including angina of effort) was recorded in 53% of the patients, vague pain of doubtful origin in 12%, and no pain in the remaining 35%. This indicates that the proportion of cases of painless cardiac infarction is greater than is commonly believed. All subjects with thrombotic occlusion of a coronary artery gave a history of related cardiac pain, but these comprised a total of 19 subjects only. The occurrence of cardiac infarction a short time prior to death was usually accompanied by sudden circulatory failure, often indistinguishable from shock. The onset was often accompanied by sudden congestive cardiac failure, at times by repeated fainting attacks, and occasionally by coma comparable to that of a cerebrovascular accident. On the other hand, in some cases old infarcts were found in subjects who had given histories of angina of effort dating from an acute painless illness, at times called influenza, for which they had not consulted a physician, and in others the infarction had apparently been associated with symptoms mild enough to be considered unimportant by the patient.

Nordisk Medicin, Stockholm

47:411-442 (March 28) 1952. Partial Index

*Treatment of Wounds with Septofyllin with Special Regard to Effect on Chronic Ulcers of Leg. R. Carleson and P. Garsten.—p. 412.
Prolapse of Mucosa Through Pylorus as Surgical Problem. P. Castrén. —p. 416.
*Corticotropin Therapy in Psychoses. B. Korner and N. P. Jepsen. —p. 421.
Rupture of Lateral Ligaments in Ankle. S. Rosendahl-Jensen.—p. 423.
Differential Diagnosis Between Strangulated Ileus and Acute Expansive Gynecologic Disease. E. Klintskog.—p. 426.
Thrombosis of Renal Vein, Clinically Characteristic Syndrome. O. J. Broch.—p. 428.
Local Treatment of Cystitis Calcarea. P. Ahnfelt.—p. 432.

Treatment of Wounds with "Septofyllin."—"Septofyllin" (a water-soluble preparation of sodium, copper, and chlorophyll with a quaternary ammonium germicide added) has been used with gratifying results by Carleson and Garsten since 1951 in about 100 cases of ulcers, wounds, and burns. Improvement occurred in all but 1 of the 32 cases of chronic ulcers of the leg, of which 2 were due to frostbite, 5 were traumatic, and 25 were varicose and arteriosclerotic. The ulcers had been present from 2 weeks to 12 years. The treatment was applied for as long as two months. In some cases a week elapsed before improvement was evident. The deodorizing and antipruritic effect was obvious, the bactericidal effect was good, and macroscopically normal granulation tissue was seen. "Septofyllin" is an adjuvant to treatment, not a substitute for adequate surgical or medical therapy.

Corticotropin Therapy of Psychoses.—Eight psychotic patients were treated with corticotropin (ACTH) in daily doses of 40 mg., usually for 7 to 10 days. In the three cases of acute delirium the effect was good, and in two cases life-saving. In a mild manic psychosis the effect was doubtful. In the three cases o endogenous psychosis and in a psychosis of schizophrenic typ the treatment with corticotropin had no effect.

47:443-472 (April 4) 1952. Partial Index

*Anal Abscesses and Fistulas: Results of Treatment. O. Poulsen.—p. 44
Thoracoplasty with Supplementary Filling with Oil ("Oleoplasty" R. Holmboe.—p. 450.
Acromioclavicular Arthroplasty. I. Spak.—p. 454.
Glove Talc in Surgical Wounds and Abdominal Cavity. R. Brandber —p. 456.
Effect of Work in Ventricular Septal Defect With and Without Ove riding Aorta. E. Varnauskas, H. Eliasch and L. Werkö.—p. 457.
Anomalies in Internal Carotid Artery. O. Malver.—p. 460.

Anal Abscesses and Anal Fistulas.—A series of anal absces and fistulas treated in Bispebjerg Hospital from 1938 to 19 is reported on. In the 211 cases of anal abscess, treatment cc sisted of simple incision, with unroofing done in only one stance. Of 146 patients given a follow-up examination, 109 w well. Recurrence was reported in 33 cases, or 23.3%. Of the cases of anal fistulas 60% were treated by excision, 40% discission. Follow-up examination showed 69 patients to be w Recurrence was reported in 13 cases, or 15.9%. Poulsen s the frequency of recurrence clearly reveals that the more conservative methods of treatment are not so reliable as more radical methods. He concludes that effective treatment of anal abscesses requires broad incision with unroofing of the abscess and removal of the crypt, and that anal fistulas should be excised broadly with vertical division of the sphincter or use of silk ligature. Suture of the sphincter, skin, and mucous membrane is not advised, as it involves considerable danger of recurrence and does not lessen the danger of sphincter insufficiency.

Pediatria, Naples

60:1-111 (Jan.-Feb.) 1952. Partial Index

In Vitro Sensitivity of Some Species of Enterobacteria to Chemotherapy and Antibiotic Therapy. G. R. Burgio and A. Albeggiani.—p. 1.

*Follow-Up Study of Persons Vaccinated with Petragnani's Anatuberculin. M. Verde.—p. 13.

Study on Blood Fibrinolysis in Children. E. Grasso.—p. 27.

Antituberculosis Vaccination with Petragnani's Anatuberculin.—In 1938 children between the ages of 2 months and 9 years were vaccinated with Petragnani's anatuberculin. Many were malnourished, underdeveloped, syphilitic, rachitic, and dyspeptic. The vaccination was done by Salvioli's technique, which consists of four simultaneous punctures arranged quadrilaterally on the forearm. The total dose of anatuberculin ranged from 0.30 to 0.50 cc., according to the age, weight, and general condition of the child. Only 28 of the children were followed. In those who died while still at the clinic autopsy did not reveal a tuberculous process. In three children seen four to seven months after the vaccination there was no reactivation of the vaccinal focus and the cutaneous allergic reaction was negative. In the other 25 children the inflammatory reaction was reactivated from 20 days to 7 months later. There were no disturbances attributable to the vaccination, and some malnourished children even gained weight. In some children the allergic reaction was still positive after two years, although they had had influenza and whooping cough. In 1950, a clinical and radiological follow-up of six of these children did not reveal a tuberculous process, and the allergic reaction was still positive despite the unfavorable living conditions and the stress of the war. The author believes the antituberculous vaccination with Petragnani's anatuberculin according to Salvioli's technique had had a specific protective action in these children.

Philippine Medical Association Journal, Manila

28:105-176 (March) 1952

Prostatic Surgery in Cardiac Patients. D. Antonio Jr. and A. M. Samia.—p. 105.

Management of Bartholin Gland Abscess and Cyst. G. Sepulveda Jr. and L. M. Ibarra.—p. 115.

Management of Philippine Hospitals. E. F. Ochoa.—p. 121.

Combined Immunization Against Diphtheria, Pertussis, and Tetanus Using *Triple Toxoid—Follow-Up* of 1,286 Cases. R. Carretero and Fe del Mundo.—p. 141.

Policlinico (Pract. Sect.), Rome

59:369-404 (March 24) 1952

Antituberculous Therapy with New Derivatives of Nicotinic Acid. B. Besta and S. Valenti.—p. 369.

Abdominal Syndrome Caused by Bite of Spider Simulating Gastric or Duodenal Ulcer. G. Gennari.—p. 373.

*Galactorrhea Following Thoracoplasty. A. Mazzarella and F. Picconi.—p. 381.

Galactorrhea After Thoracoplasty.—Nonpuerperal lactation is reported in a 34-year-old married woman after right thoracoplasty for cavitary pulmonary tuberculosis. The postoperative course was normal, but after two weeks mammary secretion began and persisted for about two months. The mechanism of postoperative lactation is not known. Several hypotheses have been advanced. During pregnancy the production of estrogen and progesterone is increased and breast development proceeds under the stimulation of these steroid hormones derived from the placenta. The onset of lactation following pregnancy is associated with secretion of prolactin by the pituitary and with the postpartum fall in estrogen and progesterone levels that follows removal of the placenta. The maintenance of lactation depends on an afferent neurogenic impulse originating in mechanical sensory stimulation due to suckling, which in turn stimulates the hypothalamus and the pituitary to cause continued prolactin secretion. Probably the most important factor associated with lactation after thoracoplasty is continuous stimulation of the neurogenic component of the lactation mechanism and consequent release of pituitary prolactin due to mechanical injury and inflammation of the thoracic nerves during the operation. The inflammatory stimulation of the cut nerve ends may be regarded as equivalent to the stimulus derived from the suckling reflex. Some authors explain lactation after thoracoplasty on the basis of the alarm reaction of Selye. In this report the authors review 14 cases of galactorrhea reported in the literature.

Prensa Médica Argentina, Buenos Aires

39:229-274 (Feb. 8) 1952. Partial Index

Penicillin Therapy and Syphilis. Comparative Clinical Study. M. I. Quiroga, J. O. Agneta, J. Chiriboga and V. Hahn.—p. 229.

Indications for Vioform in Dermatology. Clinical Results Obtained in 374 Patients. E. E. Tello.—p. 242.

Kartagener's Syndrome. Case Report. R. Dassen and D. Gotlieb.—p. 249.

*Carbuncle of the Kidney. E. Ayas, P. Maslo and I. Elkouss.—p. 252.

Hamartoma of the Liver. O. F. Longo, J. César and J. Aguirre.—p. 258.

Renal Carbuncle.—The existence of a renal carbuncle may be suspected when a patient who has recently had furuncles or some other staphylococcic infection has evidence of severe infection, with signs of localization in the kidney. Renal carbuncle should be especially suspected if the kidney is enlarged and painful to the touch, there is a diminished kidney function paralleling in degree the extent of the disease, and if the urine shows no abnormality other than a slight albuminuria (resulting from toxicity). Pyelography is a valuable diagnostic aid; the images shown are those accompanying renal tumors, including compression, displacement, deformation, or amputation of the calyces, and sometimes lacunae or cavities caused by filling of necrotic areas. Symptoms such as muscular contraction, intense local pain, and those resulting from psoitis relate more to perinephritic phlegmon, which almost always complicates the condition, than to the renal carbuncle itself. Perirenal abscess is usually posterior, between the kidney and Gerota's fascia, and if untreated will perforate the fascia and lead to infection of the retroperitoneal tissues. An anterior perirenal abscess will perforate the peritoneum, causing acute abdominal symptoms. Renal carbuncle and perirenal abscess must be differentiated from various conditions with similar symptoms, such as retroperitoneal and subphrenic phlegmon and abscesses, and renal tumors. Nephrectomy, drainage, and enucleation of the carbuncle each have their advocates; no single treatment will be satisfactory in all cases. Antibiotics will often cure mild carbuncles diagnosed early; others may be successfully treated by enucleation or partial resection. Drainage of perirenal phlegmon is usually insufficient when there is an underlying carbuncle; in any case, it should be followed by renal decapsulation. Nephrectomy should be done in cases in which the kidney is extensively involved, or the patient's condition disquieting.

39:421-476 (March 7) 1952. Partial Index

*Shoulder-Hand Syndrome in Coronary Disease. A. Navarro.—p. 421.

Nailing of Humeral Head Fractures. O. Voss.—p. 428.

Esophagopulmonary Fistula from Calcified Tuberculous Adenopathy. Carlos Rey, A. Rosenfeldt and P. Rubinstein.—p. 433.

Monilial Vulvovaginitis. G. di Paola and E. Vásquez Ferro.—p. 440.

Hypertensive Supramalleolar Ulcer. A. S. Benchimol.—p. 444.

New Reflexes of Gluteal Region. J. Dalma.—p. 449.

Shoulder-Hand Syndrome in Coronary Disease.—The massive swelling and severe pain of the shoulder-hand syndrome may not appear until some time after the causative coronary accident and may, as a result, be attributed to local conditions such as arthritis of the shoulder or hand, scapulohumeral periarthritis, or phlegmon of the hand. The underlying heart disease may be unrecognized because its symptoms resemble those of gastric or hepatic diseases. The arm on the affected side is held in a sling position and even slight movement increases the pain, which is not relieved by nitrites but is lessened by acetylsalicylic acid (aspirin) and similar substances. The local symptoms are caused by vasomotor reactions of the skin, edema of an inflammatory type in the subcutaneous cellular tissues, muscular atrophy and contracture, metacarpal and interphalangeal arthritis, and osteoporosis of the epiphyses. Complete cure takes place in some cases; in others the condition becomes chronic, with exacerbations depending on the underlying coronary disease. Treatment of the underlying disease and cervicodorsal radiotherapy (from the fourth cervical to the second dorsal vertebrae) proved beneficial in the author's cases.

Presse Médicale, Paris

60:509-528 (April 9) 1952. Partial Index

New Method of Biological Evaluation of Urinary 11-Oxysteroids. F. Coste, F. Delbarre and F. Laurent.—p. 509.

Modifications of Chemical Activity of Stomach in Patients with Gastric and Duodenal Ulcers; Their Diagnostic Value and Pathogenic Significance. A. Lambling and J. Zahar.—p. 513.

*First Trials of Transfusion of Leukocytes and Platelets Labeled with Radioactive Phosphorus in Man. J. Julliard, B. Maupin, A. Loverdo and others.—p. 518.

Transfusions of Leukocytes and Platelets Labeled with Radioactive Phosphorus.—Leukocytes obtained from normal blood were labeled by being brought into contact with radioactive phosphorus for two hours. A technique of rapid separation in siliconized flasks permitted completion of the entire procedure of collecting, separating, and labeling in six hours. A transfusion of the labeled leukocytes was then given to a man with chronic lymphoid leukemia and an equivalent amount of radioactive phosphorus in an inorganic solution was administered intravenously to another patient with chronic myeloid leukemia. The transfused leukocytes could be traced in the circulating blood of the recipient, and results showed that the radioactive phosphorus bound to the leukocytes disappeared much more rapidly from the circulating blood than the free radioactive phosphorus. Rabbits were then given intravenous injections of human leukocytes labeled in vitro with radioactive phosphorus. The liver and the lungs proved to be the organs with the greatest radioactivity in the animals that were killed 15 to 30 minutes after the injection. Most of the radioactivity was observed in the lungs, and this was correlated with microscopic demonstration of engorgement of the pulmonary capillaries with leukocytes. In the animals killed three hours to four days after the injection, these differences in radioactivity became less pronounced, and the spleen also showed considerable radioactivity. In the control animals that had been given injections of radioactive phosphorus in inorganic solution, the radioactivity of the lungs was insignificant regardless of when the animals were killed. In additional experiments platelets obtained from 5 liters of human blood were labeled with radioactive phosphorus by the same technique as that used for labeling human leukocytes in vitro. A patient with acute leukemia was given in the course of two hours a transfusion of 100 cc. of a suspension of these platelets. Studies on specimens of blood withdrawn from the patient at various intervals after the transfusion suggested that the transfused platelets disappear rapidly and that the diffusion of the radioactive phosphorus in the circulating blood is imperceptible after two hours. In rabbits killed 20 to 30 minutes after the injection of platelets labeled with radioactive phosphorus, the greatest radioactivity was observed in the spleen, which seems to play a part of selective fixation comparable to that shown by the lungs with regard to the leukocytes.

60:529-548 (April 12) 1952

*The General Adaptation Syndrome of Selye and the Phenomena of Reilly. P. Decourt.—p. 532.

Hydrazine of Isonicotinic Acid in Treatment of Tuberculosis. E. Sivrière. —p. 534.

Selye's General Adaptation Syndrome and Reilly's Phenomena. —Decourt calls attention to a report by J. Reilly and other French investigators in 1934 that was the first of a series on the role of the sympathetic nervous system in certain intestinal lesions, the first studies being made on typhoid patients. Subsequently these investigators showed the nonspecific character of this reaction and spoke of it as Reilly's phenomena. Decourt then shows that the reactions of the organism to which Selye applied the term "the general adaptation syndrome" are identical with Reilly's phenomena. These reactions are not the result of modifications of the adrenal cortex, as is maintained by Selye, but are, as Reilly and co-workers demonstrated, due to action on the sympathetic nervous system by the attacking agent. On the other hand, it has not been demonstrated how the initial lesion of the sympathetic nerve fibers provoke the nonspecific reactions in the peripheral cells. Reilly's school suggested the simultaneous action of several mechanisms: liberation of chemical mediators or toxic organic products, or direct trophic actions, and secondary endocrine reactions. The role of the adrenals in Reilly's phenomena will be discussed in a later report.

Quarterly Journal of Medicine, Oxford

21:123-248 (April) 1952

Benzidine Test for Occult Blood in Faeces. C. D. Needham and R. G. Simpson.—p. 123.

Some Aspects of Natural History of Disseminated Sclerosis. Part I. Incidence, Course, and Prognosis. Part II. Factors Affecting Onset and Course. D. McAlpine and N. Compston.—p. 135.

Treatment of Intermittent Claudication. M. Hamilton and G. M. Wilson. —p. 169.

Obliterative Disease of Abdominal Aorta and Iliac Arteries with Intermittent Claudication. A. Kelwick, L. McDonald and R. Semple.—p. 185.

*Local Circulatory Changes Associated with Clubbing of Fingers and Toes. G. M. Wilson.—p. 201.

Diagnostic Value of Serum Iron Studies in Haemochromatosis: Observations on Seven Patients. J. C. Houston and R. H. S. Thompson.—p. 215.

Metabolic Studies in Severe Diabetic Ketosis. J. D. N. Nabarro, A. G. Spencer and J. M. Stowers.—p. 225.

Circulatory Changes in Clubbing of Fingers and Toes.—Previous reports of increased blood flow through clubbed fingers were confirmed in cases of clubbing associated with cardiac and pulmonary disease. The increase in blood flow was not due to the action of vasodilator nerves. Two cases of unilateral clubbing due to arteriovenous fistulas are described. In one the fistula was in the distal part of the foot, and there was conspicuous unilateral clubbing of the toes. In the other a fistula was in the left index finger, which alone was clubbed. In both cases there was increased circulation in the affected digits. Evidence is presented that reduction in peripheral blood flow led to regression of clubbing, particularly in cases in which ligation of a subclavian artery caused asymmetry in the degree of finger clubbing. In clubbing associated with intrathoracic disease, successful surgical or medical treatment of the underlying condition led to a decrease in finger blood flow and gradual disappearance of the clubbing. It is believed that the blood flow to clubbed digits is in excess of physiological requirements, and largely passes through the numerous arteriovenous anastomoses in the distal segments. The direct passage of blood from digital arteries into the deep venous plexuses is held to account for the structural changes demonstrated in clubbed fingers. Comparison is made between such changes and those related to pathological arteriovenous fistulas in limbs. The superfluous blood flow in unilateral clubbing is thought to be due to physical abnormalities in the arterial tree. In bilateral clubbing it is probably a result of generalized circulatory disturbances.

Revista de Hospital das Clinicas, São Paulo

7:1-84 (Jan.) 1952. Partial Index

Functional Exploration of the Lungs in Decompensated Cardiopathy Due to Chagas' Disease. L. V. Décourt, H. Haebisch and E. Barbato.—p. 1.

Constitutional Capillaropathy: von Willebrand's Type and Simple Capillary Type. M. Abu Jamra, R. Lichtenstein, C. Buhler Vieira and M. O. Ribeiro Leite.—p. 12.

*Osteomyelitis Following Intramedullary Infusion in Infants. F. Pires de Camargo and J. Passarelli Jr.—p. 33.

Repair of Soft Parts in Orthopedic and Traumatological Surgery. L. Barros de Abreu.—p. 41.

Osteomyelitis Following Intramedullary Infusion in Infants.—Osteomyelitis of the tibia developed in 8 patients in a group receiving about 800 intramedullary infusions at the Hospital das Clinicas at São Paulo. Four of the patients with osteomyelitis recovered without after-effects during a 12 month follow-up period, two died, and two had serious residual disabilities. The intramedullary route should never be used for infusions in the presence of infection; osteomyelitis following intramedullary infusion may result in death or deformity. Destruction of the bone and epiphyses results in shortening of the affected limb, sometimes necessitating amputation. Intramedullary infusion should be resorted to only when no other method is feasible, and should always be carried out by a physician familiar with the problems involved. The needle should be withdrawn after each infusion; leaving it in place for a prolonged period is a frequent cause of infection. Another important precaution is immobilization of the limb to keep the needle from moving and to protect the puncture site from possible contamination. A small device for securing immobility of the needle is described by the authors.

Revista de Medicina e Cirurgia de São Paulo

12:133-214 (March) 1952

Cancer of the Esophagus and Cardia. J. M. de Freitas, M. Ramos de Oliveira, E. B. Montenegro and H. Lotufo.—p. 133.
*Hormone Treatment of Varices of Pregnancy. C. Tortelly Costa and J. Pantaleão.—p. 185.

Hormone Treatment of Varices of Pregnancy.—Varices may develop during the last part of the first trimester of pregnancy, increasing rapidly in size until the beginning of the last trimester, when they seem to become stationary. They are localized mostly in the lower part of the body, especially the genitalia and the termination of the rectum, on the outer surface of the hips, and in the legs, and are usually bilateral. Pain caused by large serpentine varices may be so severe that the patient is unable to carry on her usual activities; small vessels dilated over a large area may cause discomfort and are unsightly. Varices are a frequent complication of pregnancy; they occurred in 1,060 of a series of 1,675 pregnant women studied by the authors. The high incidence in this series is attributed to factors such as probable hormone deficiency, the fact that the women lived in hill country and were obliged to carry heavy objects, such as buckets of water, and dietary deficiency. The weakening and relaxation of the walls of the veins that results in distention has been attributed to the presence of a hormone in the circulation, and various estrogens have been found useful in treating the condition. Benzoate of estradiol (progynon B® in oil) was used with good results by the authors, who report 37 typical cases; all 37 patients had severely painful varices and all experienced relief of pain; in 25 no further increase in the size of the vessels took place, and in 12 there was a diminution in size. Intolerance to the medication was not observed, and there were no complications, either during labor or the puerperium. Benzoate of estradiol may reduce lactation, and its use is not advised when labor is near. The newborn were not affected, and there were no cases of abortion.

Semaine des Hôpitaux de Paris

28:1249-1290 (April 22) 1952

*Piperoxan Test in Arterial Hypertension. E. May, Lamotte-Barrillon, R. Heyblon and C. Laville.—p. 1249.
Atypical Myeloma; Study of Roentgenologic, Cytological and Clinical Pecularities. G. Gelin.—p. 1256.
*Epidemiology and Prophylaxis of Poliomyelitis. J. Boyer.—p. 1263.

Piperoxan Test in Arterial Hypertension.—Piperoxan (2-[1-piperidylmethyl]-1,4-benzodioxan, or 933F), in doses of 0.25 mg. per kilogram of body weight in two minutes was injected in 55 patients (33 with essential hypertension, 14 with moderate or doubtful hypertension, and 8 with normal blood pressure). Except for mild tachycardia, restlessness, and anxiety, the test was well tolerated. The patients with essential hypertension showed a temporary rise in the arterial tension and pulse rate. The rise of the systolic blood pressure was characteristic, while that of the diastolic pressure was less constant. The increase in systolic pressure was greater in the patients with essential hypertension than in those with normal blood pressure, while intermediate results were observed in the patients with moderate or doubtful hypertension. Two patients who were given a subcutaneous injection of pentamethonium shortly before the intravenous injection of piperoxan did not respond to the latter drug with a rise of blood pressure. Blocking of vasomotor activity at the level of the autonomic ganglions by pentamethonium abolished the hypertensive effect of piperoxan. It was thus confirmed that piperoxan, besides being adrenolytic, exerts a direct effect on the nervous centers, particularly on the medulla oblongata. The rise of blood pressure is of central origin and is a manifestation of vasomotor hyperexcitability. The rise of blood pressure in response to the piperoxan test in patients with essential hypertension is in contrast to the lowering of blood pressure that occurs in patients with pheochromocytoma. Three patients without pheochromocytoma in the authors' series responded to piperoxan with a lowering of blood pressure. This was not pronounced and did not always occur on repeated tests. Functional, temporary, and insignificant hyperadrenalemia may be present in such cases. Patients with hypertension associated with menopause and with hyperthyroidism deserve to be studied from this point of view. With respect to the pathogenesis of hypertension, the piperoxan test is an aid in eliminating epinephrine and arterenol (nor-epinephrine) as important factors of essential hypertension. The fact that piperoxan causes lowering of blood pressure in dogs in which hypertension had been produced by section of the nerves of Cyon and Hering proves that failure of this depressor system is not responsible for essential hypertension.

Epidemiology and Prophylaxis of Poliomyelitis.—Results of epidemiological studies of poliomyelitis carried out by the author in France for the last 16 years do not support the classical view that poliomyelitis may be transmitted by the nasopharyngeal route, but rather support the modern concept that this disease is spread by contaminated water or food. In addition to milk and butter, green vegetables, such as lettuce, that may become contaminated by manure and soil may be sources of transmission. The war and occupation of France, with the exodus of the population in 1939-1940 and its return in 1944-1945, caused intermittent outbreaks of infectious diseases transmissible through the nasopharyngeal route, such as cerebrospinal meningitis, but did not produce an increased incidence of poliomyelitis despite the fact that these shifts in population occurred during summer and fall. In the Seine district, where, because of food deficiencies during the German occupation, people were eating much fresh lettuce, the disease occurred three or four times as often in the months of July to October as in January to June, in contrast to the usually observed ratio of 2 to 1. A curve plotting the incidence of poliomyelitis between 1943 and 1950 according to the age of the patients showed that 35% were preschool children, 20% were school children, 19% were adolescents, and 26% were adults. The highest incidence of the disease may be observed at the age of 2 years because few infants have antibodies in their serum at this age, while at the age of 4 years two-thirds of the children may have acquired antibodies by an inapparent infection. Paralytic complications occur only in a small number of patients. In countries in which poliomyelitis is common the acute form is rare, because the inapparent infections occur early. It is fortunate, however, that they usually occur before the child loses all the immunity acquired from the mother in utero. On the contrary, in countries in which poliomyelitis is rare the severe acute form prevails. Thus the author concludes that, as sanitation improves, the virus becomes rarer, immunity to poliomyelitis is acquired later, and morbidity and mortality rates are increased.

Tidsskrift for Den Norske Laegeforening, Oslo

72:185-226 (April 1) 1952. **Partial Index**

*Ehlers-Danlos Syndrome: Description of Three Cases in Severely Affected Family. K. O. Husebye.—p. 185.
Mitral Stenosis—Lung Tumor: Report on Clinical Method of Examination. H. Rasmussen.—p. 189.
Radioactive Iodine and Induction of Cancer. L. Kreyberg.—p. 190.
Sino-Bronchial Syndrome. J. Dedichen.—p. 192.

Familial Danlos' Syndrome.—The cases of Danlos' syndrome reported by Husebye occurred in twin brothers and an uncle. The administration of 110 mg. of corticotropin (ACTH) in divided doses over a four day period in one case had no effect on the symptoms. Five additional cases of the syndrome in the family are mentioned. In all, four generations are represented. The disorder is believed to be transmitted by an autosomal dominant factor. Persons with this syndrome should avoid traumas, and should have careful local and general treatment following injury.

BOOK REVIEWS

Advances in Pediatrics. Volume V. Editor: S. Z. Levine. Associate editors: Allan M. Butler, Margaret Dann, L. Emmett Holt, Jr. and A. Ashley Weech. Cloth. $7. Pp. 273, with illustrations. Year Book Publishers, Inc., 200 E. Illinois St., Chicago 11, 1952.

It is fortunate that the editorial staff of this book decided to resume its publication. The high quality of the authoritative articles that has marked this publication in the past continues in this edition. The articles note and evaluate the contributions made by past and present workers. These contributions, recorded in an orderly fashion, summarize the present state of knowledge concerning pediatrics. The authors then discuss their conclusions and the conclusions held by contemporary workers. This manner of presentation is valuable, because it fills a void in medical literature. Few physicians can profitably peruse the large number of articles written about any medical topic, yet many physicians are not satisfied with the necessarily condensed and didactic discussions found in standard textbooks.

The complete bibliographies increase the value of the book as a source of reference for the medical literature concerning the subjects discussed. By the selection of authoritative contributors and topics of current interest, this edition continues to fill a noteworthy position in pediatric literature.

Modern Practice in Tuberculosis. Volumes I and II. Edited by T. Holmes Sellors, M.A., D.M., M.Ch., Thoracic Surgeon, Middlesex Hospital, and J. L. Livingstone, M.D., F.R.C.P., Physician, King's College Hospital and Brompton Hospital, London. Cloth. $27.50 per set. Pp. 355, with 104 illustrations; 441, with 159 illustrations. Butterworth & Co., Ltd., 4-6 Bell Yard, Temple Bar, London, W.C.2; distributors in U. S. A., Paul B. Hoeber, Inc., 49 E. 33rd St., New York 16, 1952.

The editors of this two volume text, surgeon and internist, respectively, in the field of chest diseases and both well-known practitioners in leading London hospitals, have brought together as contributors a majority of the chief authorities on tuberculosis in the United Kingdom. Their purpose, as indicated in the preface, is to furnish a broad picture of medical practice in the field, by inviting recognized experts to present their opinions in their own way without the requirement of conformity to a single pattern of thought. The result is a fairly well-balanced collection of monographs on both fundamental and practical aspects of the treatment of tuberculosis. The late S. Roodhouse Gloyne, formerly consulting pathologist to the London Chest Hospital, sets the stage with a scholarly introduction, stressing the rapidity of modern progress, pointing out that recent advances in understanding have brought together the biochemist, the immunologist, the physician, and the surgeon, with new bacteriological controls. The 39 chapters include treatises on resistance to tuberculosis, vital statistics, the bacteriology and morbid anatomy of the disease, methods of rehabilitation, industrial aspects, diagnosis, prevention, and medical and surgical treatment.

The order in which the chapters appear is somewhat puzzling; in volume I, at least, no planned sequence is evident. This is of little moment, however, as the book will be used as a reference for critical consideration of separate fields rather than as a text for systematic instruction. The reader interested in methods of prevention will naturally turn to the chapters on BCG vaccination and mass x-ray examinations, the student of social problems to those on rehabilitation, psychology of the patient, and tuberculosis in industry, the internist to the monographs on diagnosis and chemotherapy, and the surgeon to the many separate chapters on surgical procedures, advances in excisional therapy, and the treatment of tuberculosis in various organ systems. Each will be rewarded, for the subject is presented in each case by an expert with a background of personal experience. Special tribute must be paid to the surgical chapters. The illustrations are excellent, and the reproductions of roentgenograms, e. g., in the chapter on empyema, are of surpassing quality. The book is heartily recommended as an up-to-date treatise on tuberculosis.

Angewandte Paediatrie: Ein Nachschlag- und Lesebuch für Studierende und Ärzte. Von. Dr. med. et phil. E. Mayerhofer, o. ö. Professor der Kinderheilkunde und Vorstand der Universitätskinderklinik in Zagreb. Cloth. 85 marks. Pp. 813, with 209 illustrations. Wilhelm Maudrich, Spitalgasse 1B, Wien IX/2, 1952.

This is an unusual book, if it is regarded as a conventional pediatric textbook. Its title as well as its subtitle are misleading, since it offers little that would be helpful to medical students. The author, who has been head of a children's clinic in Zagreb, Jugoslavia, for nearly 30 years, wrote this book as a résumé of his life's work.

What seems most unusual is the emphasis on subjects that are generally considered of minor importance in the United States; for example, there are 77 pages dedicated to the discussion of climatotherapy, baths, spas, and related subjects. There are seven pages on diseases of the navel in newborn infants; yet, common diseases of clinical importance, such as acute nephritis and rheumatic fever, receive little attention. The subject matter is arranged according to age groups rather than body systems; this makes it difficult to look up concise information about a given disease. As a pupil of Pirquet's, the author understandably puts much emphasis on allergy. He regards such entities as pyloric spasms, melena neonatorum, and even hydrocele of the newborn as allergic manifestations.

Much of what the author advocates concerning infant feeding is in sharp contrast to modern American concepts. Foods are introduced into the diet late, and methods of formula calculation are complicated. Therapy often seems outmoded as, for example, the advice to administer serum to children with scarlet fever, "even in mild cases."

The illustrations are poor, and the bibliography contains references that are almost exclusively written in German. This book is of interest mainly to pediatricians who would like to get some insight into European pediatric thinking.

A Textbook of Pharmacology: Principles and Application of Pharmacology to the Practice of Medicine. By William T. Salter, M.D., Professor of Pharmacology, Yale University School of Medicine, New Haven. Cloth. $15. Pp. 1240, with 284 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; 7 Grape St., Shaftesbury Ave., London, W.C.2, 1952.

The author has attempted to write a comprehensive textbook for "the medical student and the up-to-date practitioner" wherein the examples cited "in evidence of fundamental statements are selected from material which has a clinical bearing."

The author's choice of apt quotations for each chapter superscription is original, and it is well worth the time spent in browsing through the book to read these quotes alone. Unfortunately, the text is not as concise as the quotations. One factor accounting for this is the attempt to discuss subjects such as antibiotics in a general way instead of discussing each antibiotic in detail. A rigid framework of paragraph headings has been scrupulously followed; this frequently sacrifices the reader's interest and results in needless duplication. Because the manuscript was not revised, some errors can be found, such as on page 1,043: "By the time this book is published, probably the second of these, i. e., sulfapyridine, will have been discarded to a considerable extent"; (sulfapyridine was discarded by 1943). On page 1,042 the author states: "It still remains for chemotherapy to take the field against the rickettsial type of infection," yet on page 1,049 he writes: "Why it [PABA] should be effective in typhus fever remains undetermined." In later pages chloramphenicol, aureomycin, and terramycin are all admitted to be effective against rickettsial infections. Certain chapters, such as those on cardiac drugs, anticonvulsants, and hormones, are outstanding. Other chapters, such as those on anesthesia, curare, and chemotherapy are less than average; however, although the book may be slightly inconsistent, it is readable and is the most up-to-date comprehensive text on pharmacology available.

The reviews here published have been prepared by competent authorities and do not represent the opinions of any official bodies unless specifically stated.

The Chemistry and Technology of Food and Food Products in Three Volumes. Volume III. Prepared by group of specialists under editorship of Morris B. Jacobs, Ph.D., Adjunct Professor of Chemical Engineering (Food, Flavor, and Dairy Technology), Polytechnic Institute of Brooklyn, Brooklyn, N. Y. Second edition. Cloth. $15. Pp. 1773-2580, with illustrations. Interscience Publishers, Inc., 250 Fifth Ave., New York 1; 2a Southampton Row, London, W.C.1, 1951.

The third and concluding volume of this series of excellent articles on the different aspects of the chemistry and technology of foods discusses the various commercial methods of food preservation and describes the processing of common foods. Dehydration, temperature control, the use of hermetically sealed containers, the use of micro-organisms, and the use of chemical preservatives are discussed with enough detail to give the uninformed reader an idea of the varied problems encountered in the industry. A concise chapter on the why and the wherefore of packaging ends the first part of the book.

The last section, dealing with the processing of common foods, will prove extremely interesting to those desiring a general picture of the actual methods used in manufacturing cereals, breads, sugars, confectionery, fruit juices, milk and milk products, meat, oils and fats, and beverages. Each chapter is written by an authority familiar with the food in question. The material in this last part tends to overlap the subject matter in part 4, which describes the various food groups; however, this overlapping has been kept to a minimum and is not objectionable, since it is used only to maintain the continuity of a chapter. An important aspect of this series of articles is the stress placed on the role played by adequate nutrition in modern life. The physician and dietician will especially appreciate this, since few books concerned with the technology of foods emphasize the subject of nutrition.

Differentialdiagnostische Symptomatologie von Krankheiten des Kindesalters: Klinische Vorlesungen. Von Professor Dr. med. Werner Catel, Chefarzt der Landeskinderheilstätte, Mammolshöhe/Taunus. Second edition. Cloth. 69 marks. Pp. 640, with 222 illustrations. Georg Thieme, Diemershaldenstrasse 47, (14a) Stuttgart O; agents for U. S. A., Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1951.

The author has selected a single symptom as the topic of each chapter. The diseases that might cause or contribute to this symptom are then discussed within the framework of each respective chapter. The author is concerned with enumerating and classifying the diseases responsible for a given symptom rather than with distinguishing one disease from another. Each disease is thus described as an entity. The following symptoms are discussed: dwarfism, cranial deformities, hypertension, hypotension, cardiac arrhythmia, anemia, polycythemia, skin and mucous membrane hemorrhage, cervical lymph node enlargement, vomiting, abdominal pain, jaundice, paralysis, and spasm.

In the last 50 years, books on pediatrics or internal medicine, including those on differential diagnosis, have lost contact with diagnostic medicine as it is actually practiced to an ever increasing degree. The use of laboratory tests, radiological consultations, health surveys, preventive measures, improved record keeping, and standardized nutrition have narrowed down the choice of diagnoses in a given case to such a degree that the correct diagnosis is made in most instances without the ponderous weighing of classical clinic signs and before the disease has unfolded itself to such an extent as to match its classical description. This book represents a commendable reversal of this trend. Its author has succeeded better than most of his predecessors in eliminating the so-called classic description of diseases by omitting antiquated diagnostic signs and by avoiding a chronological description of the course of diseases. In short, the discussion of diseases in this book stands closer to medicine as it is practiced today in a well-equipped hospital than will be found in most other books. This has been accomplished by the inclusion of an exceptionally large number of radiographs, most of which are technically adequate. Their interpretation by the author is generally correct except when it refers to controversial material as is the case in discussing some chondrodystrophies. The author circumvents the necessity of covering the entire field of pediatrics by giving his book the subtitle of "clinical lectures" and by pointing out in his preface that only a segment of pediatrics is dealt with "thus far," implying that future editions might be more complete.

The discussion of neurological diseases could have been improved by omitting a detailed description of normal and abnormal response of muscle to faradic and galvanic stimulation, which is nowadays of little diagnostic value. A tendency to subdivide disease entities beyond the logical limit distracts from this chapter as well as from the discussion of skeletal anomalies. The severest criticism of the book is that the bibliography contains German and Swiss references almost exclusively. This is reflected in the text by the briefness with which non-German, particularly Anglo-American, contributions to pediatrics are treated. Cystic fibrosis of the pancreas and sickle cell anemia occupy only a small paragraph, and the discussion of celiac disease is disproportionately short. There is no discussion of the recently investigated specific properties of prenatal and neonatal hemoglobin, although the chapter on blood diseases is extremely long. Electrocardiography is extensively discussed, but the existence of a fourth or fifth electrode is nowhere mentioned. A single page is dedicated to the discussion of congenital anomalies of the heart and confined to their electrocardiographic manifestations. It seems that the author is unaware of the present status of heart surgery in the United States and the need for a more accurate differential diagnosis of congenital heart lesions. He also appears to be unfamiliar with some of the modern theories on calcium metabolism. Though he is familiar with Fuller Albright's work, he quotes only Fanconi in his bibliography. He emphasizes the importance of citric acid for adequate calcium resorption and finds cow's milk inadequate in this respect. He ascribes relative antirachitic properties to mother's milk, because it contains the optimal ratio between calcium ions and citric acid radicals. This viewpoint is not generally accepted in the United States. One gains the impression that the author is a German nationalist who endeavored above all to represent the neo-German school of pediatrics from 1936 to 1950. Though this might not be objectionable to the American reader who has other sources of information at his disposal, it is probably detrimental to German pediatricians, because it isolates them further from Western thinking.

Despite these shortcomings, this book will probably gain a permanent place in pediatric literature because of its original approach and its literary unity, which is the result of single authorship. Its usefulness is limited to those pediatricians who are teachers and desire to stimulate or lead intelligent ward round discussions. The quality of paper, print, and illustrations is excellent.

The Isocortex of Man. By Percival Bailey and Gerhardt von Bonin. Illinois monographs in medical sciences, vol. VI, nos. 1, 2. Paper. $5. Pp. 301, with 136 illustrations. University of Illinois Press, 358 Administration Building, Urbana, Ill., 1951.

The immediate reason why the authors decided to embark on this investigation of the human cerebral cortex lies in their previous work on the macaque and chimpanzee. These previous studies were undertaken to lend precision to the results of physiological neuronography performed on these primates and was to be applied, it was hoped, to man. In the course of their studies, it became clear that the precise homology of which they had dreamed could be achieved only when maps of all three primates, man, chimpanzee, and macaque, had been drawn by the same observers.

The authors also point out that contrary to the commonly accepted idea the number of brains that have been adequately studied from the cytoarchitectonic viewpoint is small. In addition, some of the reports in the literature are based on the study of brains that were removed several to many hours after death and not properly fixed.

The monograph is divided into nine chapters. The longest, chapter 5, is concerned with the description of serial sections cut from 21 blocks that together comprised one whole hemisphere of a young, healthy adult who died of an accident without injury to the head. The brain was removed within one hour after death after perfusion through the arteries with a 4% aqueous solution of formaldehyde. Study of Nissl preparations was supplemented by the examination of silver impregnations and preparations stained for myelin.

From their study, the authors recognize two distinct types of cortical architecture, the allocortex and the isocortex and four variants of the latter: eulaminate, agranular, koniose, and limitrophic. These are discussed fully in chapter 4.

The brain map is discussed in more detail in chapter 6. The authors stress the fact that vast areas of the cortex are so closely similar in structure as to make any attempt at subdivision unprofitable, if not impossible, and that the drawing of sharp areal boundaries, on the basis of many structural peculiarities of varying distinctiveness and significance, is the fundamental defect of most maps and has been carried to absurd lengths by the Vogt school.

Other chapters deal with growth and weight of the brain, fissures of the brain, intercortical connections, afferent and efferent connections, and functional significance. In addition, there is an historic introduction, a bibliography, and plates illustrating 15 different regions, which, from the architecture of the cortical cells, can be readily recognized. Throughout the monograph, there is a critical discussion of previous work on the subject. Many of the 121 figures are reproduced from other authors' works in order to clarify this discussion.

The book is well printed, the figures and plates well reproduced, and the style clear and readable. The simplified cortical map that is related to functional activity will be gratefully received by the many neurologists and neurophysiologists who have difficulty in recognizing the minute differences between the numerous areas of the cortex described by other authors and who can see no useful purpose in excessive parceling of the brain.

An Atlas of General Affections of the Skeleton. By Sir Thomas Fairbank, D.S.O., O.B.E., M.S., Consulting Orthopaedic Surgeon, King's College Hospital, London. Cloth. $10. Pp. 411, with 510 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1951.

In this book, about 80 skeletal disorders that typically involve more than one bone of the skeleton are clearly and concisely discussed. This is a reference book for diagnosis, and, as such, it will be a valuable addition to the library of all orthopedic surgeons.

The technical publication of the book is excellent, and the illustrations are clear and diagnostic. The sections on the dysplasias of bone and cartilage growth are exceptionally complete and informative. Although a few good microscopic illustrations are included, the value of the book would be enhanced with more discussion of pathology with accompanying illustrations. There are occasional statements in the book that are not in agreement with most American authors. An example of this is the following statement: "As a help in diagnosis, alkaline phosphatase estimations are disappointing, and are practically useless." This viewpoint is not accepted in the United States. The author makes the statement that the alkaline phosphatase is raised in all conditions in which there is hypocalcification or osteoporosis. This statement is not substantiated by clinical experience. The author discusses aseptic necrosis of bone in a logical manner, although his conclusions are open to argument.

The 62 chapters of this book include discussions of most of the idiopathic, congenital, endocrine, hereditary, and neoplastic diseases of the skeleton. It is recommended as a reference book to all physicians engaged in the diagnosis and treatment of skeletal disease.

Leonardo da Vinci on Movement of the Heart and Blood. By K. D. Keele, M.D., F.R.C.P. With foreword by Charles Singer, M.D., F.R.C.P. Limited edition. Cloth. $15. Pp. 142, with 69 illustrations. J. B. Lippincott Company, 227-231 S. Sixth St., Philadelphia 5; Aldine House, 10-13 Bedford St., London, W.C.2; 2083 Guy St., Montreal, [n.d.].

Any work about Leonardo da Vinci, the almost fabulous man of science, should intrigue the intelligent reader and stimulate his curiosity. On reading this book, one will have renewed admiration for the genius of Leonardo and will be grateful for the skillful manner in which the author guides the reader through the maze of Leonardo's anatomic work.

The author sketches Leonardo's importance as an artist, a physicist, and an engineer but does not attempt to describe all of Leonardo's achievements in the field of anatomy. He confines his attention to Leonardo's anatomic work on the heart and blood vessels, pointing out his mistakes as well as his achievements. Leonardo, an accurate observer in most instances, was, however, so steeped in the anatomic teachings of Galen and Avicenna that he described septal pores in the heart and represented the right pulmonary veins as entering the right auricle. On the other hand, his illustrations and descriptions of the heart valves, both in systole and diastole, of the coronary arteries, of the atria, of the sinuses, later named by Valsalva, and of the moderator bond are accurate and surprisingly vivid.

Of absorbing interest are the physiological studies on the heart and circulation made by Leonardo. He describes and illustrates the action and function of the heart valves, the myocardium, the pulmonary vessels, and the veins. Although he accepts the fundamental theses of Galenic physiology, Leonardo often disagrees with Galen and views the body entirely from the mechanistic point of view.

Dr. Keele's work is written with charm and clarity. Although he stresses Leonardo's greatness, he also points out his limitations as a child of his century. The book is excellently illustrated, beautifully printed, and takes its place as an important addition to the subject of Leonardo's anatomic researches.

Die Hormone. Von Dr. med. Rudolf Abderhalden, Dozent für Physiologie und Pathologie der Hormone, Vitamine und Fermente an der Universität Basel. Lehrbuch der Physiologie in zusammenhängenden Einzeldarstellungen. Unter Mitarbeit einer Reihe von Fachmännern, herausgegeben von Wilhelm Trendelenburg und Erich Schütz. Cloth. 29.70 marks. Pp. 203, with 46 illustrations. Springer-Verlag, Reichpietschufer 20, Berlin W. 35 (West-Berlin); Neuenheimer Landstrasse 24, Heidelberg; Göttingen, 1952.

This small monograph is one of a series comprising a textbook of physiology. It makes no attempt to cover the entire subject, as it omits all anatomic considerations and refers only in passing to the clinical aspects of the subject. After a brief introductory consideration of the general principles underlying endocrine activity, each of the hormones is considered consecutively, with special emphasis on their chemical structure and physiological action. The material is sound and up-to-date but lacks coordination and critical evaluation. This is also reflected in the 700 references appended to the text. In general, this monograph may be recommended to the physiologist, for whom it was primarily written, but it will be of little interest to the clinician and student of endocrinology.

Etiology and Diagnosis in the Treatment of Infertility in Men. By Robert S. Hotchkiss, M.D., Professor and Chairman, Department of Urology, New York University, New York. Publication number 53, American Lecture Series, monograph in American Lectures in Endocrinology. Edited by Willard O. Thompson, M.D., Clinical Professor of Medicine, University of Illinois College of Medcine, Chicago. Cloth. $2.50. Pp. 73, with 15 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto, 2B, 1952.

The information in this monograph is taken largely from the author's extensive experience and research in the field of male infertility. In general, the book is well written and concise. The illustrations are of good quality, and the type is excellent for quick reading. The recognized methods and procedures for evaluating male infertility are discussed in a well-organized form. In addition, there is a brief discussion of the embryologic features of the reproduction tract and the interrelation of the endocrine system. The book can be especially recommended to the busy practitioner who wishes a short, up-to-date treatise on male infertility.

Diseases of the Nose, Throat and Ear: A Handbook for Students and Practitioners. By I. Simson Hall, M.B., Ch.B., F.R.C.P.E., Surgeon to Royal Infirmary, Edinburgh. Fifth edition. Cloth. $4. Pp. 463, with 90 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1952.

This edition of a popular English handbook on diseases of the nose, throat, and ear is directed to meet the needs of general practitioners and medical students. In this revision, the author has introduced proved, acceptable methods of therapy but has avoided discussing controversial topics.

QUERIES AND MINOR NOTES

KIDNEY FUNCTION TESTS

To the Editor:—*Other than concentration tests are there any kidney function tests like the liver function tests that will aid in diagnosis? In other words, is one kidney function test sufficient, or is more than one required, each testing a separate function?* *F. D. Cook, M.D., Menomonie, Wis.*

Answer.—The kidney, like the liver, performs many functions, some even that are unrelated to the formation and excretion of urine. For the most part, the clinical tests of renal function serve as a means of quantitative diagnosis of the degree of functional impairment. Qualitative diagnosis of the nature of the renal disease is ordinarily made from other clinical data but may be derived, in part, from the use of the specialized clearance techniques in which glomerular and tubular functions can be separately measured and their interrelations as well as their relations to renal blood flow determined. Unfortunately, these procedures are not practical except in institutions equipped with research laboratories and experienced personnel.

For ordinary clinical purposes, the most useful tests for kidney function are the test for maximal concentrating ability, which measures over-all tubular function, and the urea clearance test, which is a fair index of glomerular filtration. In advanced stages of renal disease, when acidosis and other electrolyte disturbances dominate the clinical picture, serum electrolyte determinations for sodium, potassium, chlorine, carbon dioxide, phosphorus, and calcium assume priority over other tests for renal function as a guide to treatment. In the nephrotic syndrome, quantitative measurement of urinary protein output and of the serum protein and lipid fractions can be helpful in diagnosis and treatment.

In rare types of renal disease, such as the Fanconi syndrome, hyperparathyroidism, and osteomalacia caused by the inability of the renal tubules to make ammonia, special serum and urine electrolyte studies may be the only means of making a diagnosis. It must never be forgotten that pyelographic examination is also a renal diagnostic aid of major value when used properly. Careful study of the urinary sediment is another important aid, although it is often neglected. In short, routine studies of kidney function are best performed after considering the individual clinical problem and selecting the tests adequate to furnish the information desired.

POISONOUS FISH

To the Editor:—*I have read that certain fresh fish are poisonous, especially tropical varieties. Is there any way to determine which fish are poisonous? What is the cause of the so-called poisoning? Is any treatment of value for such disturbances, which I understand are primarily gastrointestinal in nature?* *Edwin Pyle, M.D., Waterbury, Conn.*

Answer.—Ichthyosarcotoxism is a distinct clinical entity resulting from ingestion of poisonous fish flesh. The results of studies seem to indicate that these toxins are not bacterial in origin. Hence, putrefaction does not play a significant role in the production of the disease. Whether the toxin is a poisonous alkaloid or some closely related substance is not known. The disease is largely confined to tropical but widely scattered areas such as the Virgin, Marshall, and Mariana Islands. Many tropical marine fishes that are considered to be valuable commercial species in one area, e. g., red snapper, rockcod, seabass, and crevally, may be violently toxic in another. The number of poisonous fishes and their distribution is unknown; moreover, there is no simple test available at present for determining their edibility. The most reliable methods involve the preparation of tissue extracts, which are injected intraperitoneally into mice, or feeding samples of the viscera and flesh to dogs or cats. The test animal should be observed for a minimum of 24 hours for the appearance of gastrointestinal and neurological symptoms. Some of the earliest symptoms to appear in animals are vomiting, hypersalivation, and ataxia. In severe cases, the ataxia will be followed by convulsions, paralysis of the limbs, respiratory paralysis, and death. Numerous theories have been propounded on the cause of fish poisoning, but none have been supported by solid scientific evidence. The majority of ichthyologists believe the answer will be found in the food chain of these fishes.

The disease is characterized by symptoms of diarrhea, nausea, vomiting, and abdominal pain, which usually occur within three to six hours after ingestion of the fish. Gastrointestinal symptoms may be accompanied or followed later by sensations of tingling of the mouth, tongue, lips, face, hands, and feet. About 50% of these patients complain of difficulty in determining the temperatures of liquids either when taken by mouth or on the external surface of the skin. Hot objects are sometimes interpreted as feeling cold and cold objects hot. In addition there may be other types of sensory and muscular disturbances which may be bizarre in their manifestations. Extreme weakness and muscular paralysis are common. The mortality rate is said to be about 1 to 2%. The treatment is symptomatic, since there is no known specific antidote. The reader is referred to Khlentzos's article (*Am. J. Trop. Med.* **30**:785-793, 1950) for a more complete discussion on the treatment of fish poisoning. An attack of ichthyosarcotoxism does not impart immunity.

GENITAL TUBERCULOSIS

To the Editor:—*A married European woman, aged 30, has genital tuberculosis, as demonstrated by repeated biopsies of the endometrium during 1950. A year ago she was treated with streptomycin, 1 gm. daily for 60 days, simultaneously with para-aminosalicylic acid, 10 gm. daily for 50 days. During the past year, following treatment, three biopsies were negative for tuberculosis. A hysterogram taken with low pressure of the contrast medium showed bilateral pyosalpinx or hydrosalpinx at the ampullar sections of the fallopian tubes. The remainder of each tube is permeable and not dilated but seems irregular and knotted. The patient feels well and desires pregnancy. She will begin a trip to the United States and Europe in two months. 1. Should an operation be performed before she makes her trip? 2. Is radical removal of the fallopian tubes necessary? 3. If the radical operation is advisable, is there any chance that the patient would achieve normal pregnancy if a plastic operation were done? 4. What kind of plastic operation would you suggest? 5. Does any inconvenience or danger of complications, such as extrauterine pregnancy, abortion, necrosis, or postoperative adhesions, result after such a plastic operation? 6. What safety measures, such as antibiotic therapy, short wave diathermy, and contraceptives, should be applied before, during, and after the operation.* *M.D., Chile.*

Answer.—The fact that recent endometrial biopsies done one year after streptomycin therapy have shown no evidence of an endometrial lesion cannot be interpreted as indicating that there has been a cure of tubal tuberculosis, which has almost surely been present also. What little evidence is as yet available on the results of streptomycin therapy in genital tuberculosis seems to be strongly against such an assumption. Gynecologists are anything but enthusiastic about the results of tubal plastic procedures, even for nontuberculous salpingitis. It is difficult to believe that anyone would recommend these measures for the tuberculous variety, in the presence of which pregnancy is notoriously rare, not merely because of the frequency with which occlusion occurs but also because of the diseased tubal wall and

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The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

consequent impairment of its physiological function. This would apply whether the procedure involved is salpingostomy, tubal resection and tubouterine implantation, or cornual ovarian implantation.

1. There would be little or no hazard in a delay of two months or in the "strain" of a sea voyage, which is often relaxing and restful. 2. Radical removal of the fallopian tubes is, in general, the accepted and the soundest procedure for tubal tuberculosis. 3. Only a slim possibility of normal pregnancy would result from cornual implantation of the ovaries, which can usually be safely conserved. The prospects for pregnancy after any such plastic operation are very poor, especially in a patient having tuberculous disease. 4. The choice of plastic operations to be done must be based on the operative findings and would involve supplementary use of such procedures as reverse insufflation. 5. There is danger of the complications mentioned. 6. In regard to necessary safety measures, the normality of the husband should be established and the possibility of pulmonary tuberculosis in the wife excluded. Postoperative insufflations are indicated in the case of tubal plastic operations; other details of preoperative and postoperative care should be left to the surgeon.

HYPERTENSION, CIGARETTES, AND CAFFEINE

To the Editor:—*During the last few months, I have checked some R. O. T. C. boys for hypertension. Without exception, so far, all those with hypertension have shown normal blood pressure about 48 hours after they discontinued smoking cigarettes and drinking coffee and cola drinks. At the same time psychotherapy has been administered. Will you please comment on the foregoing?*

H. D. White, M.D., Dallas, Texas.

Answer.—It seems probable that cigarettes, coffee, and cola drinks have a greater pressor action in prehypertensive and hypertensive subjects than in normotensive ones. It is from the group of prehypertensive that the truly hypertensive patients are drawn. This problem is discussed by Page and Corcoran (Arterial Hypertension: Its Diagnosis and Treatment, ed. 2, Chicago, The Year Book Publishers, Inc., 1949). Nicotine is well known to have a pressor effect in many but not in all patients. Whether only the prehypertensives show an abnormally great response has not been determined. Roth has discussed in some detail this part of the problem in her recent monograph (Tobacco and the Cardiovascular System, The Effects of Smoking and of Nicotine on Normal Persons, American Lecture Series, Publication 100, Springfield, Ill., Charles C Thomas, Publisher, 1951). Caffeine in moderate doses has little effect on arterial pressure but in excessive amounts, by overstimulating the brain, leads to a prolonged slight rise in blood pressure. Obviously many youngsters greatly abuse smoking, and drinking of coffee and cola containing beverages. Those with hypertensive propensities should probably avoid them.

YAWNING

To the Editor:—*Why is a yawn "catching?"*

Edward Dengrove, M.D., Asbury Park, N. J.

Answer.—Yawning may be considered to be a form of inspiration tic, associated with dyspnea. P. Janet describes yawning as related to sighing, sobbing, and hiccoughing, and refers to it as an example of "imitative contagion" (The Major Symptoms of Hysteria, New York, The Macmillan Company, 1907, p. 259). It is a complex nervous respiratory reflex associated with mild boredom, irritating mental effort, or mild emotional anxiety. It is classified by R. Burton-Opitz (A Textbook of Physiology: For Students and Practitioners of Medicine, Philadelphia, W. B. Saunders Company, 1920, pp. 592-593) among the association or perception reflexes. These "skirt the realm of volition without being actually influenced by it and may be invoked in us in consequence of a visual impression of some one else already engaged in the act." Yawning is catching as a conditioned visual reflex when persons in a group are subjected to the same general boring or frustrating environment.

PHYSICAL RISK WITH ONE KIDNEY

To the Editor:—*A 30-year-old white man has been in good health all his life. While in military service during World War II, his right kidney was removed following trauma (concussion). He is now in good health and able to work and is active in softball and bowling. Yearly physical examinations show him to be normal. I would like your opinion concerning the physical risk to this patient resulting from the removal of one kidney, as there is a difference of opinion on this point.*

A. J. Wagner, M.D., Tucson, Ariz.

Answer.—Several papers have been written on the subject of longevity in persons with only one kidney. One of the most informative papers on this subject is that by the late Dr. Herman Kretschmer (*J. A. M. A.* **121**:473-478 [Feb. 13] 1943). Judging from his data, it is evident that life expectancy of such persons is about the same as that of persons with two kidneys, providing the remaining kidney is normal. To establish the fact that the one kidney is normal not only would necessitate the usual physical examination but would require special studies including a thorough, carefully done urinalysis, studies of renal function, and pyelographic examination. After such a complete study is made, it would be safe to assure the patient with one kidney that there is no physical risk involved in carrying on the usual types of work or play.

INFECTION OF LINGUAL TONSIL

To the Editor:—*A 35-year-old woman has recurrent attacks of a purulent infection of the lingual tonsil. These infections run a violent course causing a high temperature, and last for about one week. They do not respond to treatment with antibiotics and sulfonamides. This patient does not have tonsils or lymphoid tissue in the throat or nasopharynx. Her lingual tonsil is hypertrophic. Since surgical treatment is not intended, what results could be expected from treatment with roentgen irradiation?*

M.D., Ohio.

Answer.—The efficacy of roentgen irradiation in treatment of infected lingual tonsils is questionable. Application of a 10% solution of silver nitrate directly to the lingual tonsils once daily for several days may have a beneficial effect. Electrosurgical reduction of hypertrophied lingual tonsils should be attempted if mild cauterization proves inadequate.

FALSE POSITIVE SEROLOGIC TEST FOR SYPHILIS

To the Editor:—*A 33-year-old white man took an insurance examination, which was negative, except that on one of several examinations the left pupil was slightly smaller than the right. Two Kahn tests, taken at a two week interval, were 4+. The man gave a history of having had a 4+ serologic reaction 13 years before. At that time he received neoarsphenamine for three weeks, which produced nausea, vomiting, and headache. Four years later, he had a 4+ reaction, and a recheck showed a 2+ reaction. He then received penicillin injections five times daily for 10 days. Tests taken a week later and others taken several months afterward were still 4+. His father has a negative serologic test. His deceased mother had two miscarriages followed by four normal births. The patient's three siblings have negative tests. This man denies any possible contact other than his wife, who also has a negative serologic reaction. His spinal fluid Wassermann is negative, and his colloidal gold test reaction is normal. What is the consensus regarding "Wassermann-fast" persons? Does he have syphilis, and should he be turned down as an insurance risk? What treatment is recommended?*

M.D., Arizona.

Answer.—This is the sort of situation in which the physician should suspect that the patient in question does not have syphilis but does have a biologic false positive serologic test. The factors that prompt this suspicion are the absence of any known exposure to syphilis, the absence of a history of infection, a normal physical examination, normal spinal fluid, and negative epidemiological investigations of parents, siblings, and wife. The probability of biologic false positivity is not ruled out by the fact that the patient's blood serologic test has been known to be positive for 13 years.

The differentiation between syphilis and the biologic false positive phenomenon can be made by means of the newly developed treponemal immobilization test (Mohr, C. F.; Moore, J. E.; Nelson, R. A., and Hill, J. H.: Studies on the Relationship of Treponemal Antibody to Probable Biologic False Positive Serologic Tests for Syphilis, *Am. J. Syph.* **34**: 405 [Sept.] 1950.) This test for treponemal antibody is valid in spite of the fact that the patient has been treated for syphilis.

If the treponemal immobilization test is negative, it may be assumed that the patient does not have syphilis. In that case, the question arises: "Why does he have a biologic false positive serologic test?" This problem can be solved only by a fairly elaborate clinical and laboratory survey of the patient and perhaps by follow-up examinations repeated over a period of years. If the patient does have syphilis (the treponemal immobilization test being positive), no treatment is recommended. The question of seroresistance is discussed both from the standpoint of the physician and the patient by J. E. Moore (Seroresistance [Wassermann-Fastness] in Syphilis: A Discussion for the Patient, *Am. J. Syph., Gonor. & Ven. Dis.* **30**: 125 [March] 1946).

The eligibility of the patient for life insurance, assuming that he has syphilis, is likewise discussed by Schamberg and Moore (The Eligibility of Syphilitic Persons for Life Insurance, *J. A. M. A.* **134**: 1532 [Aug. 30] 1947).

HOT FLUSHES

To the Editor:—*A married woman, aged 28, complains of hot flushes occurring midway between her menstrual periods. She experiences some 6 to 10 episodes in 24 to 48 hours, each lasting about 10 to 20 minutes. Her menses are normal and regular. She has two children. Several days after she delivered her first baby she had eclampsia. She has had one ovary removed. Pelvic examination reveals no abnormalities. This woman, according to her mother, seems to have undergone some mental change since her eclamptic state four years ago and is despondent and worried about her condition. What treatment do you suggest?* *M.D., South Carolina.*

Answer.—It is unlikely that the hot flushes of which this woman complains are associated with her ovarian function. Hot flushes are usually the result of waning or complete cessation of ovarian function associated with the climacteric. Undoubtedly, her vasomotor symptoms have a psychosomatic origin and are related to her nervous state. Therapy should be directed at an understanding of her emotional difficulties and their eradication. Mild sedatives, such as phenobarbital, are helpful. Hormone therapy is not indicated.

CORTICOTROPIN AND RH-NEGATIVE MOTHER

To the Editor:—*I understand that corticotropin (ACTH) will produce changes in agglutination and sensitivities that will change positive results of a Coombs' test to negative results. Has any work been done on the use of corticotropin in Rh-negative mothers with multiple pregnancies? Would an Rh-negative patient with a high titer count and a positive Coombs' test reaction be helped by a series of treatments with corticotropin in the latter months of pregnancy?*

Ardow Ameduri, M.D., Utica, N. Y.

Answer.—No convincing evidence has been published that corticotropin or cortisone therapy affects antigen-antibody reactions or that it changes positive Coombs' test results to negative ones. Nor is there any rationale for the use of such therapy for sensitized Rh-negative mothers in order to reduce or eliminate the Rh antibody titer. Even assuming that corticotropin (ACTH) or cortisone therapy could stop antibody production, such therapy would be undesirable and impracticable for the following reasons. 1. The injections would have to be given daily throughout the entire nine months of pregnancy. 2. The therapy, if it were effective, would stop all antibody production, not merely Rh antibody production alone. This would leave the mother vulnerable to a fatal sepsis caused by invasion into her blood stream of pathogenic organisms to which she will have lost her immunity. 3. The effects, if any, of the therapy would show themselves very slowly, due to the long half life of 35 days of antibody molecules (Wiener, A. S.: The Half-Life of Passively Acquired Antibody Globulin Molecules in Infants, *J. Exper. Med.* **94**:213-221 [Sept.] 1951). Thus, if the initial antibody titer is 100 units, for example, it would take at least 35 days until the titer dropped to 50 units, and another 35 days for it to drop to 25 units. Considering that the titration method itself has a large technical error of about 100 to 200%, even if the therapy were effective it would take at least two or three months before the titrations could reliably demonstrate any fall in titer. Certainly, injections given only during the latter months of the pregnancy would be useless. Summarizing, therefore, there is no rationale for the use of corticotropin (ACTH) or cortisone for treating Rh sensitization, and such therapy is not recommended.

A DISTURBED CHILD

To the Editor:—*An 8-year-old boy is physically and mentally sound but emotionally unstable. His birth was normal, and his only severe sickness was rubella at age of 15 months, at which time he had one convulsion with a temperature of 105 F. His physical development is good except that he is very fat. A basal metabolism rate cannot be taken owing to his fearfulness. The child wakes at night crying. He craves sweets and vegetables, will not eat meat, and will not fight back when intimidated. Would you advise application of thyroid extract,* d*-amphetamine sulfate (dexedrine®), diet with punishment to make him eat a rounded diet, sedatives at night, or psychiatry?*

M.D., North Carolina.

Answer.—This boy undoubtedly has a complex of fears and frustrations, which causes the symptoms enumerated, night crying, craving for sweets and vegetables, and refusal to eat meat. Thyroid extract, *d*-amphetamine sulfate, and diet and punishment will be unavailing until his fears and frustrations are investigated. Psychiatry or mental hygiene would seem the best solution for this child's problems; if these are not immediately available, sedatives at night might be useful.

MECHANICAL APTITUDE

To the Editor:—*A father is interested in working with mechanical and electrical machines, although his vocation is that of a department store manager. He declares that his son does not know about his interest and ability in practical mechanics but insists that the boy has inherited his mechanical ability. He feels that the interest and ability that his son exhibits is an inherited characteristic. I would appreciate your opinion about this.* *M.D., Pennsylvania.*

This inquiry was referred to two consultants, whose respective replies follow.—Ed.

Answer.—Possible inheritance of mechanical aptitudes has been little studied, but L. Dewey Anderson found such abilities, in junior high school boys, were largely independent of the economic status of the home or even of such factors as the number of tools owned by the father. General intelligence is found to account for only about one-sixth of a person's mechanical ability, and the very gifted children studied by L. M. Terman in California actually fell below the average of their schools in mechanical ingenuity. This may have been due partly to a lack of interest and to overspecialization along abstract lines. But all of these lines of investigation suggest that the basis of mechanical abilities is in some way inherited and is not dependent merely on training or imitation.

Answer.—There appears to be no significant investigation on the possible inheritance of mechanical ability. An unequivocal answer to the question is therefore impossible at this time. There is, however, no valid reason for doubting the possibility and even the probability of a genetic basis for mechanical competence, although of course environment must also play a part in the development of such competence.

ENTERITIS IN TOURISTS

To the Editor:—*I have read with much interest the views expressed on the subject of enteritis in tourists in* The Journal, *January 12, 1952, page 164. Enteritis in tourists is a well-recognized phenomenon in India, and it probably is due to the same cause there as in countries like Mexico and Egypt. The condition is found in countries in which the climate is tropical or subtropical and the prevailing standards of hygiene are poor.*

We have investigated many cases of this condition, which is due to bacillary dysentery. Examination of fecal specimens early in the illness will reveal the presence of bacillary exudate, which is often profuse. The causative organism is a bacillus of the Flexner type. In this part of India, the condition is found less often in tourists, since we have few tourists, than in newcomers and visitors. Newcomers from Europe are notoriously likely to get bacillary dysentery within a short time after their arrival. It would probably be no exaggeration to say that all get it. Immunity to the local strain is soon acquired. In the case of "permanent" residents, a holiday visit to a hill-station or to a distant part of the country is likely to entail an unwelcome but inevitable introduction to a different strain of the causative bacillus, manifested by another attack of dysentery. These are commonplace facts known to discerning medical men and laymen.

The remedy is not to be sought in avoiding any particular type of food or cooking medium or even in confining one's milieu to international hotels. The disease is an infection highly amenable to prompt treatment by sulfaguanidine. If this is realized, the need for transoceanic telephone calls or rushing from the country in panic never to return need not and indeed will not, arise.

W. B. Roantree, M.D., Kolar Gold Field,
Champion Reefs, Post Office, South India.

TULAREMIA

To the Editor:—*In* The Journal *Nov. 17, 1951 (page 1184), in Queries and Minor Notes is a question about tularemia. I treat about 12 cases of tularemia every year in this community, and I have found that streptomycin, given in a dose of 0.3 gm. every 3 hours for a patient weighing 150 lb. (68 kg.), is very effective. In three to five days all symptoms disappear.*

In a serious disease such as tularemia it would not be advisable to experiment with a dose of ½ or 1 gm. given every 24 hours. In the treatment of children the fraction by weight of the above dose will accomplish the same results, and the same is true of a patient excessive in weight. I treated many patients for tularemia before the advent of streptomycin and, therefore, have been able to compare the effect on the prolongation of the disease of streptomycin therapy and other therapy. Results with the use of streptomycin have been amazingly good. I would suggest that the patient mentioned be given streptomycin, even at this late date.

G. T. Stewart, M.D., Mena, Ark.

ANTIBIOTIC THERAPY OR TONSILLECTOMY

To the Editor:—*In response to the question in Queries and Minor Notes* in The Journal (148:978 [March 15] 1952) *"Can a diseased tonsil be cured by antibiotic therapy without an operation?" I wish to report that I have had several patients in whom antibiotics administered in aerosol form have apparently cured chronically diseased tonsils with secondary lymphadenopathy. The most important factor is selection of the proper antibiotic, which is best done by culture and in vitro inhibition testing. It is also possible that a suitably selected antibiotic administered by other means than the aerosol method may be effective.*

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GAS EMBOLISM AND THE RUBIN TEST

To the Editor:—*In reply to the question in* The Journal *April 12, 1952 (page 1364), whether gas (carbon dioxide) embolism has been seen after tubal insufflation, it is stated that there is no record of such an occurrence. May I call attention to my report published in* The Journal (89:522-523), *Aug. 13, 1927, in which two cases of death following insufflation of the fallopian tubes were reported. In one patient, oxygen was used. In the second patient (a 36-year-old white woman), carbon dioxide insufflation was done; the tubes were found to be closed, and dilatation and curettage were then performed. The woman collapsed and died on the operating table. Necropsy, done a few hours after death, showed the tubes to be closed, with blood and gas in the right fallopian tube and numerous gas bubbles in the right side of heart, the inferior vena cava, and the right femoral vein.*

I thought you would like to be informed of this case to keep the record straight. Anent the fatality with carbon dioxide insufflation of the tubes that I reported, Rubin (Fertil. & Steril. **3**:179, 1952), *reported three embolic episodes. Luckily, these were not fatal, but they might have been. My case is, therefore, within the realm of possibility. To assume the occurrence of an air embolus owing to the curettage is a rather specious supposition. How many times after the millions of uterine scrapings that have been done has the occurrence of an air embolus been reported? This does not defend in any way the use of curettage after insufflation. I agree with Rubin that carbon dioxide insufflation is the therapy of choice. I believe the iodized oil method and other methods should be used only to determine the exact site of the tubal obstruction, if the patient desires operative interference.*

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SYPHILOPHOBIA

To the Editor:—*In* The Journal, *March 8, 1952, page 877, the answer to a query on syphilophobia states:*

> *"There are some physicians who feel that if such efforts fail, he [the patient] might be given treatment with repository penicillin for early syphilis as a psychotherapeutic measure, provided there is some chance that this treatment will eradicate his phobia. Others suggest that placebo injections of sterile water would be equally effective."*

The treatment of a severe phobia such as described in this case by penicillin or by placebo injections is illogical. There could be no better way of reinforcing and fixing the syphilophobia than by treating the patient for the nonexistent disease that he fears. Confirming his fears by such injections not only would not eradicate his phobia but would make subsequent psychotherapy just about impossible.

This patient is in urgent need of psychiatric help. Syphilophobia in a 30-year-old male after extramarital intercourse points to the presence of severe guilt feelings and emotional conflict. Such a phobia may, in certain instances, be the first symptom of a more malignant condition, such as schizophrenia.

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To the Editor:—*This letter concerns a reply to a question about syphilophobia, which appeared in the March 8 issue of* The Journal *under Queries and Minor Notes.*

Certainly the rationale for treating a psychiatric condition with penicillin is about as logical as treating a surgical condition with psychotherapy. I feel sure that most other psychiatrists will agree that the use of penicillin in syphilophobia is not only useless but harmful. Statements suggesting a totally inappropriate therapy for psychiatric conditions appear all too often. Why cannot psychiatric treatment be openly recommended for an obviously emotional condition?

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ANESTHESIOLOGY: A CHALLENGE

CHAIRMAN'S ADDRESS

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What I have to say is neither scientific, pedagogic, nor semantic, but consists rather of a forthright analysis, in retrospect, of the challenges a physician accepts in the field of anesthesiology. Have we ever stopped to consider our reasons for studying anesthesia and why others should join us? Have we ever wondered why anesthesiology catapulted from an empirical art to an accepted specialty based on sound laboratory and clinical studies in the short span of three decades,[1] why residencies increased in number from 38 to 196 in 10 years,[2] why the American Medical Association, the armed forces, and various institutions accepted anesthesiology as a specialty,[3] why our membership soared from 3,500 to over 3,800 this past year, and why in this postwar era there has been an ever-growing demand for anesthesiologists [4] by the surgeon, patient, and hospital administrator? There must be some cogent reasons for this phenomenal progress and for the entrance into our specialty of interns, general practitioners, and doctors of physiology and pharmacology. What challenges in anesthesiology are being accepted? One should pause to reflect whether any or all have been accepted.

It should be clearly understood that the anesthesiologist is first a physician and only secondly a specialist. His field, in its entirety, was accepted by him as the ideal *modus operandi* through which the Hippocratic Oath can best be fulfilled. Accordingly, his duties, obligations, and responsibilities are carried out resolutely and cheerfully, for he is certain that it is his optimal approach in providing comfort and care to the sick and wounded. There is no place in anesthesia for those who are not prepared to accept this fundamental challenge. "The great use of life is to spend it for something that outlasts it."[5] No doctrine of ethical conduct that overlooks this wider destiny for person and community has anything but a stopgap value.

The history of anesthesiology dates back to time immemorial [1]; however, a survey of its progress during the past century reveals a series of doubts, claims, accusations, and defiances that have run the gamut from a single clinical trial to a congressional controversy, from a wave of enthusiasm for a new anesthetic agent to a wave of condemnation, until it has been stabilized and accepted as a result of sound experimental and clinical evaluation. Such a mixture of doubt and certainty is not accidental. It is the essence of scientific method; moreover, it is not a characteristic peculiar to science. Anesthesiology was destined, therefore, to flourish but not without its trials. The decisions in some of these challenging problems must be left to posterity—for others to make.

PROFESSIONAL CHALLENGE

The first challenge is the obvious and important professional one engendered of necessity by the rapid strides in the field of surgery. Anesthesiology and surgery are inseparable fields, interdependent and having mutual interest. The anesthetic requirements of surgery, we well know, have changed from the transient analgesia and unconsciousness of the old days to the elaborate, highly technical needs of today. Our challenge is to provide what may now be termed modern anesthesia. It may be defined as a reversible depression of the central nervous system (general anesthesia) with loss of pain and consciousness, graded skeletal muscular relaxation, control of adverse autonomic reflexes, and minimal interference with heat and fluid balance and with the activity of the other vital systems of the body. Such a challenge has extended modern anesthetic management to include preanesthetic and postanesthetic care and the supervision of fluid and inhalation therapy.[6] It demands not only finesse in technique but, more important, a constant

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Read before the Section on Anesthesiology at the 101st Annual Session of the American Medical Association, Chicago, June 11, 1952.

[1] Keys, T. E.: The History of Surgical Anesthesia, New York, Schuman's, 1945.

[2] Approved Residencies and Fellowships, report of the Council on Medical Education and Hospitals, J. A. M. A. **147**: 405 (Sept. 29) 1951.

[3] (*a*) Report of the Council on Medical Education and Hospitals, J. A. M. A. **111**: 2213 (Dec. 10) 1938. Essentials of a Registered Hospital, report of the Council on Medical Education and Hospitals, ibid. **114**: 1259 (March 30) 1940. (*b*) Walson, C. M.: Station Hospital Organization, Army Medical Bulletin 54, United States Army, October, 1940, p. 3.

[4] Need for Anesthesiologists, editorial, J. A. M. A. **145**: 906 (March 24) 1951.

[5] Mumford, L.: The Conduct of Life, New York, Harcourt, Brace and Company, Inc., 1951, p. 130.

[6] Martin, S. J.: The Present-Day Anesthesiologist, J. Iowa M. Soc. **40**: 438 (Sept.) 1950.

alertness to the principles of physiology and pharmacology to avoid aberrations during anesthesia. Today, no one denies that surgical and anesthetic morbidity and mortality have been significantly decreased by the present methods of anesthetic management.

The professional challenge to anesthesiology has come also from quarters other than surgery. The problems of obstetric analgesia and anesthesia and of infant resuscitation have confronted us for many years.[7] Recently, these problems, like those of pediatric anesthesia,[8] have been studied with such zeal that specialties within our specialty have developed. Some difficulties of internists have been shared also by us. Medical literature is replete with reports of diagnostic and therapeutic regional anesthetic techniques and of the treatment of gas and drug poisoning of the nonsurgical patient.[9] The general practitioner has also presented a challenge to anesthesiology in his search for analgesic substitutes and methods for treatment of asphyxia and the control of convulsions. In a sense, therefore, the professional challenge to anesthesia is inescapable and permanent.

Anesthesia is now an established specialty, and its continued progress is assured. The pertinent question is "How have we met its professional challenge?" Have we eased the surgeon's burden and contributed to his success? Have we made our services indispensable to the hospital's staffs? Have we developed the *esprit de corps* with our surgical and medical confreres that results in mutual respect, admiration, and esteem? In the course of our patient-relationship, have we succeeded in obtaining the patient's confidence, cooperation, and good will? Do we possess adequate knowledge of the basic medical sciences, proficiency in our techniques, and mature judgment in our anesthetic management? Our continued progress proves that the vast majority of anesthesiologists have successfully met the professional challenge.

EDUCATIONAL CHALLENGE

Of importance, also, is the educational challenge to anesthesiology. Our pioneers both here and abroad were self-taught and self-desciplined, advancing only through the uncertainties, setbacks, and rewards of trial and error, with limited means of public expression and professional communication. There was an occasional preceptor whose clinical (and perhaps laboratory) experiences were mature, organized, and enthusiastically presented to apprentices. We should praise such men and their works, but a still more important duty is to improve on what they have done. Our educational challenge was finally crystallized by the inauguration of the residency or fellowship program of instruction in anesthesiology in 1927. As our appreciation and correlation of basic medical sciences developed and our application and perfection of newer anesthetic agents and techniques improved, instruction became more fruitful and served as a great stimulus to our progress. The American Board of Anesthesiology was founded in 1937, our Section on Anesthesiology was authorized by the American Medical Association in 1940,[3a] and in the same year the specialty was recognized by the Army.[3b] Recognition by civilian hospitals and universities promptly followed. Necessities of World War II and its civilian aftermath created an even greater demand for instruction in anesthesiology. Today, enthusiasm for teaching others is predominant, while the urge to learn flourishes.

A variety of instruction programs in anesthesiology are available and vary from that qualifying a physician for certification by the American Board of Anesthesiology to one day sessions for those interested in anesthesiology on a part-time basis. There are now 196 approved residencies for some 600 candidates, a few preceptorships, and over 110 instruction centers for the general practitioner for one day to six month courses. In addition, there are annual refresher course programs sponsored by the Committee on Medical Schools and Postgraduate Education of the American Society of Anesthesiologists and countless numbers of regional, state, county, municipal, and local hospital meetings on anesthesiology. The challenge to our educational needs has been met also by innumerable laboratory investigations by both fellow anesthetists and workers in the fields of physiology, pharmacology, and chemistry. Such studies have been mutually stimulating and productive. They have proved to be indispensable in the scientific development of anesthesia.[10]

The educational challenge to anesthesiology, while slow in onset and only recently met with vigor, is a natural one. Our status as a specialty is relatively new and our progress gratifyingly rapid. Have we been immature, naïve, and overly impulsive in meeting our educational challenge, or have we not met it adequately? Without question, it appears that we have fallen short of our goal. Our national and state placement committees as well as many centers of instruction have been virtually deluged with requests for anesthesiologists. Although our society membership is increasing annually, the disparity between the number of demands and the number of available competent anesthesiologists continues to increase. Urgent requests have come not only from surgeons and hospital superintendents but, significantly, from lay members of hospital boards of trustees as well. This situation is acute, and our educational challenge now is formidable; however, our over-all teaching effort and facilities can absorb three times the number of trainees. What, then, is our problem? Bluntly speaking, it is our inability, by precept or example, to entice enough future anesthesiologists. Our failure, in a great measure, is due to inadequate or improper indoctrination of medical school students and interns in the principles and practice of anesthesia. Our educational effort could be most productive at this grass roots level, and it must be strengthened.[11] One wonders if the responsibility for meeting this challenge could not be shared with the American Board of Surgery, the deans of medical schools, and the directors of house staff instruction.

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[7] Snyder, F. F.: Obstetric Analgesia and Anesthesia: Their Effects upon Labor and the Child, Philadelphia, W. B. Saunders Company, 1949. Hershenson, B. B.: Modern Trends in Obstetrical Anesthesia, Am. J. Obst. & Gynec. **63**: 559 (March) 1952. Little, D. M., Jr., and Tovell, R. M.: The Role of Analgesia and Anesthesia in the Production of Asphyxia Neonatorum, J. Indiana M. A. **42**: 201 (March) 1949.

[8] Leigh, M. D., and Belton, M. K.: Pediatric Anesthesia, New York, The Macmillan Company, 1948. Martin, S. J., and Feeney, T. M.: Anesthesia in Pediatric Urology, J. A. M. A. **148**: 180 (Jan. 19) 1952.

[9] Papper, E. M., and Rovenstine, E. A.: Symposium: Control of Pain: Newer Analgesic Methods and Agents of Value in Control of Pain, New York State J. Med. **50**: 281 (Feb. 1) 1950. Lundy, J. S.: Symposium on Abdominal Surgery: Remarks Concerning Diagnostic and Therapeutic Blocks for Some Types of Abdominal Pain, S. Clin. North America **30**: 1109 (Aug.) 1950. Adams, R. C., and Ridley, R. W.: Value of Anesthesiologist to Various Fields of Medicine and Surgery, J. Missouri M. A. **47**: 658 (Sept.) 1950.

[10] Dripps, R. D.: Research and Its Relationship to Clinical Anesthesiology **10**: 690 (Nov.) 1949.

ECONOMIC AND ETHICAL CHALLENGES

Economics and ethics in the practice of anesthesia constitute two more significant present-day challenges. Their thorough appreciation and application is imperative if anesthesiology is to survive the insidious growth of the institutional practice of medicine, if we are to retain free enterprise in medicine, and if we are to continue to provide the modern anesthetic management our patients need and our surgeons demand. The administration of anesthetic agents is the practice of medicine, and, when it is regarded as such, the relationship of the anesthesiologist to his patient becomes clear-cut and inviolate. His conduct and responsibilities are similar to those in any other patient-physician relationship. The American Medical Association's "Guides for Conduct of Physicians in Relationships with Institutions,"[12] approved last December, and the American Society of Anesthesiologists' "Statement of Policy"[13] have been invaluable statements of our challenge and course of action in the best interest of our patients. Admittedly, these reports have served as a great impetus to our progress.

The Committee on Economics of the American Society of Anesthesiologists, in an extensive study in 1949,[14] revealed that 75% of the anesthesiologists in this country were engaged in private anesthesia on a fee-for-service basis. Of even greater importance is the 1951 report of the Armed Forces Committee of our society,[15] which showed that close to 80% of anesthesiologists are engaged in the private practice of anesthesia. This is very significant, for with such an economic future and accepted ethical standard, more will enter our profession; however, there is still much to be accomplished to meet our economic challenge. If the stand of the American Medical Association and the American Society of Anesthesiologists is correct and if the national trend is in favor of the policy of private fee-for-service anesthesia, then is it irreconcilable that 20% of our members do not conform? Is their interpretation of the national policy on economics and ethics in the practice of anesthesia based on local, regional, or other factors not yet clarified or known? Or, is it the dogmatic and persistent opposition of the American Hospital Association that makes conformity difficult?

SOCIOLOGIC CHALLENGE

Another challenge to anesthesia, one of increasing significance at long last, is the sociologic challenge. This is to be expected, for anesthesia, like other specialties, cannot divorce itself from the role played by medicine in contributing to America's leadership in world health and achievement. While the anesthesiologist is first a physician and secondly a specialist, he must not forget that he is also a citizen and a member of a community. Accordingly, he welcomes his community responsibilities.[16] His participation in various charity drives and his membership in athletic, political, and, religious groups provides him the opportunity to show understanding and leadership as a representative of the medical profession. With the individualization of anesthetic management for optimum safety and success, the close patient-physician relationship, the support by surgical colleagues, and the favorable commentaries on anesthesiology in lay journals, the public has become less mystified, less fearful, and less reluctant to accept anesthesia. In fact, some patients have gone further and not only inquire about the anesthesia they are to receive but demand the services of an anesthesiologist. History repeats itself, for it was only about two decades ago that parturient women began to prefer the services of the obstetrician and the general practitioner trained in obstetrics to those of the midwife or other technician.[17]

Anesthesia is an indispensable adjunct to the modern practice of medicine. As we meet our sociologic challenge successfully, the public will join our medical colleagues in contributing to our progress and stimulating us to greater effort. With increasing public demand and the already established need for modern anesthesia by our medical colleagues, it is not too early to predict the fulfillment of the recommendations of the American College of Surgeons, concurred in by the American Medical Association, that[4] there be at least one competent anesthesiologist serving each hospital in which anesthetics are administered. It is also not too unrealistic to say that such a recommendation not only will improve public relations but will also make our professional, educational, and economic challenges in anesthesiology less arduous to meet and more willingly accepted.

SUMMARY

Anesthesiology as a more recently accepted specialty in medicine, is faced with many challenges. The professional, educational, economic, and sociologic challenges are of paramount importance at the present time. Considerable success has been achieved in meeting the professional challenge; however, there is much to be accomplished in meeting the educational, economic, and sociologic challenges, if anesthesia is to maintain its rightful respected place in medicine. Our efforts must be strengthened, and with the support of our medical colleagues and an enlightened and aroused public, we cannot help but succeed in meeting all our challenges. Let us do all in our power to follow a precept of Lucretius that "Men live in a mutual dependence and, like runners, transmit the torch of life."[18]

114 Woodland St. (5).

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11. Eckenhoff, J. E.: Annual Report of Subcommittee on Medical Schools, read at the American Society of Anesthesiologists, Inc., November, 1951.

12. Guides for Conduct of Physicians in Relationships with Institutions, Abstract of Proceedings of the House of Delegates at the Clinical Session in Los Angeles, Calif., Dec. 4-7, 1951, J. A. M. A. **147**: 1684 (Dec. 22) 1951.

13. Statement of Policy, read at the American Society of Anesthesiologists, Inc., 1951.

14. McQuiston, W. O.; Foregger, E., and Foregger, R.: A Survey of the Practice of Medical Anesthesia in the United States, read at the Section on Economics, American Society of Anesthesiology, Inc., 1949.

15. Tovell, R. M.: Analysis of Questionnaire of Armed Forces Committee, read at the American Society of Anesthesiologists, Inc., Nov., 1951.

16. Stewart, H. B.: Professional Obligations: Chairman's Address, J. A. M. A. **147**: 1005 (Nov. 10) 1951.

17. De Lee, J. B.: Obstetrics Versus Midwifery, J. A. M. A. **103**: 307 (Aug. 4) 1934.

18. Lucretius: De rerum natura, Loeb Classical Library, New York, G. P. Putnam Sons, 1924, book 2, lines 75-78.

ACUTE POISONING WITH SODIUM FLUOROACETATE (COMPOUND 1080)

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Edward W. Rees, B.Sc., Philadelphia, Robert H. Peters Jr., M.D., Lewis C. Reese, M.D.
and
Theodore Baker, M.D., Kingston, Pa.

Since the report of Kalmbach [1] on the rodenticide activity of fluoroacetate (compound 1080) this compound has been extensively used by government agencies, the armed forces, and privately employed exterminators. In spite of its high toxicity to all species, relatively few cases of human poisonings have been reported so far, none of which have ended fatally.[2] All of the victims took small, but unidentified, quantities of fluoroacetate. The toxic dose for human beings has not been established, but on the basis of animal studies it has been theorized as being approximately 5 mg. per kilogram of body weight. The case reported here ended fatally, pathological examination was performed, and the quantities of fluoroacetate in the organs were determined. For the experimental literature on fluoroacetate we may refer the reader to the recent reviews by Chenoweth [3] and by us.[4]

REPORT OF CASE

A white man, about 40 years of age, was found on Feb. 3, 1949, noon, in his bedroom at home in an unconscious state. He was admitted to the emergency ward with the information from his wife that he had probably taken sodium fluoroacetate.

Past History.—According to his wife, who had known him since birth, he never had any serious illness or operation until after being discharged from the Army after World War II. At this time he was hospitalized with battle fatigue for six months. He received shock treatment for "severe depression." He was discharged December, 1940, and readmitted from October to December, 1947. The diagnosis was manic depressive psychosis. His family was warned of danger of possible suicide. The family history was essentially negative.

Examination on Admission.—He was unconscious and had nystagmus of both eyes and slight muscular spasms. The heart rate was 92, rhythm was irregular, and no murmurs were heard. The abdomen was soft, and between muscle spasms peristalsis was heard.

Therapy and Course.—The stomach was lavaged and magnesium sulfate instilled. He was given a soft soap enema. Following this, nystagmus became pronounced and he had an epileptiform convulsion. For sedation, 2 grains (0.12 gm.) of phenobarbital (luminal®) sodium was given four times and 1/50 grain (1.2 mg.) of scopolamine hydrobromide. Blood pressure had fallen to 90/40 mm. Hg. Plasma was administered and three intracardial injections of 5 ml. of 1% procaine hydrochloride given. Oxygen was administered. Blood pressure rose to 120/180. Muscle spasms continued, perspiration became profuse, the face flushed, he frequently retched and occasionally opened his eyes, and frothing at the mouth occurred. At midnight blood pressure rose to 118/75, pulse 100, respiration 28, and temperature in the axilla 97; he was very restless. Because of excess mucus secretion 1/150 grain (0.4 mg.) of atropine sulfate was given and calcium gluconate was given intramuscularly. At 3 a. m. respiration was labored, and at 5 a. m. the patient was pulseless, ceased breathing, and was pronounced dead.

Pathological Examination.—The skin had a yellowish tint; the scleras were not markedly tinted. There was a small amount of tenacious mucus in the oral cavity. About 100 cc. of clear yellow fluid was found in the left pleural cavity. Numerous epicardial petechiae were mainly about the base of the heart and along the course of the coronary vessels. Two needle punctures were in the right ventricle near the septum. The myocardium was of average normal color and thickness. On standing the color changed to a coppery red color and it had a slightly mottled appearance as though there was interfascicular hemorrhage present. The endocardium and the valves showed no abnormalities. Coronary arteries were free of sclerosis and blood clot. No significant sclerosis was found in the aorta or arterial trunks, and the blood tended to remain fluid. Much diffuse dark red color was seen in the lungs. There were a few small petechiae on the diaphragmatic surface. The cut surfaces were dark red, and on pressure very little air or thick, sanguinous fluid could be expressed. There was intense congestion of the bronchial mucosa. A small amount of sanguinous mucus was in the bronchi.

Tan discoloration of the lower third of the esophageal mucosa was present, and many hemorrhagic partly confluent erosions were on the cardiac portion of the stomach. Toward the pylorus only the crests of the rugae were involved; there was no definite ulceration. The stomach contained about 100 cc. of dark brown fluid. Intense hemorrhagic erosions appeared on the crests of the duodenal mucosa. These became less numerous as the jejunum was approached and were not seen more than a foot beyond the ligament of Treitz.

On the cut surface of the kidneys a slight congestion of the arcuate vessels was observed. The bladder was distended with about 600 cc. of clear yellow urine. Many discrete and confluent petechiae were seen in the mucosa. The cut surface of the liver was more yellowish than usual. There was an irregular prominence of the sinuses suggesting irregular distribution of blood or extravasation. The gallbladder, spleen, and adrenals showed no pathological change on macroscopic examination.

There was a considerable quantity of subarachnoidal fluid of normal appearance present. The arachnoid was studded with whitish granules of 1 to 2 mm. in diameter, which resembled pacchionian granulations but were more widespread. On the floor of the fourth ventricle, immediately subjacent to the ependyma, there were several small petechia-like lesions.

From the Nesbit Memorial Hospital, Kingston, Pa. (Drs. Reese, Peters, and Baker), La Wall and Harrisson Research Laboratories, Philadelphia (Dr. Harrisson and Mr. Rees), and the Department of Pharmacology and Biochemistry of the Philadelphia College of Pharmacy and Science, Philadelphia (Drs. Harrisson, J. L. Ambrus, and C. M. Ambrus).

[1] Kalmbach, E. R.: "Ten-Eighty." War-Produced Rodenticide, Science **102**: 232 (Aug. 31) 1945.

[2] Gajdusek, D. C., and Luther, G.: Fluoroacetate Poisoning: Review and Report of Case, Am. J. Dis. Child. **79**: 310 (Feb.) 1950. Williams, A. T.: Sodium Fluoracetate Poisoning, Hosp. Corps Quart. (no. 1) **21**: 16 (Jan.-March) 1948. Wright, E. R.: Personal communication, cited in Hutchens, J. O.; Wagner, H.; Podolsky, B., and McMahon, T. M.: Effect of Ethanol and Various Metabolites on Fluoroacetate Poisoning, J. Pharmacol. & Exper. Therap. **95**: 62 (Jan.) 1949.

[3] Chenoweth, M. B.: Monofluoroacetic Acid and Related Compounds, J. Pharmacol. & Exper. Therap. part 2, **97**: 383 (Dec.) 1949.

[4] Harrisson, J. W. E.; Ambrus, J. L., and Ambrus, C. M.: Fluoroacetate (1080) Poisoning, to be published.

Microscopic Examination.—Ecchymoses was present in the epicardial fat, with diffuse venous congestion. Heart muscle seemed to be normal. There was a minimal degree of anthracosis in the lungs. Diffuse vasocongestion and scattered foci of alveoli showed endothelial proliferation suggesting passive congestion. In the bronchi there was marked hemorrhagic edema of the submucosa. There were foci of lymphocytes in the deeper tissues suggesting that there had been a chronic peribronchitis of a mild degree, probably existing for some time. There were a peribronchial node, slight anthracosis, and nodules of caseous tuberculous material with definite peripheral activity (lymphocytes, endothelial cells, and giant cells).

Atropic gastric mucosa was found in the stomach. Numerous erosions, without ulceration, were characterized as small submucosal petechial ecchymoses. There was diffuse tubular degeneration in the kidneys, affecting mainly the convoluted tubules and to a minor extent the collecting tubules and the loops of Henle. The convoluted tubules contained protein exudate. There was general vasodilation. The interstitial tissue was compressed so that it was hardly visible. The glomeruli seemed normal. There was slight, diffuse edema of the sinuses of the liver with some evidence of biliary stasis in the perilobular portions of the lobules and marked passive congestion of the splenic pulp.

In the adrenals there was marked lipoid vacuolization of the fascicular zone and partially of the reticular zone with occasional evidence of pyknosis. The medulla showed considerable evidence of antemortem degenerative changes. The pancreas was essentially normal. Occasional islets were seen in which there were cells with dense eosinophilic cytoplasm.

Adjacent to the substantia nigra there was diffuse vasodilatation with small to moderate sized extravascular hemorrhages. There were numerous small petecchial hemorrhages scattered through the corpora quadrigemina. There was evidence of bleeding both of fresh blood as well as blood that was present long enough to be split to hemosiderin in the phagocytic cells in the perivascular spaces.

TABLE 1.—*Calculated Quantity of Sodium Fluoroacetate in Organs of Patient*

Organ or Contents of Organ	Total Weight of Organ or Quantity of Material	Weight of Sample Tested	Fluorine, Mg. per Kilogram of Organ Weight	Fluorine Calculated to Sodium Fluoroacetate, Mg. per Kilogram of Organ Weight	Calculated Total Sodium Fluoroacetate in Organ, Mg.
Urine, from bladder	600 cc.	300 cc.	70.0	368.0	220
Liver................	1,726 gm.	210 gm.	11.1	58.4	100
Brain	1,450 gm.	237.5 gm.	14.4	75.7	109
Stomach content...	105 gm.	105 gm.	22.5	118.0	12.4
Kidney........ Right: Left:	170 gm. 170 gm.	117 gm.	12.3	64.7	11

TABLE 2.—*Fluorine in Normal Human Organs*[*]

	Organ		
	Brain	Liver	Kidney
Organs from 2 adult males, Zdarek[†]....	0.23-0.27	0.68-0.80	1.34-1.54
Individual organs from different adult males, Gautier and Clausmann[‡].......	3.07	2.13	0.95
Organs from 1 adult male, Roholm[§]....	....	0.50	1.10

[*] Measured in milligrams per 100 gm. of dry weight.

[†] Zdarek, E.: Über die Verteilung des Fluors in den einzelnen Organen des Menschen, Hoppe Seyler's Ztschr. f. physiol. Chem. 69:127 (Oct. 24) 1910.

[‡] Gautier, A., and Clausmann, P.: Le fluor dans l'organisme animal: C. cerveau, glandes, muscles, sang, lait, excrétions, Compt. rend. Acad. d. sc., Paris 157:94, 1913.

[§] Roholm, K.: Fluorine Intoxication, Copenhagen, NYT Nordisk Forlag, 1937; Fluorschädigungen, Leipzig, Johann Ambrosius Barth, 1937; Fluorvergiftung, eine "neue" Krankheit, Klin. Wchnschr. 15:1425, 1936; Fluor und Fluorverbindungen, in Heffter, A.; Heubner, W., and Schüller, J., editors: Handbuch der experimentellen Pharmakologie, Berlin, Julius Springer, 1938, vol. 7, p. 1.

Toxicological Examination.—Fluorine content of the organs and body fluid was determined according to the distillation method of Willard-Winter[5] using a thorium nitrate back titration, following preliminary ashing with an excess of calcium hydroxide to fix the fluorine. From these data the theoretical sodium fluoroacetate content was calculated. The results are shown in table 1. In table 2 the fluorine content of normal human organs is set forth on the basis of the literature.

COMMENT

A fatally ending case of fluoroacetate poisoning is presented. The clinical course suggests that in human beings the central nervous system as well as the cardiac effect of fluoroacetate are manifested. It is known from the experimental literature[6] that some species, such as rabbits, goats, horses, and spider monkeys, exhibit only cardiac symptoms; others, such as dogs, guinea pigs, and frogs, have only convulsions; and again others, including cats, pigs, chicks, and rhesus monkeys, show cardiac as well as central nervous system effects.

The pathological observations are essentially similar to those found by Foss[7] in acute intoxication with fluoroacetate of experimental animals. Human cases and animal intoxications with sodium fluoride exhibit pathological changes[8] indistinguishable from those seen when fluoroacetate is used. Since, however, sodium fluoroacetate is much more toxic than sodium fluoride, it seems likely that the above changes were caused mainly by fluorine, while the special biochemical actions of fluoroacetate produced no pathological changes of diagnostic significance. The death from fluoroacetate poisoning[6] seems to be due to the failure of the heart, exhaustion because of cramps of central origin, and probably toxic depression of the respiratory and vasomotor centers.

It is interesting that in the Langerhans' islands dense eosinophilic cytoplasm was seen. Although it is difficult to draw conclusions on the basis of one case, the observation may indicate a preservative action of fluoroacetate on the enzymic granules.

The distribution of fluoroacetate was relatively uniform in the organs tested. The high level in the urine indicates a rapid urinary excretion. This is in agreement with the findings of Ramsey and Clifford,[9] who found in poisoned rats an even distribution of fluoroacetate between brain, heart, liver, and kidneys. It is significant that in spite of the gastric lavage and the fact that death ensued about 17 hours after the ingestion of the poison, important quantities of the poison were found in the stomach. Brandl and Tappeiner[10] and Gadaskina and Shtessel[11] presented data on the excretion of fluoride through the gastrointestinal tract, and Charnot[12] described fluoride excretion in the bile. Thus it is possible that gastroenterohematic circulation of fluorine compounds exists. Because of these data and the high toxicity of fluoroacetate repeated gastric lavages seem to be advisable.

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[5] Official Methods of Analysis of the Association of Official Agricultural Chemists, ed. 7, Washington, D. C., Association of Official Agricultural Chemists, 1950.

[6] Chenoweth.[3] Harrisson.[4]

[7] Foss, G. L.: Toxicology and Pharmacology of Methyl Fluoroacetate (MFA) in Animals, with Some Notes on Experimental Therapy, Brit. J. Pharmacol. 3:118 (June) 1948.

[8] Tappeiner, H.: Zur Kenntniss der Wirkung des Fluornatriums (Zum Teil unter Mitwirkung von Dr. N. Oblonsky), Arch. f. exper. Path. u. Pharmakol., Leipz. 25:203, 1888-1889; Zweite Mittheilung über die Wirkungen des Fluornatrium, ibid. 27:108, 1890. Heidenhain, R.: Neue Versuche über die Aufsaugung im Dünndarm, Arch. f. d. ges. Physiol., Bonn 56:579, 1894. Hedström, H.: Skandinav. Vet. Tidsskr. 22:55, 1932. cited by Roholm (see table 2 §). Dalla Volta, A.: Zur Kenntniss des Experimentellen Flurornatriumvengiftung, Deutsche Ztschr. f. ges. gerichtl. Med. 3:242, 1924.

[9] Ramsey, L. L., and Clifford, P. A.: Determination of Monofluoroacetic Acid in Foods and Biological Materials, J. A. Offic. Agr. Chemists 32:788-797, 1949.

The total quantity of fluoroacetate recovered from the organs and body fluids tested was 465 mg. Since the patient weighed approximately 140 lb. (63.5 kg.), disregarding the fluoroacetate present in organs from which no sample was available for toxicological study, he ingested a minimum of 6 mg. per kilogram of fluoroacetate.

The therapy used in this case was essentially symptomatic: barbiturates to decrease convulsions, intracardial procaine hydrochloride to inhibit ventricular fibrillation, atropine sulfate to decrease bronchial mucus secretion, and plasma transfusion and oxygen to offset vasomotor and respiratory failure. Experimental studies have suggested the use of certain specific antidotes.[6] Of these monacetin was directed for the treatment of this case; however, it arrived too late to be employed.

SUMMARY

1. A case history of fluoroacetate poisoning has been described together with postmortem examination and chemical analysis of the fluoroacetate content of organs and body fluids.

2. It appears that there is no important difference between postmortem findings of sodium fluoroacetate and sodium fluoride poisoning. The additional biochemical effects of sodium fluoroacetate thus seem to have no pathological manifestation of diagnostic significance.

3. Because of indications of gastrointestinal excretion and reabsorption of fluorine compounds, repeated gastric lavages seem to be advisable.

4. Other therapeutic aspects are briefly discussed.

1921 Walnut St. (Dr. Harrisson).

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ELECTROENCEPHALOGRAPHIC CHANGES IN PROFESSIONAL BOXERS

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and

Albert J. Silverman, M.D., Denver

Since the days of the Roman gladiators, men have fought each other and other persons have watched. The sport of boxing, as it exists today, is largely the product of psychological forces that have not changed in essence for 2,000 years. It is the feeling of many persons that in spite of periodic criticism and the efforts to ban prize fighting, the sport will persist. Since it appears unlikely that athletic contests requiring physical contact will ever cease, the physician should attempt to safeguard the physical and mental health of the participants.

Much has been accomplished to protect the athlete in such sports as football and ice hockey, but protective measures for the amateur and professional pugilist have not kept pace. Jokl[1] in his monograph (1941), which is concerned with the medical aspects of boxing, recognized and deplored this situation, and he collected a wide variety of case material that was representative of possible sequelae due to boxing injuries. Raevuori-Nallinmaa[2] in a recent publication states his belief that the usual cause of death related to boxing is intracranial hemorrhage. Tragic and sobering though they be, deaths of this kind are relatively rare. More important, and certainly much commoner, are the mild psychic changes observed in a high percentage of boxers. These changes are due to brain damage, which is also responsible for the less frequently seen, so-called "punch-drunk" person, who is in fact in a state of traumatic dementia and reveals severe psychic and neurological abnormalities.

The syndrome of boxing encephalopathy is probably due to multiple concussion hemorrhages as well as to contusion and laceration of the brain. The "knockouts" and "technical or near knockouts" (representing states of commotio cerebri) are usually the result of a trauma that alters brain function. If the trauma is severe or is mild but repeated at intervals that do not permit the brain to return to normal functioning, permanent damage may result.

Many investigators have reported on the value of the electroencephalogram in the detection of brain injuries. Dow, Ulett, and Raff,[3] in doing electroencephalograms of persons immediately after injury, reported that a

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Mr. W. Asmus, Executive Director, and Mr. E. Bohn, Chairman of the State Athletic Commission of Colorado, assisted in the preparation of this study.

From the Electroencephalograph Laboratory of the Colorado Psychopathic Hospital and the Division of Psychosomatic Medicine, University of Colorado Medical Center.

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2. Raevuori-Nallinmaa, S.: Brain Injuries Attributable to Boxing, Acta psychiat. et neurol., supp. 60, p. 51, 1951.

mild cerebral trauma produced electroencephalographic changes that decreased in abnormality within a few minutes. If amnesia was associated with the trauma, but the patient was clear-minded when his record was taken, there was only a slight increase in the percentage of abnormal records. If consciousness was at all impaired at the time of the electroencephalogram, there were invariable, accompanying electroencephalographic changes. Abnormal findings recorded immediately after head trauma may become normal, however, for Dow and associates found that a larger number of records were abnormal when taken less than a half hour after injury. This points to some mechanism in concussion other than such factors as petechia, contusion, and emboli, each of which would take several days to disappear. Williams [4] found invariable electroencephalographic changes accompanying altered consciousness as a result of head injury and stated that concussion is the result of widespread disorganization of cerebral function. Strauss [5] found diffuse slowing in patients with clouded consciousness but noted a more selective slowing in frontal areas of patients showing facetiousness. Ward and Clark [6] indicated that concussion or localized blows to the cortex resulted in changes in the electroencephalogram identical to those produced by rapid rises in intracranial pressure. Jokl [1] supported this by his contention that at the *moment of* the blow to the head there was a sharp rise of intracranial pressure.

In their experimental studies Zimmerman and Putnam [7] reported that minimum cell changes could occur without electroencephalographic changes but found that when the trauma was severe enough to cause "incompletely reversible cell changes" there was a direct relationship between severity of cell changes and severity of electroencephalographic abnormalities. They also reported a reduction in voltages that was directly proportional to the amount of force applied. Heppanstall and Hill [8] stated that focal findings were generally significant of symptomatic rather than idiopathic disorders and that there was a greater chance of permanent electroencephalographic changes in persons who sustained head injuries when they were under the age of 20. Kaufman and Walker [9] reported that in about two-thirds of their patients convlusive disorders developed after acute and severe head injuries. Abnormal electroencephalograms were seen in 90% of those patients in whom seizures developed and in more than 75% of those without seizures. Focal abnormalities were noted in over 80% of the epileptics and in over 65% of the nonepileptics. Greenblatt,[10] reporting on post-traumatic patients with convulsions, fainting spells, psychoses, or other personality disorders, found that 68% had disturbed electroencephalograms. Regarding minor head injuries, Harris and co-workers [11] stated that abnormal waves may be found in hyperventilation only and that a good prognosis can be made from serial electroencephalograms in which the tracings become more normal. Other investigators, in discussing electroencephalographic changes following head injury, reported different findings. Amplitude asymmetries [12] and bursts of six to eight per second activity [13] were noted by some, while others [14] reported decreases in voltage and increases or decreases in frequency.

Thus, it is evident that the electroencephalogram has no specific pattern in head injury and that any abnormality may be seen, depending on whether there is a temporary physiological disturbance or actual organic damage. The site of injury also has some bearing on electroencephalographic findings.

METHOD AND PROCEDURE

In order to help prevent serious injuries or death, the state athletic commission of Colorado, acting on the recommendation of one of the authors of this article (E. W. B.), unanimously passed a regulation requiring periodic electroencephalographic examinations on all professional boxers performing in Colorado. Although several states recommend the examination in specific cases, the authors do not know of any other state in which such electroencephalograms are compulsory. The regulations as set up by the state athletic commission were as follows: 1. A professional boxer must have an electroencephalogram at least once a year. 2. In the case of a "knockout," an electroencephalogram must be done within two weeks of the injury. 3. Frequent, repeated examinations could be done in the case of a boxer with suspicious recording.

Our purpose in this article is to record the results obtained from the first year's electroencephalograms as an initial report and to attempt to evaluate the worth of the electroencephalogram in safeguarding the health of those who participate in boxing. A Grass eight-channel electroencephalograph was used. So-called active lead placements were left and right frontals, motors, occipitals, and anterior and posterior temporals. Reference electrodes included the ears and vertex leads. Additional leads were used in specific cases as indicated. Records were classified according to the method described by Gibbs, Gibbs, and Lennox.[15] Activation with hyperventilation was performed routinely. Monopolar and bipolar tracings were done in each case, and a natural sleep record was obtained whenever possible.

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RESULTS

Thirty electroencephalograms as outlined above were done on 24 boxers. Their age distribution (divided arbitrarily into four groups) was as follows:

No. of Men	Age Group, Yr.
3	18-20
12	21-23
5	24-26
4	27-29

Of the electroencephalograms done on these men, 4 showed severe disturbance, 5 were moderately dysrhythmic, and 15 were within normal limits; thus, the total number of records showing disturbance amounted to 37.5% of the series. This is well above the 10 to 15% reported by Gibbs, Gibbs, and Lennox [15] and others in control groups, whose findings are in accord with those in our own control series.[16] *These figures have statistical significance in that there are only three chances in a hundred that they are due to coincidence.*

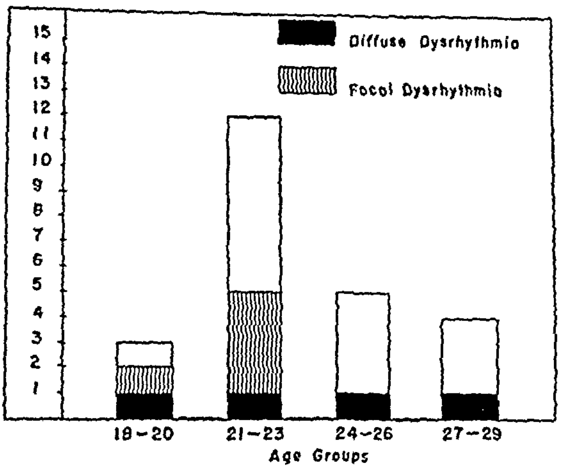

Fig. 1.—Incidence of abnormal electroencephalograms in boxers by age groups.

Type of Abnormality Seen.—There were four records showing severe disturbance. Three of these showed a temporal slow-wave focus, and one revealed diffuse slowing and paroxysms. Five records showing moderate disturbance were noted. Three of these were diffusely slow, and two were indicative of temporal slow-wave foci. Thus, in totaling the severely and moderately disturbed records, five were found to be focal and four showed diffuse findings. (In addition, paroxysms were seen in one each of the focal and diffuse records.) One record showing diffuse disturbance was found in each of the four age groups, while all the focal disturbances were present in the two youngest age groups. It was also noted that, although abnormalities were found in each age group, records showing disturbance seemed to be more frequent in the youngest men; but because of the few cases involved, this could not be statistically corroborated (fig. 1). In order to evaluate the report of decreased voltages in post-traumatic cases,[14] our records were also reviewed with this in mind, and six were found in which voltages were generally decreased. This was not significantly more than the 20% of low-voltage records expected in normal persons.

When we attempted to correlate history of knockouts with electroencephalogram disturbance, the following facts were noted: Ten of the 24 fighters had been knocked out in their ring careers at least once. Four of the 10 had dysrhythmic records, three being severely abnormal and one only moderately so. In the remaining 14 fighters, none of whom had ever been knocked out, one record showing severe disturbance and four showing moderate abnormalities were found. Because the number of records under study was not large enough, these findings are not statistically significant. It is noteworthy, however, that although there was an equal number of records showing disturbance in both groups, the men who had been rendered unconscious had electroencephalograms showing the severer disturbances. These findings were consistent with those of other investigators previously mentioned (fig. 2).

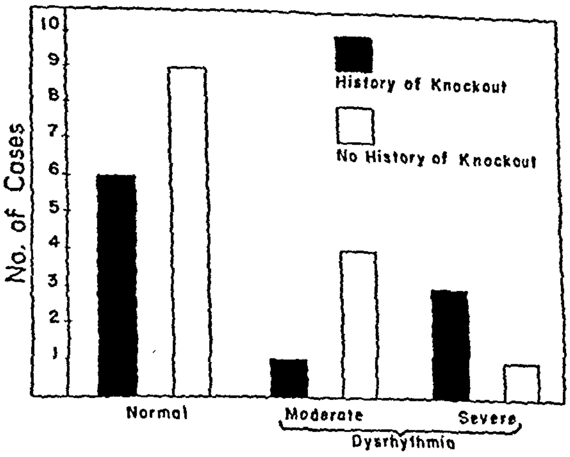

Fig. 2.—Comparative electroencephalographic findings of boxers with and without a history of knockout.

An attempt was also made to determine if length of ring career could be correlated with electroencephalographic disturbances, but no correlation was found. Electroencephalograms showing severe disturbances were seen in boxers who had been in from 2 to 50 fights, moderate dysrhythmias in men who had had from 1 to 15 fights, and normal records in men who had participated in from 1 to 106 fights. This lack of correlation is commented on below.

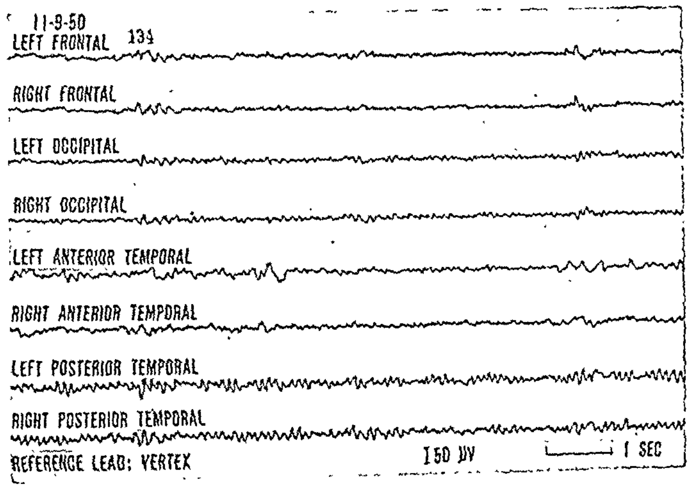

Fig. 3.—Initial electroencephalogram of professional boxer in case reported. Note the slow-wave focus in the left anterior temporal area.

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[16] In our control series of 329 patients, whose ages ranged from 18 to 54 years, it was noted that 36 (10.94%) showed moderate disturbances and 10 (3.04%) severe dysrhythmias, so that the total of records showing disturbance was 13.98%.

REPORT OF A CASE

The patient, a professional boxer, aged 21, was first seen by us in November, 1950. He had had three professional fights prior to his first electroencephalogram. In July, 1950, he was knocked out, but the following month he won his next fight by a technical knockout in the first round. Three days before a scheduled fight in November, 1950, the patient had his first electroencephalogram, which revealed a left temporal slow-wave focus. The fight was cancelled. A repeat electroencephalogram revealed more extensive temporal focal findings. (The patient, incidentally, showed no abnormal neurological or psychological signs or symptoms at any time.) He was temporarily suspended from boxing, but continued to train. A third electroencephalogram in January, 1951, revealed the persistence of the focus and the addition of paroxysms. This suggested that he had received additional brain injury during the period of training, from which he could not legally be prohibited. It was now believed that this man had suffered some degree of irreversible damage, and he was permanently suspended from boxing on Jan. 16, 1951, for his own good. It was felt that in this case the electroencephalogram had picked up intracranial damage before it had become clinically apparent and that late neurological disability or even death had possibly been averted by this decision (fig. 3, 4, and 5).

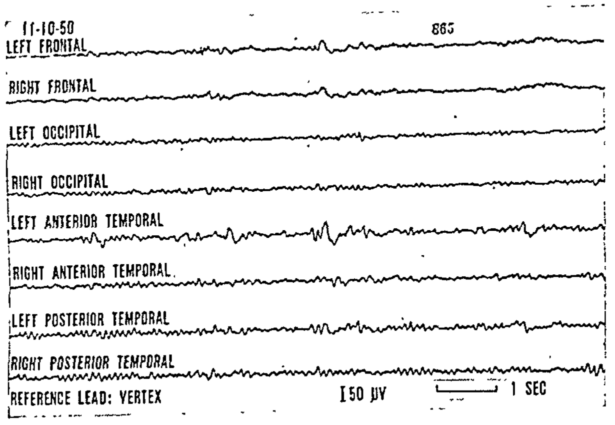

Fig. 4.—Repeat electroencephalographic examination of boxer in case reported. By comparison with figure 3, the focal disturbance now appears to consist of higher amplitude and slower waves.

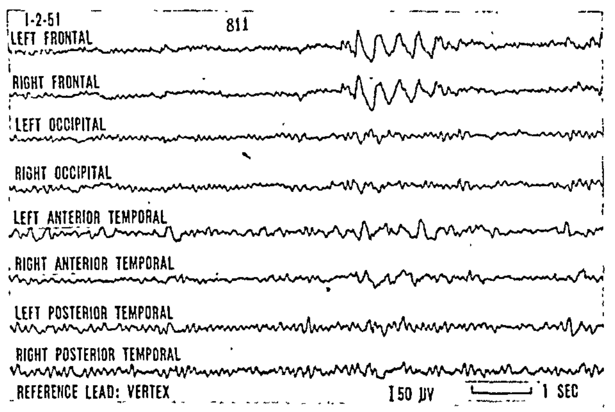

Fig. 5.—Final electroencephalographic examination of boxer in case reported. Note the persistence of the left anterior temporal slow-wave focus and its occasional reflection in the right temporal area. Note also the brief paroxysm of slow waves occurring in the frontal area.

COMMENT

Even in this small group of professional boxers used as a pilot study, it was at once apparent that their series of electroencephalograms were more frequently abnormal than those seen in control studies. It was noted that as many disturbed records were seen in the group who had not been knocked out as in their more unfortunate colleagues. In spite of this there was some indication that those who had definitely been knocked out showed severer disturbances. It should be pointed out that very little background material was actually available to us; for example, the number of times a man had been hit severely enough to cause him to be dazed without actual loss of consciousness, the number of amateur fights, and the extent and severity of training were not known. Thus, because of lack of such information, the correlation between number of professional fights and the electroencephalogram reading had no apparent meaning.

Concerning the abnormalities seen, diffuse records were found in every age group, while focal records were confined to the younger men. Thus, abnormal findings appeared more frequently in the younger age groups because of this very preponderance of focal records in them. These findings support the well-known fact that focal disturbances are good evidence of actual brain damage. There were relatively fewer records showing disturbance in the older men in our study. One can speculate that these men, for the most part, had been fighting for years and, having escaped brain damage in their first fights, were more experienced and better able to avoid injury in the ring. In addition, attention should be directed to the established fact that there are individual differences regarding susceptibility to trauma, so that in any group of boxers exposed to injury over a period of time it is likely that the more susceptible men are soon damaged permanently and retire from the ring, leaving their less susceptible fellows to carry on. An interesting clinical observation made during this study was that the men who showed focal temporal electroencephalographic abnormalities appeared to be, generally speaking, much more aggressive while fighting; and it is our feeling that this aggressiveness, together with the hint of impaired judgment as noted, may be associated in some way with the temporal lobe foci seen in these men. We feel that case material provided by boxers will provide much information about minor repeated head injuries, and future, better controlled, more extensive studies on larger groups may bring much interesting knowledge to light.

SUMMARY AND CONCLUSION

Compulsory electroencephalographic examinations were made on 24 professional boxers with the cooperation of the state athletic commission of Colorado. A statistically significant, increased incidence of dysrhythmic electroencephalograms was found in nine boxers (37.5%) in our series. There was some indication that men who had been knocked out showed severer electroencephalographic disturbances than those who had not been knocked out. Focal disturbances were more frequent in the younger men than in the older men, who also, generally speaking, had fewer electroencephalographic disturbances. A case of a boxer is presented who was finally suspended from boxing largely on the basis of progressive encephalographic abnormalities. From the above findings it is concluded that periodic, compulsory electroencephalographic examinations contribute to the protection of boxers and should be a required part of routine examinations.

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J.A.M.A., Aug. 23, 1952

DETECTION OF OCCULT BLOOD AS A ROUTINE OFFICE PROCEDURE

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Death from malignant diseases now holds the second position as a destroyer of human life in America. Most physicians have had the disquieting experience of making the diagnosis of malignant disease too late to be able to offer any effective treatment. Our great hope for the future lies in the discovery of some agent that will destroy malignant tissue wherever it exists or perhaps will even prevent the very inception of malignant disease. Until the day when such an agent becomes a practical actuality, we must content ourselves with the use of the tools at hand.

A nation-wide campaign is under way to educate the public in the early detection of cancer. It therefore behooves us as physicians to reexamine and to use every practical means available to detect the presence of cancer in its early phases. The American Cancer Society and the National Cancer Institute have found that cancer of the alimentary tract accounts for one-half of the deaths resulting from cancer in the United States.

In 1949, Hoerr, Bliss, and Kauffman [1] presented an interesting and provocative study concerning the use of the guaiac test as a screening procedure for the detection of gastrointestinal cancer. These authors based their study on stool specimens collected from unselected hospitalized patients and suggested a simple method of stool analysis with the use of guaiac reagent. They expressed the opinion that the guaiac test is a satisfactory screening procedure in the detection of occult bleeding, because it is not highly sensitive and therefore does not require the use of a meat-free diet. The authors further suggested the use of this test as a routine office procedure.

It is the purpose of this report to present a follow-up study of this procedure as applied to office practice and to determine whether the test has practical application as a screening procedure for gastrointestinal bleeding caused by cancer of the alimentary tract.

THEORETICAL BACKGROUND

Since 1864, when van Deen [2] first used guaiac reagent for the detection of blood, this reagent has been widely used in several modified and refined procedures. However, after Adler and Adler [3] developed the benzidine test, in 1904, and especially after it was refined by Gregersen,[4] in 1919, it became apparent that the guaiac test was not particularly sensitive for the detection of occult blood. As other more sensitive tests were developed, such as phenolphthalein, orthotolidine, ferrocyanide, and spectroscopy, the guaiac test fell into general disuse. It was no longer considered sensitive enough for practical application.

Several articles have appeared in the literature describing comparisons of the sensitivity of the various tests for the detection of occult blood. The guaiac test has generally been considered the least sensitive. Peranio and Bruger [5] recently reported their studies of the comparative sensitivity of the orthotolidine, benzidine, phenolphthalein, and guaiac tests on the feces of normal subjects who ingested measured amounts of blood. They found that, when their subjects were placed on a meat-free diet and given 1.0 cc. of blood, the orthotolidine test would give a positive reaction but that the others would not. When 5.0 cc. of blood was given, the benzidine and phenolphthalein reactions also became positive but not the guaiac reaction. It was necessary, however, to feed their subjects 20 cc. of blood before the result of the guaiac test also became positive; even when their subjects were placed on a regular diet, the result of the guaiac test did not become positive unless they were also fed 2 to 3 cc. of whole blood.

Schiff and co-workers [6] found that, when subjects were placed on an unrestricted diet, 2 out of 14 stools gave positive guaiac reactions. A further survey of the literature, including seven separate studies,[7] indicates that, with an unrestricted diet, it requires 0.5 to 10.0 cc. or more of ingested blood to produce a positive guaiac reaction of the stool.

In searching for a test that might be applicable to the detection of occult blood in unselected patients on a general diet, one could not help favoring this least sensitive procedure, the guaiac test. Its critical reaction point appears to lie close to, but just under, positive, in the normal patient on an average diet. On theoretical grounds, based on the literature, one might expect to find that about 15% of unselected patients would have positive guaiac reactions with their accustomed diets and that the other 85% would have negative reactions unless they were bleeding into the gastrointestinal tract in quantities of 0.5 cc to 10.0 cc. of blood. Other factors, however, enter into this expectation and must be considered in any survey study of bleeding.

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First, we have considered primarily bleeding from the upper intestinal tract. Bramkamp[8] has demonstrated that it takes 90 times more blood to give a positive reaction when it is ingested orally than when it is mixed directly with the feces. These authors expressed the belief that pancreatic digestion accounts for this difference. Thus, one might expect to detect small amounts of blood lost from lesions in the colon or rectum much more readily than from lesions in or above the duodenum.

Second, the possible ingestion of such interfering substances as sulfonamides, salicylates, bismuth subnitrate, bromides, or potassium permanganate is an important factor. Gettler and Kaye[9] have reported on the influence of these substances on results of tests for occult blood.

Third, of course, the amount of meat the patient has ingested is a factor. Cowie[7d] has indicated that cooked meats seldom give a positive guaiac reaction, but 20 gm. or more of uncooked beef in the diet will produce guaiac-positive stools. White[7e] has stated that the guaiac test often gives negative results when ¼ to ½ lb. (113 to 227 gm.) of meat has been ingested. It would be anticipated that the patients who had eaten large quantities of meat the day or two prior to examination might be expected to have a positive guaiac reaction. Schiff and co-workers[6] found that stools from patients ingesting 250 cc. of blood may give positive reactions for as long as 10 days.

PROCEDURE AND CLINICAL MATERIAL

In this study the guaiac test was performed as recommended by Hoerr,[1] with the use of feces wiped from the finger cot after examination of the rectum. The group of patients consisted of the first thousand who were interviewed and examined during 1950 in our office. These patients were adults of both sexes who either were new patients or had returned for annual or routine examinations. Each patient also was subjected to the usual laboratory studies, including a complete blood cell count, determination of sedimentation rate, urinalysis, serologic tests, and fluoroscopic examination of the chest; many were studied in the laboratory for other conditions relative to their complaints. All patients were on their accustomed diets at the time of their examinations. The guaiac test was not used as the sole determinant as regards further studies of the gastrointestinal tract; reliance was placed primarily on clinical judgment. However, all patients who exhibited positive guaiac reactions were requested to submit stool specimens for benzidine testing after a three-day meat-free diet. Some patients who had negative guaiac reactions were nevertheless given benzidine tests when it was felt that such a procedure was indicated.

Thus, the patients were studied primarily on the basis of their clinical symptoms and findings according to the custom of office practice. We have attempted to apply the results of the guaiac test in this group in an unbiased manner and to check the ultimate findings against the reaction in order to evalute it as a screening procedure. The patients have been followed now for a period of one year in order that we might obtain any evidence of disease that was not manifest at the time of initial examination. Although some patients have been "lost," the group remains essentially intact.

GUAIAC-POSITIVE GROUP

There were 152 patients, or 15.2% of the total group of patients examined, who had positive guaiac reactions on routine rectal smear examinations. All these patients were requested to go on a meat-free diet for three days and then to submit a stool specimen for benzidine testing. Forty-one of these patients (27%) failed to cooperate by submitting such a specimen even after follow-up letters had been sent to them. This was somewhat of a surprise but seems to point out further the difficulties involved in testing large groups of persons for occult blood in the stool. It is not a test that enjoys much popularity with patients. The majority of patients who did not cooperate in this test were those who had presented no gastrointestinal complaint and in whom there was no evidence of gastrointestinal disease other than a positive guaiac reaction.

Of the 111 remaining patients whose stools were subjected to the benzidine test, 25 (22.5%) were found to have positive reactions. In this group a variety of bleeding gastrointestinal disorders came to light on physical, roentgenographic, and proctoscopic examinations. It is of particular interest that there were six instances of cancer of the gastrointestinal tract in this group. All were present in the large bowel. Four patients had frank adenocarcinomas; one patient had a diffuse degenerating polyposis of the colon, and the sixth patient had a lymphosarcoma that had invaded and ulcerated into the large bowel.

A further analysis of the 152 patients who exhibited positive guaiac stools revealed that 66 (43%) had some definite disorder of the alimentary tract. Five other patients were thought to have a definite disorder, but no diagnosis could be established. No disease was found in 74 (48%) patients, and in 7 others there was inadequate patient cooperation to permit a satisfactory conclusion. It is of further interest that, of these 152 patients, only 84, or 55%, disclosed any symptoms referable to the alimentary tract in their histories.

Among the 1,000 patients examined there were 48 in whom the application of the guaiac test procedure failed. All the failures resulted from an inability to obtain a satisfactory stool specimen on the examining finger cot. One patient in this group was found to have a benign rectal polyp.

GUAIAC-NEGATIVE GROUP

Eight hundred, or 80% of all the patients tested, had negative guaiac reactions. As with all the patients included in this study, each patient was evaluated regardless of the outcome of the guaiac test. Almost 49%, or 389 of the patients of the guaiac-negative group, had some complaint referable to the gastrointestinal tract. Only 94 of these patients exhibited evidence of a disorder of the gastrointestinal tract on physical examination. X-ray studies of the intestinal tract and/or proctoscopic examinations were felt to be indicated and were performed on 255 of the 389 patients who presented such complaints. A total of 165 instances of organic disorder were found in the total guaiac-negative group of 800 patients, and 154 patients in this group were found to have functional disorders to account for their symptoms. Some of the patients who had organic disorders and who had guaiac-negative stools were nevertheless found to have occult blood in the stools when subjected to the benzidine test.

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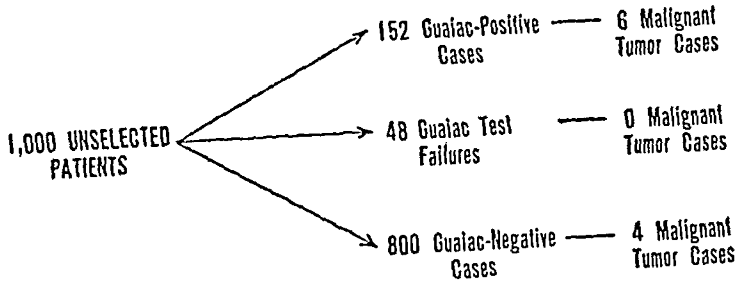
[8] Bramkamp, R. G.: The Benzidine Reaction: Some Observations Relating to Its Clinical Application, J. Lab. & Clin. Med. 14: 1087, 1929.

[9] Gettler, A. O., and Kaye, S.: The Phenolphthalein Test for the Detection of "Occult" Blood, Am. J. Clin. Path., Tech. Sect. 7: 77, 1943.

Since the primary interest in this report is concerned with the detection of malignant lesions, we do not feel that a report of other lesions found is appropriate here. Suffice it to state that the commonest disorders found were hemorrhoids and peptic ulcers. In this guaiac-negative group, however, four cases of cancer of the intestinal tract were found. In addition, two patients were found to have well-developed polyps, which, however, had not undergone malignant degeneration.

COMPARISON OF GUAIAC-POSITIVE AND GUAIAC-NEGATIVE GROUPS

Among the 1,000 patients comprising this study, 10 were found to have cancer of the alimentary tract. When the feces of these patients were tested with the guaiac method, four were found to be negative and six positive. A breakdown of the group is indicated in the accompanying figure.



Summary of the incidence of malignant tumors found in 1,000 unselected patients subjected to the guaiac test.

In spite of the theoretical background suggesting the practicability of employing the guaiac test as a screening procedure for occult blood, a comparison of the incidence of malignant disease in the patients studied rather clearly indicates that the test is unreliable for this type of problem. Gastroenterologists and others interested in the detection of malignant tumors of the alimentary tract have generally not been enthusiastic about the employment of occult blood tests. The studies of many investigators have clearly indicated that malignant tumors of the alimentary tract can best be detected by roentgenogram, the sigmoidoscope, and the rectal digital examination. A feeling of security cannot be warranted by a negative occult blood reaction when one is searching for malignant disease. Certainly the premalignant lesions are even less likely to be detected by this method alone.

SUMMARY AND CONCLUSION

A renewed interest in the guaiac test has recently been expressed. The application of this test for the detection of occult bleeding in patients on an unselected diet shows promise on theoretical grounds. A favorable clinical report has appeared in the literature.

In this study an attempt was made to apply the guaiac test to a large group of unselected patients encountered in routine office practice with particular interest in the detection of malignant lesions of the alimentary tract. This was done to determine whether the guaiac test had merit as a screening procedure.

The test itself is particularly easy to perform and requires little time and equipment. The patient need not be on a special diet, and therefore the test can be applied to almost all patients examined in the physician's office.

Unfortunately, however, in the group of 1,000 patients so tested, there were 4 patients with frank malignant lesions of the alimentary tract who did not have guaiac-positive stools as opposed to 6 patients with similar malignant lesions who did have guaiac-positive stools.

We therefore feel justified in condemning the use of this test as a screening procedure for the detection of malignant or premalignant diseases of the alimentary tract. While the various tests for occult blood in the feces undoubtedly have some merit in special situations, we heartily agree that the carefully taken history, the complete physical examination, x-ray studies, and the proctoscopic examination will disclose the presence of most malignant and premalignant diseases of the alimentary tract. So far, there does not seem to be a satisfactory shortcut.

We do not intend that this report should be construed to be a condemnation of occult blood tests in general nor the guaiac test in particular. When these tests are employed as an adjunct to clinical study and interpretation, we believe they have meaning; if they are used merely as a starting point for further study in screening patients, we believe the results will be most disappointing.

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Brachial Plexus Syndromes.—There is certainly no one cause for the variety of symptoms which have been from time to time attributed to pressure on the brachial plexus at the root of the neck. Equally certainly, the results of operations designed to relieve such pressure have not led to the survival of much enthusiasm for exploration of the plexus in any but the more uncritical minds. Even when a nervous syndrome is associated with a true cervical rib, removal of the rib appears all too often to do no more than perhaps to arrest the syndrome, and patients with even the largest cervical ribs often show no demonstrable evidence of local pressure when their lowest trunks are exposed. The discovery of a frank neuroma, though not unknown, is quite rare, in contrast with the known local effects of pressure on nerves in other regions.

The story of the treatment of such symptoms in patients who have no demonstrable cervical rib is a sorry record of uncritical, speculative theory despite much really careful observation. As a history of "arm-chair" pathology, the exposition of the scalenus anterior syndrome, of costo-clavicular compression, of abnormal fibrous bands and of unexplained and unmeasured hypertrophy of scalene muscles must have few equals, apart from the history of surgical thought about corresponding syndromes in the lower limb. These theories, just like many discarded concepts of backache and sciatica, were based either on equivocal eponymous physical signs or on attractive speculations on too few operation findings. The physical signs were usually interpreted without reference to an adequate series of normal controls, and the operations without reference to the dissecting room. The recent demolition of most of the accepted clinical "proofs" of compression in the neck has cleared away a good deal of ill-founded theory.—D. Ll. Griffiths, M.D., The Thoracic Inlet, *Journal o Bone and Joint Surgery,* May, 1952.

RESULTS OF EXPLORATION IN PATIENTS WITH CLINICAL DIAGNOSIS OF SUBDURAL HEMATOMA

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Medical literature contains many descriptions of the clinical features and operative findings in cases of subdural hematoma.[1] The criteria for diagnosis, however, are usually discussed only in terms of proved subdural hematoma. Little attention has been directed to cases in which a subdural hematoma was suspected clinically but was not found at operation. It is our chief purpose in this report to compare two such groups in order to evaluate better the diagnostic and prognostic features of this important clinical entity.

Another aspect of this disorder that has received relatively little consideration is the clinical course following surgical treatment. It is often the impression of those not intimately connected with neurological or neurosurgical services that recovery is to be expected after removal of a subdural clot. We shall show that this conception, while sometimes true in the individual case, cannot be applied to subdural hematomas in general.

CLINICAL MATERIAL STUDIED

One hundred patients admitted to the Third and Fourth Neurological Divisions of Bellevue Hospital (New York University Division) from 1948 through 1950 were studied. These represented all the patients during this three-year period in whom the diagnosis of subdural hematoma was considered seriously enough to warrant bilateral trephination or other definitive diagnostic procedures. The age of the patients ranged from 24 to 77 years. All but a few were examined by at least one of the authors. Of the 100 patients, 67 proved to have subdural hematomas or hygromas by trephination, and one was found unexpectedly at postmortem examination. These will be referred to as the "positive group." In the remaining 33 patients, subdural hematoma was excluded by trephination in 27 and by angiography in 6. These will be referred to as the "negative group." In general, the basis of the presumptive diagnosis was a combination of (1) a history or other evidence of head trauma, (2) mental changes, and (3) an unfavorable clinical course.

The material on which this study was made consisted of patients admitted to a large general hospital, including a disproportionately large psychiatric division. Many of these patients entered the hospital in a moribund condition or were out of contact in other respects, so that an accurate history could not always be obtained. A great many had had previous admissions for alcoholism, head injuries, and other disorders. The following features were investigated: (1) clinical course prior to operation, (2) operative findings, and (3) postoperative course.

CLINICAL FEATURES

Reason for Admission.—Of the entire series, 76% of the patients were admitted to the hospital because of changes in behavior or disturbances in the state of consciousness.[2] This preponderance of mental symptoms may be related to the fact that the psychiatric division accounts for more than one-third of all patients admitted to Bellevue Hospital. Eighty-one per cent of the positive group and 61% of the negative group were admitted for mental changes of relatively recent origin.

Only 16% in the entire group were admitted specifically for head injuries per se. The remainder of the patients came to the hospital because of seizures (5%), headache (2%), and hemiparesis (1%). Analysis of these features in the two groups failed to show significant differences.

History.—Trauma: There was a history or external evidence of head injury in every patient. Histories of alcoholism and the scars of old head injuries were common.

Seizures: Thirty per cent of the entire group had convulsions at some time prior to operation. Half of these had generalized seizures and half focal attacks. There was no significant difference between the two groups in respect to the number of patients with seizures or the nature of the attacks.

Examination.—State of Consciousness: On admission, less than one-third of the entire series were considered fully alert, the others showing various degrees of disturbance of consciousness. In this respect there were no differences between the two groups, but it is interesting to note that about one-fifth (19%) of the positive group were restless, often requiring mechanical restraint, whereas restlessness was not noted in any patient of the negative group.

Mental State on Admission: The patients were classified as (*a*) those with an organic mental syndrome (50%), (*b*) those whose mental state was clear (5%), and (*c*) patients with whom no verbal contact could be established because of unconsciousness, aphasia, or psychotic behavior (45%). Comparison of the two groups again failed to reveal any significant differences.

Neurological Findings: 1. Pupils. The pupils were considered abnormal (definite inequality or fixation in miosis or mydriasis) in 27% of the entire group, with no appreciable difference between the positive and negative

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[1] (*a*) Munro, D.: Cerebral Subdural Hematomas: A Study of 310 Verified Cases, New England J. Med. **227**: 87-95 (July 16) 1942. (*b*) Echlin, F.: Traumatic Subdural Hematoma—Acute, Subacute and Chronic: An Analysis of 70 Operated Cases, J. Neurosurg. **6**: 294-303 (July) 1949. (*c*) Kennedy, F., and Wortis, H.: "Acute" Subdural Hematoma and Acute Epidural Hemorrhage: A Study of 72 Cases of Hematoma and 17 Cases of Hemorrhage, Surg., Gynec. & Obst. **63**: 732-742 (Dec.) 1936. (*d*) Laudig, G. H.; Browder, J., and Watson, R. A.: Subdural Hematoma: A Study of 143 Cases Encountered During a 5-Year Period, Ann. Surg. **113**: 170-191 (Feb.) 1941. (*e*) Munro, D., and Merritt, H. H.: Surgical Pathology of Subdural Hematoma Based on a Study of 105 Cases, Arch. Neurol. & Psychiat. **35**: 64-78 (Jan.) 1936.

[2] They were found by their families or associates to be lethargic, acting peculiarly, or unable to speak as well as before. Sometimes they were found wandering, dazed, about the streets and were brought in by the police or ambulances as drunk or psychotic. The disturbances in consciousness varied from drowsiness to coma.

cases. Unilateral and bilateral pupillary abnormality also occurred equally in both groups. Seventeen patients (25%) of the positive group showed a dilated pupil on the side of the hematoma. Two patients, however, had dilated pupils on the side opposite the lesion, and in one of these the dilated pupil became smaller immediately after removal of the hematoma. Similar findings in regard to the pupils were reported by Echlin.[1b]

2. Eyegrounds. Papilledema, with or without retinal hemorrhages, was present in 15% of the entire series, but it occurred over three times as often in those with subdural hematoma (21%) as in those without it (6%).

3. Other neurological signs. Of all the localizing neurological signs, including hemianopsia, hemiparesis, hemisensory defect, focal seizures, reflex inequality, aphasia, and eye signs (noted above), only in respect to aphasia was there a marked difference between the two groups; four times as many in the positive group had aphasia. Because of the mental changes and disturbed states of consciousness, however, the actual number of patients with aphasia could not be accurately determined. Therefore, no significance could be attached to this apparent difference.

4. Value of signs in determining laterality of the lesion. As an indicator of the side of the pathological process, unilateral signs were reliable in only half (49%), the cases of one-sided subdural hematoma; in the remainder they were on the wrong side (4%), bilateral (22%), or absent (27%). Conversely, when hematomas were found on both sides, bilateral signs were present in only 26%, whereas they were unilateral in 48% and absent in the remaining 26%. In this series of patients, then, unilateral and bilateral subdural hematomas could not be differentiated on the basis of the neurological findings, nor could the side of a single hematoma be predicted accurately.

Laboratory Findings: 1. The cerebrospinal fluid was examined in every case. Some abnormality (elevated pressure, blood, xanthochromia, increased cells, or high protein) was found in 82% of all the cases. Normal fluid was encountered in only 10% of the positive group, whereas 26% of those without subdural hematoma had no abnormality. The major abnormalities (in over 80% of the cases) consisted of blood or xanthochromia (50%) and increased pressure (35%). The type of abnormality had no relation to the presence or absence of subdural hematoma. It should be emphasized that subdural hematomas were found even when the cerebrospinal fluid was completely normal.

2. Electroencephalograms were performed in 21 patients, 14 in the positive group and 7 in the negative group. The electroencephalographic findings were not specific for any type of pathological change, and similar records occurred in both groups. Most of the tracings contained unilateral or bilateral slow wave foci, with a localization usually corroborating the clinical or operative findings. Only one record was within normal limits (a proved subdural hematoma). Reduction of alpha activity in the region of the lesion, often regarded as characteristic of subdural hematoma, did not constitute a prominent feature of the records. Generally speaking, the greater the damage to the underlying brain, the greater the electroencephalographic abnormality; the presence or absence of subdural hematoma made no remarkable difference in the record.

3. Carotid angiography was performed in 12 cases, 6 in each group.[3] In our cases, the angiographic picture was characteristic when subdural hematoma was present and equally reliable in excluding it. In one patient, the postoperative angiogram showed that a previously demonstrated abnormality had disappeared.

Preoperative Course.—The only significant feature of the preoperative course in the two groups was fluctuation in the clinical picture, particularly the state of consciousness. Definite fluctuation occurred five times as often in the positive as in the negative group. Twenty-nine per cent of the patients with subdural hematomas had a fluctuating course, whereas only 6% of the negative group showed such variations.

The vast majority of patients of the entire series were operated on (or subjected to angiography) because of increasing disturbance of consciousness or failure to improve (95%); there were no appreciable differences between the two categories. The remainder (5%) improved to a great extent, but in two instances angiography revealed the presence of a subdural hematoma and trephination was carried out. In one of these cases, despite satisfactory clinical improvement, progressive displacement of a calcified pineal gland led to angiography and operation.

OPERATIVE FINDINGS

Of the entire group, unilateral or bilateral subdural hematomas were seen at operation in 67, and in one case postmortem examination disclosed a previously unsuspected clot. One-quarter of these patients also had other brain lesions, which could be seen well enough to be described by the surgeon at the time of trephination. These abnormalities included contusions, lacerations, subarachnoid bleeding, epidural clot, and intracerebral hemorrhage. The limited field of vision provided by burr holes, however, makes it probable that the actual incidence of associated lesions was much higher.

In the 33 cases of the negative group, both the surgeon's report and subsequent developments (clinical course, pneumoencephalography, or postmortem examination) indicated that no subdural hematoma was present. Some of the brains in these cases appeared normal to the surgeon, but in most of them (68%) laceration, contusion, congestion, subarachnoid hemorrhage, cerebral atrophy, arachnoiditis, or other lesions were discernible beneath the trephine openings or became evident on subsequent air encephalographic examination.

Thirty-eight of the subdural hematomas were considered recent by the surgeon on the basis of the observation of fresh, unchanged blood, recently clotted blood, or the absence of organization or membrane formation. Twenty-eight subdural hematomas were judged to be old, because of the color of the blood and the state of organization.

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[3] The angiographic examinations were made by Dr. Joseph M. Stein, who has made the detailed findings, indications, and technique the subject of a separate report (Stein, J. M.: Cerebral Angiography in Subdural Hematoma, Neurology, to be published).

[4] Deleted on proof.

An attempt was made to correlate the time between injury and operation and the surgeon's estimate of the age of the subdural hematoma. There was generally a significant relationship in cases of recent subdural hematoma. No "fresh" subdural clot was found in a patient whose lesion was two months old or more, and in only one was the lesion older than one month. In the "old" subdural hematomas, however, the correlation was not good, and the age of the lesion, according to the histories available, varied between one week and eight months. Munro[1a] has pointed out that the visual signs used to estimate the age of these lesions are not reliable.

POSTOPERATIVE COURSE

Of the group with subdural hematoma, 47% recovered sufficiently to be discharged to their homes. Twenty per cent required commitment to state mental hospitals or were discharged in the custody of their families because they failed to improve during the month after operation. Twenty-three per cent died, presumably as a result of their cerebral lesions or of other, though related, causes. Thus over half of these patients, with proved subdural hematomas successfully removed, either died or remained severely disabled. In five postmortem examinations, no cause of death aside from the brain lesions could be ascertained, so that these deaths, as far as we know, occurred as a result of head injuries and their complications. The mortality rate was only slightly higher (40%) in the group with fresh subdural hematomas than in those with older lesions (34%).

In the negative group, 36% were discharged to their homes improved, 30% were committed or discharged in custody because of failure to improve, and 34% died. Although a smaller proportion of the patients with subdural hematoma recovered, the over-all mortality and morbidity rates of the two groups were about the same.

Further analysis shows that complications (contusion, lacerations, and epidural hemorrhage) increased the mortality from subdural hematoma, since 47% of these patients died, whereas, in cases in which the brain underlying the subdural hematoma looked normal to the surgeon, the mortality rate was only 20%. In the negative group, the mortality in the presence of complications such as contusion or lacerations was only 25%. Thus, when a subdural clot is found in association with lesions of the brain itself, the prognosis is poor, even with successful evacuation of the hematoma.

In considering the immediate results of operation for subdural hematoma, we made a study of the time consciousness returned or improved after operation. In the positive group half (51%) of the patients who were destined eventually to live recovered consciousness within 24 hours. The remainder improved more slowly over the next few days, weeks, or even months. It is significant, however, that 24% of the negative group also recovered consciousness within 24 hours after operation or angiography, even though no subdural hematoma could be found. We cannot explain this coincidence, but it suggests that the natural course of the disease may play a more important role than is generally assumed.

COMMENT

From the data presented several points seem worthy of emphasis. First, the prognosis after successful removal of a subdural hematoma should always be guarded. This opinion is supported by the fact that the morbidity and mortality rates in the negative group were proportionately as high as in the positive group. Since the history and clinical course in both the positive and negative groups were so much alike, we cannot assume that the subdural clot per se was the predominant factor in causing death or even the significant clinical signs. It seems probable that the determining factor leading to death and disability is disease of the brain itself, whether this disease was acquired at the time of injury or existed before then. Essentially the same conclusion was reached by Browder and Rabiner[5] from the study of pathological material. A fair assessment of prognosis must consider both lesions, not only the obvious, removable, and therefore deceptive, subdural clot.

Although abnormalities in the state of consciousness are, of course, not pathognomonic for this condition, when they exist in combination with a history or other evidence of head trauma they constitute the only valuable diagnostic sign. This criterion led to a correct diagnosis in 68% of the entire series, which compares well with the diagnosis of other neurological entities.

Neurological signs, such as pupillary changes, aphasia, and hemiparesis, did not occur with sufficient frequency to be reliable. These signs probably reflect brain damage incurred by the trauma rather than pressure produced by the clot.

Reliance could not be placed on lateralizing signs, in view of the frequency of their occurrence in the group of patients without subdural hematoma. The number of hematomas proved to be bilateral or on the unsuspected side justifies the practice of performing bilateral trephinations in every case.

Two general schools of thought exist in regard to the electroencephalographic picture, characterized by reduction of alpha activity over the hematoma. The second believes that subdural hematoma may give rise to a wide variety of nonspecific electroencephalographic patterns, including the alpha "suppression," but also points out that areas of reduced alpha may appear in other pathological states. In our cases, electroencephalography was of no value in differentiating patients with subdural hematoma from those who had head injuries without subdurals. With one exception, the electroencephalograms were all abnormal. The finding that the severity of electroencephalographic changes was proportional to the severity of the damage to the underlying brain agrees with the conclusions of Kaplan, Huber, and Browder[6] and is in accord with experimental studies on the electrical activity of the brain following mechanical injury.[7]

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[5] Browder, E. J., and Rabiner, A. M.: The Role of Intracerebral Swelling in Subdural Hematoma, Tr. Am. Neurol. A., 1951, to be published.

[6] Kaplan, H. A.; Huber, W., and Browder, J.: Electroencephalography in Subdural Hematoma, Electroencephalog. & Clin. Neurophysiol. 3: 372 (Aug.) 1951.

[7] Cohn, R.: Subdural Hematoma: An Experimental Study, Arch. Neurol. & Psychiat. 59: 360-367 (March) 1948. Ulett, G.: Electroencephalogram of Dogs with Experimental Space-Occupying Intracranial Lesions, ibid. 54: 141-149 (Aug.) 1945.

The results of this portion of the study suggested that subdural hematoma does not give rise to a characteristic electroencephalographic pattern and that distinction between the various types of brain injury cannot be made by this means.

SUMMARY

1. One hundred consecutive cases of suspected subdural hematoma, occurring during a three year period, were reviewed. A comparison was made between two groups, 67 patients who had subdural hematomas and 33 who did not.

2. No reliable clinical features distinguished the two groups. When we used as criteria the combination of head injury, changes in consciousness, and failure to improve, the suspected diagnosis proved correct in two-thirds of the cases.

3. The number of deaths among the patients without subdural hematoma was proportionately as high as in those patients with subdural hematoma. Also, there was little difference in the incidence of permanent disability in both groups.

4. Disease of the brain itself, whether or not it was due to injury, was probably the chief determinant of mortality and morbidity.

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FAMILIAL AUTONOMIC DYSFUNCTION

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Up to 1949 there had appeared several isolated case reports [1] of what now seem to be examples of a definite clinical entity. This is characterized by evidences of vegetative dysfunction, such as excessive perspiration, drooling, erythematous blotching of the skin, intermittent hypertension, and defective lacrimation; hyporeflexia and emotional instability are also present. In 1949 my co-workers and I reported five illustrative cases.[2]

Since then our experience with the condition has grown with new cases and repeated observations on old cases. Other hospitals and physicians have made available the records of additional cases, so that we are now able to present a more definitive picture of the condition, based on a total of 33 cases. Their sources are shown in table 1.

TABLE 1.—*Sources of Thirty-Three Cases*

Hospital or Physician	City	No. of Cases
Babies Hospital	New York	11
Mount Sinai Hospital	New York	5
New York Hospital	New York	4
Children's Hospital	Boston	2
Dr. H. F. Saunders	Cleveland	2
Dr. H. S. Altman	New York	1
Dr V. Tappan	Tucson	1
Engel and Aring [1b]	Cincinnati	1
Bellevue Hospital	New York	1
Beth Israel Hospital	New York	3 *
Dr. F. A. Disney	Rochester, N. Y.	2

* Two of these cases have been reported in the literature (Aronson, N.; Stern, G. S., and Cohlan, S. Q.: Paroxysmal Hypertension Simulating Pheochromocytoma, Pediatrics 8:661, 1951).

CLINICAL FEATURES

In table 2 the common features are listed and their frequency in this series shown. These are arranged in two groups, the first (group A) being made up of those that are almost constant and the second (group B) of those that have occurred frequently enough to make them appear relevant.

Group A.—1. Race: All the patients who showed the full syndrome have been of Jewish extraction. We have seen three persons of other races who failed to produce tears when crying, but none of these has shown any of the other features.

TABLE 2.—*Occurrence of Features in Thirty-Three Cases*

Feature	No. of Cases with Information Available	Cases with Feature	
		No.	%
GROUP A			
1. Jewish extraction	33	33	100
2. Defective lacrimation	33	33	100
3. Skin blotching	31	30	97
4. Excessive perspiration	31	31	100
5. Drooling	27	27	100
6. Emotional instability	30	30	100
7. Motor incoordination	27	27	100
8. Hyporeflexia	31	30	97
9. Relative indifference to pain	22	22	100
GROUP B			
1. Hypertension	27	23	85
2. Cyclic vomiting	30	19	63
3. Frequent pulmonary infection	31	23	74
4. Frequent unexplained fever	24	16	67
5. Breath-holding spells in infancy	22	16	73
6. Urinary frequency	24	18	75
7. Mental retardation	31	17	55
8. Convulsions	32	16	50
9. Corneal ulceration	31	11	35

2. Defective Lacrimation: All the patients have failed to produce tears in normal fashion when crying. Usually this has been absolute, but in one or two cases an occasional tear has been seen. The eyes are not dry, and tear production seems adequate to maintain corneal health in most instances. The exceptions to this will be discussed in group B. It has been difficult to evaluate corneal sensation accurately, but in some cases complete anesthesia has been present and in others sensation seems to be diminished.

3. Skin Blotching: In all but one of the group erythematous, sharply defined blotches appeared on the face, shoulders, and upper part of the thorax when the patient was excited or was eating. These would disappear

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[1] (*a*) Day, R., and Klingman, W. O.: Effect of Sleep on Skin Temperature Reactions in Case of Acrocyanosis, J. Clin. Investigation **18**: 271, 1939. (*b*) Engel, G. L., and Aring, C. D.: Hypothalamic Attacks with Thalamic Lesion: Physiologic and Psychologic Considerations, Arch. Neurol. & Psychiat. **54**: 37 (July) 1945; Anatomic Considerations, ibid. **54**: 44 (July) 1945. (*c*) Wechsler, I. S., and Gross, I. H.: Some Clinical Manifestations of Hypothalamic Dysfunction, Confinia neurol. **7**: 89, 1946.

[2] Riley, C. M.; Day, R. L.; Greeley, D. M., and Langford, W. S.: Central Autonomic Dysfunction with Defective Lacrimation: Report of Five Cases, Pediatrics **3**: 468, 1949

within 10 to 15 minutes after withdrawal of the stimulus. They were usually (but not always, as will be shown below) symmetrical and tended to recur in the same areas. The one patient in whom this was not observed was 14 months old and showed only mottling of the extremities when excited.

4. Excessive Perspiration: All the patients perspired excessively, especially under conditions of excitement. In one patient in whom the skin blotching was asymmetrical, perspiration was also asymmetrical, particularly noticeable on the forehead.[1]

5. Drooling: In all about whom adequate information was available, drooling continued long past infancy. This was exaggerated during excitement.

6. Emotional Instability: Their emotional make-up was similar, in that they all responded to minor emotional stimuli in an "all or none" fashion. This made their relationship with other persons very difficult, and their management in the home has proved a major problem. Their span of attention is short, and usually their behavior is unpredictable.

7. Motor Incoordination: These children have all been awkward with their hands and have a peculiar shuffling gait. It is not possible to demonstrate regularly any specific neurological abnormalities except for the hyporeflexia to be discussed. They also have a characteristic thickening of speech.

8. Hyporeflexia: In most of the patients it has been impossible to elicit any deep tendon reflexes. In a few, however, these reflexes were obtained with difficulty but were definitely hypoactive. In two patients it was reported that reflexes could be elicited on some occasions and not on others. In only one patient were reflexes reported to be normal.

9. Relative Indifference to Pain: Information with regard to pain sensitivity was available in only 22 cases, and in each of these a relative indifference was present. Because of the strong emotional response of the patients to any stimulus, it was difficult to evaluate their response to pain itself. The abnormality was called to my attention by the fact that, when a limb was immersed in ice water for a cold pressor test after previous conditioning to the procedure, there was no complaint of pain even after 60 seconds. Subsequent questioning of parents revealed in many instances that their children did not seem to mind minor bruises. One child, in fact, had a broken wrist discovered only by the presence of swelling. This is not true anesthesia or hypesthesia, since the patients recognize pinpricks readily.

Group B.—In the next group of features are those which, although frequently dramatic and often associated with complaints, have not been present consistently.

1. Hypertension: Intermittent hypertension was a frequent finding evident in about 85% of the patients in whom it could be evaluated. It occurred in response to excitement, and not infrequently a resting blood pressure of 100/60 mm. of mercury would rise under the threat of venipuncture to 160/130 mm. Many of the patients who exhibited recurrent attacks of vomiting had hypertension persisting throughout the episode, with the blood pressure returning to normal levels as the vomiting subsided. One patient at times manifested such severe degrees of hypotension that he had "blackout spells." Recently this patient had bouts of vomiting associated with the expected hypertension and followed by severe and persistent hypotension associated with a shock-like picture; this seemed to be modified by treatment with cortisone. The hypertension has been so prominent that in five cases surgical exploration for pheochromocytoma was undertaken.

2. Cyclic Vomiting: Almost two-thirds of the patients had severe attacks of vomiting recurring at intervals ranging in different patients from a few days to several months. Associated with these attacks were personality changes of pronounced degree. A previously cheerful and cooperative child would become sullen, withdrawn, and destructive toward himself and others. Usually the vomiting and personality change would terminate abruptly after two to three days. At times these attacks appeared to be induced by known emotional upsets; at other times there was no apparent inciting factor. This was one of the most distressing and incapacitating features.

3. Frequent Pulmonary Infection: In 23 of the group, repeated bronchitis or bronchopneumonia caused an inordinate amount of hospitalization. In several of the children this began in the early months of life and was the basis for a tentative diagnosis of cystic fibrosis of the pancreas, for which condition careful studies were made. In those who have survived, the frequency of pulmonary infection has diminished with advancing years.

4. Frequent Unexplained Fever: Two-thirds of the patients in whom the facts were known had frequent fever that was not readily explainable. Before the diagnosis was made, this fever had been attributed in some instances to a disturbance of the "temperature-regulating center."

5. Breath-Holding Spells in Infancy: Information about this characteristic is available for only 22 patients. Out of these it was found that almost three-quarters gave a history of this phenomenon in the first year of life. When the patients were first seen, at the age of 4 or 5 years, breath-holding had usually been forgotten by the parents; they were reminded only by direct questioning.

6. Urinary Frequency: A large proportion of the children showed urinary frequency, which appeared to be the result of nervous tension rather than real neurogenic disturbance of the bladder.

7. Mental Retardation: Among the first patients in our series many had mental retardation, and it was originally thought that this was a regular feature; but as the series has grown the findings have been less consistent. More than half the patients have shown mental defectiveness; on the other hand, several of those who were not retarded have shown actually superior intellectual endowment.

8. Convulsions: Convulsions have occurred in half the patients. In some cases there has been a history of repeated convulsions, but usually only one or two seizures associated with fever have occurred.

9. Corneal Ulceration: The last clinical manifestation of significance is one that seems to be secondary to one of the cardinal features, defective lacrimation. Although in most instances there was no interference with corneal health, in 11 cases corneal ulcers developed either with slight trauma or after no known trauma. Usually these healed rapidly, but in three patients healing was so unsatisfactory that permanent scarring developed and seriously interfered with vision.

Miscellaneous.—Two other findings have been noted that suggest muscular disturbance secondary to central nervous disorder but that are too infrequent to be considered necessarily part of the picture. Scoliosis has been found in five of the patients. In one of these it was of severe degree. "Pes cavus" has been reported in two cases.

SPECIAL TESTS

The diagnosis of this condition depends for the most part on the observation of the clinical features discussed above. While many exploratory investigations have been carried out in these cases, in general these do not add to an understanding of the disturbance. Signs of basic metabolic disturbance, such as hypoglycemia or electrolyte imbalance, have not been present. Nitrogen retention has been observed only during periods of dehydration associated with vomiting, except in one case, when death appeared to be due to uremia that was thought to result from renal insufficiency secondary to hypertensive episodes, since nephrosclerosis was observed at autopsy. In general, however, chemical abnormalities have not been remarkable.

The result of the cold pressor test has been abnormal when used. If the patient was previously acclimated to the procedure, so that emotional factors did not intrude, there was no change in blood pressure. It is suggested that the indifference to pain previously discussed explains this lack of reaction.

The benzodioxane test[3] has been carried out in four cases. This test could be applied only when high blood pressure was maintained over a considerable period. In several instances a fall in blood pressure resulted with the administration of the drug. However, in contrast to the results occurring in cases of pheochromocytoma, the fall in blood pressure lasted only two to three minutes, whereas with the usual positive result the lowered blood pressure persists for 15 to 20 minutes.

Pneumoencephalograms made in six cases have shown no consistent pathological findings. Electroencephalograms have been recorded in 21 cases. In 16 of these the tracing was reported as abnormal. Often it suggested "convulsive disorder," but there was nothing specific in the findings.

FAMILIAL INCIDENCE

The occurrence of this syndrome in siblings appears to be too frequent to be attributed to chance. Table 3 summarizes the familial incidence. Among the 33 patients there are four pairs of siblings; in addition 7 other offspring appear in their histories to have been afflicted with a similar condition. Considering only the siblings of the index cases (that is, the cases that brought the family to the attention of a physician), there is a total of 33, of whom 11 are known to be or are thought from history to be affected. This proportion is compatible with a mendelian recessive characteristic, but no definite conclusions can be reached in this respect with such a small group. No history has been obtained of similar disorders among parents or collateral relatives. Also, there has been no evidence to date of consanguinity among the parents.

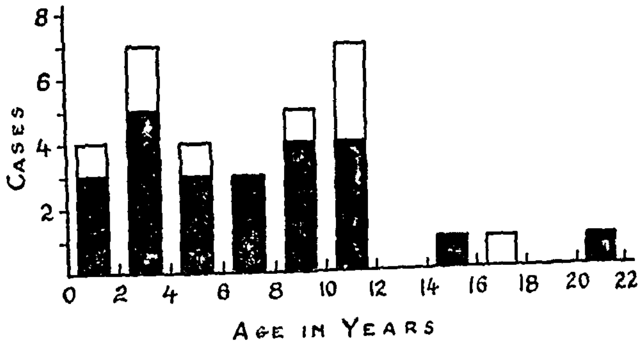
AGE DISTRIBUTION AND MORTALITY

Since the above evidence suggests that this syndrome is congenital in nature, there should be no preponderance of any one age range in a random group. This is borne out by the distribution of ages among the patients of our series as represented in the accompanying figure. The oldest patient is a girl of 20 years; one boy died at 17 years. In all the cases in which a detailed history was available, difficulty was encountered in early infancy. This usually consisted of difficulty in feeding, with the baby appearing to have trouble with swallowing. Abdominal distention was another common complaint. As a result of these features, the babies tended to be poorly nourished during the first year of life.

TABLE 3.—*Familial Distribution*

Total families	28
Total offspring	61
Known cases	32
Probable cases (by history)	7
Families with more than one child	22
Total siblings of index cases	33
Siblings affected or probably affected	11

Also shown in the accompanying figure are the ages at which death occurred in the 10 patients who have died. In eight of these the cause of death was attributed to aspiration pneumonia. In one case it appeared to be due to renal failure as mentioned above, and in the 10th case death occurred at home with a terminal episode of hematemesis.



Distribution of patients by current age (March, 1952) or age at death. The black portion of the bars represents the living and the white portion the dead.

POSTMORTEM FINDINGS

Autopsy has been performed in nine instances. Among these, pathological conditions in the central nervous system have been noted in only two. In one there were multiple abscesses in the brain, which were apparently secondary to aspiration pneumonia and presumably occurred terminally. In the second case, a report of which has been published,[1b] a small cyst was found in the thalamus. The absence of a similar lesion in the other cases suggests that this was an incidental finding.

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[3] Goldenberg, M.; Snyder, C. H., and Aranow, H., Jr.: New Test for Hypertension due to Circulating Adrenalin, J. A. M. A. 135:971 (Dec. 13) 1947.

TREATMENT

Treatment has been limited to efforts to modify the more disturbing manifestations, such as vomiting, hypertension, defective tearing with corneal ulceration, and the extreme emotional instability. Among the agents that have been tried are various types of anticonvulsants and sedatives, autonomic-active drugs, muscle relaxants, dimenhydrinate (dramamine®), and even cortisone; none of these has made any significant difference. The surgical procedure, previously reported,[4] of splitting the tentorium, though followed by temporary improvement, made no lasting difference. Lumbar sympathectomy in one case produced only temporary modification of blood pressure. Psychotherapy has proved unavailing, largely because it has been difficult to establish any real contact with the patient. In one patient whose vomiting was accompanied by an acute psychotic state and persisted for a dangerously long period, electric shock therapy has appeared on several occasions to interrupt the attack; when it was used prophylactically, however, it apparently had no effect. The most radical approach tried in a patient totally incapacitated by the frequency of her vomiting attacks has been prefrontal lobotomy. For almost a month after this procedure the patient did no vomiting but thereafter relapsed into her former state.

This summary of failures is given merely to show the variety of approaches that have been tried. Also, the fact that such heroic approaches seemed justified serves as an index of how disabling the symptoms can be.

COMMENT

The 33 cases summarized in the foregoing presentation have so many features in common that it justifies the concept that they are examples of a real and easily recognizable clinical entity of familial nature. This syndrome is manifested primarily by disturbances in function of the autonomic nervous system.

The syndrome can be recognized with considerable assurance in children over 2 years of age when all the features are evident. In younger children deficient lacrimation, absent reflexes, and skin blotching make the diagnosis fairly certain, especially if associated with a history of feeding difficulty. In the differential diagnosis in the first year of life acrodynia and cystic fibrosis of the pancreas may come up for consideration. In older children, pheochromocytoma or primary psychiatric disorder have had to be ruled out. The presence of all or most of the cardinal features discussed makes the differentiation relatively simple.

There is insufficient evidence to permit one to determine the pathogenesis of the syndrome. The fact that the neurogenic symptoms are so widespread strongly suggests a central rather than a peripheral origin. The presence of autonomic disorder, hyporeflexia, relative indifference to pain, psychic abnormality, and frequently mental retardation make it difficult to localize the disturbed function in any one area of the brain.

The question of the most suitable terminology for the condition has not been easy to settle. In the absence of knowledge of the causes or the pathologicoanatomic changes, a partially descriptive term is proposed that points out the most striking disability: "familial autonomic dysfunction," or, with the use of a Greek derivative to make the title shorter, "familial dysautonomia."

Finally, with the experience gained from this group it seems reasonable to speculate on the possible distribution of cases. All the patients described herein were brought to medical attention because of some incapacitating feature, but no one patient showed all the severe features. It is likely that there are patients with none of the extreme features who might present only behavior problems in childhood or personality problems in adult life. There is nothing in the basic condition incompatible with long life, so that it seems probable that as knowledge of the syndrome becomes more widespread more adult examples of it will be found.

SUMMARY

A composite picture of the syndrome "familial autonomic dysfunction," or, more briefly, "familial dysautonomia," based on 33 cases is presented (table 2). The nearly constant features of the condition are (1) Jewish extraction, (2) defective lacrimation, (3) skin blotching, (4) excessive perspiration, (5) drooling, (6) emotional instability, (7) motor incoordination, (8) hyporeflexia, and (9) relative indifference to pain. Other variable manifestations that have been disabling were present in all cases. It is an easily recognizable entity, not basically lethal, and presumably a congenital disorder of the central nervous system, in which the familial incidence suggests a genetic origin.

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[4] Bridges, T. J.; Pool, J. L., and Riley, C. M.: Central Autonomic Dysfunction with Defective Lacrimation: Preliminary Report of Effect of Neurosurgery in One Case, Pediatrics 3: 479, 1949.

Undetected Gastric Ulcer.—Roentgenologic examination is the chief method for the diagnosis of gastric ulcer. However, roentgenologists know well that a certain percentage of gastric ulcers cannot be found even with very careful examination including compression technic with spot films. The number of these apparently undiscoverable ulcers does not alone depend upon the skill and thoroughness of the observer. The number noticed depends greatly upon the number of checks done either by gastroscopy or by exploratory laparotomy. In places where gastroscopy is done only in rare cases, as an exceptional procedure, the patient in whom nothing was found at x-ray examination will be discharged after treatment and nobody may ever discover that in fact he had a gastric ulcer. If gastroscopy is done in every single instance in which a gastric disease is suspected, but in which roentgenologic examination failed to reveal any pathologic change, then quite a few gastric ulcers are found. Therefore it is difficult to compare statistics.

It must not be forgotten that many gastric ulcers cannot be seen at gastroscopy, and thus some ulcers will not be found even when both methods are employed. How frequent such diagnostic failures are we will never know, unless some new third diagnostic method is developed. It is clear that exploration cannot be undertaken routinely and therefore does not constitute such a third method.—R. Schindler, M.D., Large Gastric Ulcers, Not Demonstrable at Roentgenologic Examination, *The Medical Clinics of North America*, July, 1952.

PROBLEM OF EMOTIONAL TRAUMA IN HOSPITAL TREATMENT OF CHILDREN

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and
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The importance of the emotional needs of the child in the hospital, and especially of the child who is to be operated on, has been receiving more attention in recent years by nurses, pediatricians, surgeons, and psychiatrists. The fact that emotional trauma occurs is widely accepted. The definition of the term, however, and the causes and means of prevention of the condition are sometimes inadequately understood. For the purpose of this discussion, emotional trauma will be defined as the intensification of any feeling that may be deleterious to a satisfactory adjustment to life. Feelings that are likely to be involved in the hospital treatment of children are anxiety, suspiciousness, resentment, hostility, inadequacy, insecurity, and a desire to retaliate.

From our study of the psychological aspects of hospitalization, anesthesia, and surgery in a group of children, certain conclusions seem justified concerning the nature, etiology, and prevention of emotional trauma associated with such experiences. The children, whose ages ranged from 3 to 8 years, were admitted to the hospital for tonsillectomy. Before admission, a psychiatric social worker interviewed the mother and the child in the home, obtained a record of the child's behavior, and evaluated his emotional characteristics and his relationship to his parents. A history of dreams and mannerisms was included in an attempt to elicit the presence of repressed feelings. This interview was repeated several times after the operation. Final checks after 18 months are now being made.[1] In most of the children, anesthesia was induced with open-drop vinyl ether (vinethene®) administered by an anesthetist with special interest and experience in dealing with emotional problems.[2]

In this study, a child who was able and willing to cooperate in the hospital procedures, who accepted the usual treatments with a minimum of resistance, and who went through the induction of anesthesia without evidence of serious fear was considered to have met the experience adequately. If during the postoperative interviews no behavior changes were found that indicated the intensification (overt or repressed) of any of the abovementioned negative feelings, it was concluded that the child had suffered little or no emotional trauma.

In this age group, traces of some or all of the negative feelings under consideration are already present. They exist in varying degrees in every child. Thus, it is impossible, at this age, to initiate a new negative feeling by any single experience. Even as threatening an incident as hospitalization and surgery is merely another feeling-experience. It is the combined responsibility of parents, physicians, and hospital personnel to make it a constructive experience or at least to keep its emotional damage to a minimum. In the prevention of emotional trauma in the hospital-anesthesia-surgery experience, there are three important factors: 1. The child must have made a reasonably adequate adjustment to his environment. 2. He must have proper preparation for the specific experience. 3. The experience must be modified to meet the child's endurance. The first of these factors, the adjustment of the child to his environment, depends almost entirely on the relationship between the child and his parents. If he has been wisely loved and adequately supported, he will be trusting and self-assured. If his parents are suspicious or retaliatory, he will be likely to follow this pattern. Just as his physical traits are determined by those of his parents and grandparents, his emotional make-up will largely parallel that of his forebears. The difference is that, while purely physical characteristics are modified only by generations of evolution, any parent can learn to understand and improve his feeling-pattern and pass on to his child a different set of emotional reactions from those he learned from his parents. The importance of this principle lies in the fact that the emotional equipment required for dealing adequately with a hospital experience is no different from that needed in dealing with the other problems of life.

Our experience has shown that the child best able to meet the hospital-anesthesia-surgery situation is the child who is able to trust the physicians and nurses to treat him fairly. He assumes that painful procedures are necessary, are for his own good, and are not punitive. He is willing, therefore, to place himself in the hands of others and expects from them the kindness, understanding, and support to which he is accustomed at home. He is not overwhelmed by his own fear, because he is able to trust someone else who is unafraid. Obviously, such a child must have a firm foundation of trust in his parents, a deep assurance that they are always fair and always consistent in their love. He has already learned that a certain amount of unpleasantness and deprivation are unavoidable in life and that they are not in any way punishment for misbehavior. In the hospital, he expects some unpleasantness and pain and accepts the situation without resentment.

With such a background of preparation for all difficult experiences, preparation for hospitalization is simple. The parents should explain honestly and simply what is going to happen and why. The child's questions should be answered patiently and reassuringly. It is important that the parents avoid communicating to the child any apprehension that they may have. On the other hand, the

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[1] Winkley, R., and others: Emotional Reactions and Behavior of Children in the Home: Method of Study, J. Pediat. **38**: 476-481 (April) 1951.

[2] Jackson, K.: Psychologic Preparation as a Method of Reducing the Emotional Trauma of Anesthesia in Children, Anesthesiology **12**: 293-300 (May) 1951.

child's fear should be accepted as normal and be neither denied nor ridiculed. In other words, the child should be allowed to be frightened but should be able to look to the adults and find them adequate and unafraid.

The time of admission to the hospital for elective procedures should be determined with regard to the child's emotional state. In case of a death in the family, especially that of a parent or a sibling, or serious marital difficulty between parents, operation should be postponed until the child has made his best adjustment to the situation. Time should also be allowed for his becoming accustomed to school, a new home, a new baby, or a new stepparent before he is sent to the hospital.

Shortly before admission to the hospital, either the family physician or the surgeon should tell the child what is going to be done. If, along with tonsillectomy, any other procedure, even the removal of a tooth or a wart, is anticipated, this should be explained. One of the children in our group had been told by a child who had had a hernia repaired at the time of his tonsillectomy that his tonsils would be taken out through his abdomen. The child's misunderstanding was corrected before surgery, but he had several days of anxiety. Whenever there is a period of anesthesia, a child may become apprehensive about what has been done to him while he was unconscious. Only his trust in his parents and the physician protects him from fantasies of mutilation.

For even the best-prepared child, and especially for the anxious one, considerate treatment in the hospital is necessary if emotional trauma is to be avoided. It might seem that busy nurses and physicians could not possibly meet each child's emotional needs, and it is true that they cannot perfectly replace the parent, but children who have been loved wisely at home develop a self-assurance that makes them less demanding. They need only considerate understanding and they will cooperate. Specific examples of desirable means of administering treatment could be given, but the important factor is the feeling of the adult toward the child, for the person who really has the child's welfare at heart will intuitively choose the least traumatic methods. Lack of experience sometimes leads to minor errors. A student nurse was inadvertently responsible for several days of anxiety in one of our children, because she told him that the catheter through which rectal anesthesia was administered was only "a puppy dog's tail." The child could not remember the catheter being removed. Naturally, not all persons have the emotional maturity, the warmth, or the wisdom to be supportive and understanding; if, however, the child is making a satisfactory adjustment to life in general, he will probably not be seriously disturbed by unenlightened treatment for a few days in the hospital.

The exception to this occurs in separating the very young child from his mother at the time of surgery. When there has been no previous separation, this aspect of the experience is probably the greatest source of trauma. For most children under 4 years of age, it is our observation that no amount of love and understanding will effectively substitute for the mother. When physicians realize how inextricably a child's emotional welfare is bound up with physical welfare, provisions will be made for a parent to remain with the hospitalized child. If only in the interest of physical well-being, a consideration of the child's emotional needs must eventually take precedence over rules, schedules, and the polish on the floor. Certain qualifications must be made to the general statement that a parent should stay with the child hospitalized for tonsillectomy. Since the purpose of the parent's presence is to support the child in a difficult situation, any parent who does not help the child is of no use. In fact, the overanxious parent may add to his child's burden, and, certainly, the parent who overidentifies to the point of hysteria (not a rare occurrence in our study) does the child more harm than good.

In the interest of reducing emotional trauma during the hospital stay, treatments such as enemas and venipunctures should be kept to a minimum. Having anything inserted into the anus, especially by a stranger, is extremely objectionable to most children and should be avoided whenever possible. If the child has learned to hold a thermometer in his mouth, he should be permitted to do so at the hospital. Waking the child at midnight on the preoperative day to take his temperature because the chart calls for a reading seems unkind and unnecessary. A preoperative reading after he has awakened is more informative and less traumatic. Giving unavoidable enemas and taking temperatures are things that can be done by the mother if she is allowed to stay with the child.

Among the children in our group, needles caused the greatest resentment. It was our practice to reduce the use of needles to a minimum, so that we usually were able to protect the child from all but the finger prick for the hemoglobin reading and the atropine injection. The latter was given in the arm rather than in the buttocks, because, early in the study, we found that less resentment was aroused when the child could see what was done. Venipuncture for the Wassermann test, which is required by hospital regulation, was done during anesthesia.

Although this was the usual procedure in our study, an opportunity was afforded to observe reactions to venipunctures in the same children. Another department began research that involved securing several specimens of venous blood, and, before an understanding and agreement could be reached, several children in our group had been used as subjects. The reactions to this experience were more intense than to any other procedure. Most of the children remembered and resented it at the final 18 month check-up. Obviously, it is impossible to avoid venipuncture entirely, but it seems wise to restrict its use to occasions when it is really necessary. It should then be done by persons who have had experience in doing venipunctures. Such experience can be acquired by doing the necessary venipunctures on anesthetized children or on children with "medical" conditions, who are not being subjected to the additional stress of anesthesia and surgery.

The use of a bed with sides is sometimes taken as an insult by children who have outgrown them at home. It is accepted, however, if the sides are explained as a necessary protection against a painful or dangerous fall. The young patient should not be required to stay in bed, especially alone in a room, when he is feeling well. On the day before surgery, he will be happier and more cooperative if he is allowed to go about the ward and play with other children.

The atraumatic administration of anesthesia poses a special problem. Unless precautions are taken, the child is likely to mistake the onset of unconsciousness for impending death and to struggle accordingly. Verbal reassurance alone is ineffective and must be augmented by specific information about the experience and its accompanying sensations. With the method described in an earlier paper,[2] almost all of our children went to sleep without resistance or evidence of serious fear. Exceptions were a few children who gave evidence of previous emotional disturbance; most of these children were under 4 years of age. A few 3-year-old children went to sleep without protest, but they had the advantage of good support and reassurance by a parent or parent substitute during induction of the anesthesia.

The inability of children under 4 years of age to understand and anticipate a threatening experience might suggest the advisability of using some form of basal anesthesia. A few of our children were given thiopental sodium rectally, too few to warrant conclusions concerning its efficacy in preventing emotional trauma. It was noted, however, that the children who went to the operating room asleep showed some postoperative anxiety and doubt about whether their tonsils had really been removed. At the time of emergence from anesthesia, the child again has a great need for the comforting presence of the parents. In fact, even if parents are barred at all other times from the pediatric ward, they should be allowed to be present at the bedside of the awakening child. Even under the best conditions, the child has been through a frightening and threatening experience and needs the immediate assurance that only a mother or father can provide.

Most children come to the hospital with less than perfect intellectual and emotional preparation. Deep-seated anxieties and feelings of insecurity and inadequacy make the prospective experience a real threat to their emotional adjustment to life. To these children, physicians, nurses, and other hospital personnel can render valuable aid. They can supply the love and the support that the child must have if he is to withstand the hospital experience without further emotional damage. The child who is anxious and suspicious is the child who will cry and resist the most, and he is the one who most needs kind treatment. It requires emotional maturity and understanding to be "good" to a belligerent, assaultive child, but the willingness and the ability not to retaliate at such a time will pay dividends in reducing the child's hostility and resistance and in the satisfaction of having made a constructive contribution to his emotional well-being.

DATA

During the two year period of our study, 105 children were observed through the first three postoperative months. At the first interview after the operation, many were found to have been babyish and clinging, to have slept restlessly, or to have adopted substitute or new self-comforting mannerisms. At the end of the three month period, this less acceptable behavior had persisted in 17 children. Some degree of emotional handicap existed in almost all the patients, but whereas 88 of these children survived the experience without evidence of damage, an examination of the emotional background of the 17 who did not is indicated. Correlations are being made in all cases between the emotional factors considered above and earlier potentially traumatic experiences and postoperative behavior changes.

SUMMARY

During two years work on a special study of the psychological aspects of hospitalization, anesthesia, and surgery for children, it was observed that a child is least likely to suffer from emotional trauma if he is well adjusted to a family environment of love, trust, and security. Certain factual preparation is necessary before hospitalization, in order to prepare him for the experience. The time of hospitalization should be chosen according to his emotional balance.

All phases of the experience should be gaged to the child's ability to adjust, and all procedures to which he is subjected should be carried out in a considerate and friendly manner. Painful treatments should be kept to the necessary minimum. In all children, but especially in those who are inadequately equipped for hospital experience, physicians and hospital personnel can, by kindness, understanding, and support, greatly reduce traumatic effects. Of 105 children studied, 17 showed behavior changes at the end of the third postoperative month.

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SULFHEMOGLOBINEMIA FOLLOWING HABITUAL USE OF ACETANILID

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and

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There are numerous reports in the older medical literature of cyanosis following the prolonged ingestion of acetanilid;[1] the cyanosis was assumed to be due to the presence of methemoglobin. In 1930, Harrop and Waterfield called attention to the fact that acetanilid poisoning could cause sulfhemoglobinemia.[2] There are a few other reports in the literature of single instances in which this fact was demonstrated.[3] Nevertheless, cyanosis with acetanilid poisoning is still frequently ascribed to methemoglobinemia.

Differentiation between the two pigments is relatively easy by methods now available and is of considerable practical importance, particularly with regard to therapy. Methemoglobin is rapidly reversed to normal

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hemoglobin by the enzyme systems in the red blood cell as soon as the offending poison is removed. Treatment of methemoglobinemia of a mild degree is, therefore, often unnecessary. In severe cases, methylene blue (1 to 2 mg. per kilogram), given orally or intravenously, is dramatically effective and may be life-saving. Sulfhemoglobin, on the other hand, is not reversible to hemoglobin, and, once formed, it persists for the life of the red cell. No known treatment is beneficial. Diagnosis is important chiefly because of the implications it carries regarding the patient's drug intake.

Cyanosis that persists after the causative agent is withdrawn is probably due to sulfhemoglobin rather than methemoglobin. Since many patients with this disorder are drug addicts who often obtain supplies by surreptitious means, it is difficult to be certain when drug ingestion has been stopped; therefore, spectroscopic examination of the blood is essential for a definite differentiation. The absorption bands of methemoglobin (630 mμ) and sulfhemoglobin (618 mμ) can be confused when a hand spectroscope is used, but they are easily differentiated after the addition of a small amount of neutral cyanide solution; this dissipates the methemoglobin band, but the sulfhemoglobin band is unaffected. The following case histories of six patients seen during a relatively brief period in the wards of a general hospital indicate that sulfhemoglobinemia and not methemoglobinemia is the usual result of acetanilid overdosage and that acetanilid habituation is still a problem in clinical medicine.

REPORT OF CASES

Case 1.—A 52-year-old white woman was admitted to the Los Angeles County Hospital on July 13, 1950, with the complaint of severe, crushing precordial pain of six hours' duration. The heart was moderately enlarged; the blood pressure was 110/70; and marked cyanosis was present, which did not clear with oxygen inhalation. An electrocardiogram indicated a recent anterior myocardial infarction. Because of the persistent cyanosis, methemoglobinemia was suspected. In the blood sent to the laboratory, an abnormal band in the red spectrum was found, which was thought to be caused by methemoglobin. Eighteen per cent of the total hemoglobin of 14 gm. per 100 cc. (Sahli acid-hematin method) was so affected. This estimate of methemoglobin concentration was made by the method of Chase and associates,[4] which makes use of absorption ratios at appropriate wave lengths. It does not differentiate between methemoglobin and sulfhemoglobin. After repeated questions, the patient admitted taking "some" bromo-seltzer® (2.5 grain [0.16 gm.] of acetanilid and 5 grains [0.32 gm.] of sodium bromide per teaspoonful). Her son stated that she had been taking bromo-seltzer® in large quantities for several years to relieve nervousness, tension, and headaches. She ultimately admitted taking from five to six teaspoonfuls daily, which the son felt was less than she actually used. She denied taking any other medication. The blood bromide level four days after entry was 12.6 mEq. per liter. It was not appreciated that the spectrophotometric method failed to make a distinction between methemoglobin and sulfhemoglobin; accordingly, 7.5 cc. of 1% methylene blue was administered intravenously on July 29. There was no change in the cyanosis, and the methemoglobin level on Aug. 1 was still 16% of the total hemoglobin. Ascorbic acid, 500 mg. daily by mouth for 12 days, failed to lessen the cyanosis. Convalescence from the myocardial infarction was uneventful. The blood bromide level fell to 2.3 mEq. per liter on Aug. 14. At the time of discharge, on Aug. 21, the blood still contained 11% abnormal hemoglobin. The patient specifically stated on admission that she had not been constipated, and this was not a problem during the hospital stay.

COMMENT

In retrospect, the abnormal hemoglobin responsible for the cyanosis was unquestionably sulfhemoglobin. There was no response to reducing agents, and the cyanosis was of long enough duration to exclude the possibility of methemoglobinemia. (The falling blood bromide level proved that bromo-seltzer® ingestion was not continued.) This case illustrates an error in diagnosis leading to unnecessary and ineffective therapy because of the failure to employ the simple cyanide differentiation test. Because of the heart disease, transfusion may have been indicated in an effort to increase the oxygen carrying capacity of the blood. Such treatment rarely would be necessary, since, in none of our patients was more than 20% of the hemoglobin changed to sulfhemoglobin.

Case 2.—A 51-year-old white man was admitted to the Los Angeles County Hospital on Aug. 28, 1950, with diffuse, poorly-described abdominal pain and generalized weakness of several weeks' duration. He was somewhat belligerent and talkative, and his memory seemed poor. There was a pustular acneform eruption over the chest and back. The mucous membranes and lips were deeply cyanotic, and the skin had a pasty, grey appearance. His hospital record extended back to 1943, with numerous clinic visits for multiple, poorly-described complaints, which included headache, weakness, and abdominal and chest pain. On several occasions, cyanosis of the lips had been noted. On one hospital entry in 1948, there was a history of bromo-seltzer® ingestion, and the blood bromide level was 24.2 mEq. per liter. At first the patient denied taking bromo-seltzer,® but gradually, on repeated questioning, he admitted his estimated intake to be 12 to 16 oz. (340 to 454 gm.) weekly. His wife told us that he took considerably larger amounts at times and that physicians repeatedly advised him to discontinue the medication; however, he seemed unable to do so. He stated that he had taken bromo-seltzer® for "at least 10 years," because it gave him relief from headaches, nervousness, and abdominal pain.

On Aug. 29, the blood bromide level was 19.0 mEq. per liter, and, by spectrophotometric examination, 16% of the hemoglobin was found to be present in an abnormal form, presumed to be methemoglobin. The total hemoglobin level was 13.5 gm. per 100 cc. (Sahli acid-hematin method), and the arterial oxygen saturation was 82% (Van Slyke method). A total of 100 mg. of methylene blue was administered intravenously on Aug. 30 and again on Aug. 31, with no reduction of cyanosis. The amount of abnormal hemoglobin found on spectrophotometric examination was unchanged on Sept. 1. Ascorbic acid, 1,000 mg. daily for five days, had no effect on the cyanosis or the level of abnormal hemoglobin. The abnormal pigment was then tested for its stability to cyanide, using the quantitative

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method of Evelyn and Malloy.[5] The absorption peak was not affected by the addition of cyanide solution, indicating that the pigment was sulfhemoglobin. There was no detectable methemoglobin in a fresh sample of blood.

The patient was warned of the possible consequences of continued bromo-seltzer® ingestion. During his hospital stay of 12 days, he manifested marked nervousness and irritability, which were only partially controlled by barbiturates. He seemed anxious to leave the hospital, presumably to obtain more bromo-seltzer,® although he stated he would never take it again. He has since been seen several times at the clinic, with cyanosis, sulfhemoglobinemia, and high blood bromide levels, but he is unwilling or unable to discontinue taking bromo-seltzer.® Constipation was rarely present; the patient stated that he took cathartics on the average of once a month.

Comment.—As in case 1, the fact that the abnormal hemoglobin was sulfhemoglobin was not appreciated until the cyanide differentiation test was performed. The diagnosis was not even suspected for several years, although the patient was seen by a number of physicians and given extensive symptomatic therapy for numerous vague complaints that were probably the result of rather than the cause of his drug habit. This patient did not have a significant degree of constipation nor was it an outstanding finding in the majority of the patients in this series. Constipation has been assumed to be an important factor in sulfhemoglobinemia, leading to abnormal hydrogen sulfide absorption and thus providing a source of sulfur. Sulfhemoglobinemia has often been ascribed to this substance alone in so-called enterogenous cyanosis, even though attempts to produce sulfhemoglobin in vivo with hydrogen sulfide have been disappointing.[6] Judging from the histories of our patients, constipation is not an essential factor in sulfhemoglobinemia. The diagnosis of enterogenous cyanosis, if such an entity exists, requires the most careful exclusion of acetanilid (or other drug) ingestion.

CASE 3.—A 42-year-old white woman was brought to the Los Angeles County Hospital on July 24, 1951, by the police department; she was unable to give a history. Information was obtained from a friend to the effect that she had been acting peculiarly, had difficulty with her speech, and had seemed "groggy," for approximately two months. For about the same period of time, a purplish color of the lips had been noticeable. He stated that she had been taking large quantities of bromo-seltzer® for a period of several months. The landlady had called the police because of the patient's peculiar behavior.

At time of admission to the hospital, the patient was semistuporous, confused, and unkempt. The mucous membranes were deeply cyanotic, and the skin was a slate-grey color. The blood bromide level was 23.8 mEq. per liter. On July 26, spectrophotometric examination of the blood indicated the presence of 0.72 gm. per 100 cc. of sulfhemoglobin, no methemoglobin was detectable. The total hemoglobin was 14.4 gm. per 100 cc. (Sahli acid-hematin method). The blood bromide level gradually fell to 10.3 mEq. per liter on Aug. 8. By Aug. 2, the patient's mental state had markedly improved. She admitted taking one bottle "or more" of bromo-seltzer® daily for approximately two weeks before admission "to settle her nerves." She denied long-term ingestion of bromo-seltzer,® although the history obtained from the friend contradicted her statement. No reliable information regarding constipation was obtained. Cathartics were not prescribed during the 12 day hospital stay. At the time of discharge, on Aug. 6, the cyanosis and slate-grey skin color were still evident. She did not return for a follow-up examination.

Comment.—This patient's symptoms were undoubtedly caused by the high blood level of bromide and not by the relatively small amount of sulfhemoglobin present, which actually would interfere only mildly with oxygen transport. The diagnosis of bromide intoxication might not have been made without the history provided by the friend. In a patient with these symptoms, the finding of cyanosis due to sulfhemoglobinemia should suggest the possibility of bromide toxicity from bromo-seltzer.®

CASE 4.—A 43-year-old white woman was brought to the Los Angeles County Hospital on April 10, 1950. She was semistuporous and irrational. The only history obtainable was given by a friend, who indicated that she had been "sick" for four days, had been taking bromo-seltzer® (amount not determined) for years, and had been drinking recently. Her old hospital record revealed that at the time of a laparotomy for suspected ectopic pregnancy, in 1935, one of the surgeons had noted that "the blood was extremely dark and bluish in color, suggesting methemoglobinemia." She was seen in the hospital admitting room in 1943, complaining of abdominal pain and vomiting, and, at this time, she gave a history of taking bromo-seltzer® almost daily since the age of 19.

At the time of admission, she was febrile, deeply cyanotic, and tachypneic, and there were rales at the right lung base. The cyanosis did not improve when oxygen was administered. The blood bromide level was 26.4 mEq. per liter. When the blood was examined with the spectrophotometer, 20% of the hemoglobin was found to be in an abnormal form, presumably methemoglobin. (A cyanide test was not done.) A total of 50 cc. of 1% methylene blue was given intravenously after the above determination. Cyanosis was reported to have "slightly decreased" but was still pronounced the next day and until the time of death. Her condition grew worse in spite of penicillin therapy, and she died on April 13. No further spectrophotometric examinations of the blood were made. Bronchopneumonia in the right lung was found at autopsy.

Comment.—Although definite proof of sulfhemoglobinemia is lacking in this patient, the failure of the cyanosis to disappear when a large dose of methylene blue was administered and the long-standing habituation to bromo-seltzer® strongly suggest its presence. The possibility that pneumonia could have been responsible for the continued cyanosis after methylene blue was administered seems remote, since the pneumonia was not extensive, and oxygen was used continuously without benefit. There was no reliable information available regarding constipation.

CASE 5.—A 70-year-old white man was admitted to the Los Angeles County Hospital on Jan. 30, 1949, complaining of exertional dyspnea and precordial pain of six months' duration. He also had frequent headaches and had noticed a constant blueness of his lips for the same period of time. He had been constipated "all his life." The blood pressure was 210/100 mm. Hg, and the heart was slightly enlarged. The mucous membranes and nail beds were deeply cyanotic. There were no evidences of congestive failure. The electrocardiogram indicated left ventricular hypertrophy.

Cyanosis persisted in spite of oxygen inhalation and complete relief of dyspnea. Blood withdrawn for chemical analysis had a brownish tinge. A history was then obtained of taking five teaspoonfuls of bromo-seltzer® daily for approximately 18 months. The blood was examined spectrophotometrically on Feb. 4; 30% of the hemoglobin was found to be present as an abnormal form, presumed to be methemoglobin. A cyanide test was not done. At the time of discharge, on Feb. 6, the blood bromide level had fallen to 7.6 mEq. per liter, and the cyanosis had decreased only slightly. Cathartics were prescribed four times during the hospital stay of 11 days.

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5. Evelyn, K. A., and Malloy, H. T.: Microdetermination of Oxyhemoglobin, Methemoglobin, and Sulfhemoglobin in a Single Sample of Blood, J. Biol. Chem. **126**: 655, 1938.

6. Meyer, E.: Ueber das Verhalten des Nitrobenzols und einiger anderer aromatischer Nitrökörper im Organismus, Hoppe Seyler's Ztschr. f. physiol. Chem. **46**: 497, 1905. Harrop and Waterfield.[2]

Comment.—Again, proof of sulfhemoglobinemia is lacking; however, the fact that the abnormal hemoglobin was present five days after admission is strong evidence that it was sulfhemoglobin. Methemoglobin would have been reduced to hemoglobin in this period of time, unless there had been continued ingestion of bromo-seltzer,® which was fairly well ruled out by the falling blood bromide level. The failure of the cyanosis to improve with oxygen inhalation should lead to examination of the blood for the brownish blue color of methemoglobin or sulfhemoglobin, which does not redden with oxygenation.

Case 6.—A 77-year-old white woman was first seen at the Los Angeles County Hospital in June, 1947, with complaints of insomnia and headache and with poorly described abdominal pain. The liver and spleen were both palpable. The hemoglobin was 8.0 gm. per 100 cc. (Sahli acid-hematin method), and the red blood cell count was 2,400,000 per cubic millimeter. No cause was found for the hepatosplenomegaly and anemia. The patient was seen again in January, 1948, with similar vague complaints and the same findings. Moderate cyanosis of the skin and lips was noted but was not explained.

On the patient's third entry, in 1948, hepatosplenomegaly and anemia were still present. The hemoglobin was 10.0 gm. per 100 cc., the red blood cell count 2,650,000 per cubic millimeter, and the mean corpuscular hemoglobin 39 micromicrograms. There was diminished vibration sense in the lower extremities and an absence of free gastric acid after histamine injection. Examination of the bone marrow revealed hyperactive erythropoiesis without megaloblastosis. Pernicious anemia was suspected, but there was no appreciable reticulocyte response to liver extract.

On the fourth entry, in December, 1948, the findings were unchanged. Cyanosis was again noted, and spectrophotometric examination of the blood indicated that 25% of the hemoglobin was in an abnormal form, presumed to be methemoglobin. (The cyanide test was not done.) Drug ingestion was denied. She stated that her skin had turned blue at times over a period of many years. Congenital methemoglobinemia was considered, but ascorbic acid, 500 mg. daily by mouth for seven days, failed to change the level of abnormal hemoglobin.

In November, 1950, the patient was again admitted to the hospital with considerable cyanosis. When the blood was examined spectrophotometrically, 15% of the hemoglobin was found to be in an abnormal form. The addition of cyanide did not affect the pigment, indicating that it was sulfhemoglobin. The level of sulfhemoglobin did not change over a 15 day period of hospitalization. Conversation with relatives disclosed that the patient habitually took "Dr. Miles anti-pain pills" (2.0 grains [0.13 gm.] of acetanilid, tincture of capsicum, and caffeine [unspecified amount] in each tablet), often as many as 30 a day. When confronted with this evidence, the patient admitted (reluctantly at first) ingestion of this medication over a period of 50 years, stating that it "settled her nerves and relieved abdominal pain." She would not admit taking the pills in the amounts described by her relatives, claiming that four or five a day was her maximum intake. There was no evidence of withdrawal symptoms during any of the time she spent in the hospital. Tests of hepatic function, including serum albumin and globulin, sulfobromophthalein (bromsulphthalein®) retention, prothrombin time, thymol turbidity, and cephalin-cholesterol flocculation, were within normal limits. The patient complained of mild, chronic constipation, necessitating the use of a cathartic once or twice a week over a period of several years.

COMMENT

In most of the cases, the history of drug habituation was not obtained from the patient, illustrating the importance of using accessory sources of information such as the friends or relatives. Whether chronic acetanilid use represents habituation or true addiction has not been settled.[1a] No definite withdrawal symptoms were noted in any of the patients in this series, suggesting that psychic dependence was present rather than true addiction. Acetanilid appears to be the substance responsible for the many cases reported in the older literature of habitual use of various headache remedies, including bromo-seltzer.® The fact that bromo-seltzer® is easily obtainable is probably the reason for the high percentage of habitual users of this compound found in our series. Acetanilid is undoubtedly also the substance responsible for sulfhemoglobin production in our patients. We have never seen sulfhemoglobinemia in sodium bromide or triple bromide intoxication.

Judging from the numerous reports in the older literature,[7] anemia frequently occurs in patients with chronic acetanilid poisoning. The pathogenesis of anemia is unknown. It is often said to be macrocytic, on the basis of the color index or mean corpuscular hemoglobin. Gross suggests that this may be caused by falsely high hemoglobin estimations in the colorimetric methods because of interference by the color of sulfhemoglobin.[8] Anemia, supposedly macrocytic, was present in the patient in case 6; it was not seen in any of the other patients in the series. Hepatosplenomegaly is occasionally mentioned in reports of chronic acetanilid poisoning.[9] The pathogenesis is obscure, and the findings in the few patients who came to necropsy were nonspecific. Enlargement of the liver and spleen was noted in only one patient in this group. The degree of enlargement was not marked; it did not progress over a period of years; and there was no laboratory evidence of impaired liver function.

SUMMARY

The case histories of six patients seen over a two year period are discussed, illustrating the following points. Acetanilid habituation still occurs. It will seldom be discovered unless sources of information other than the patient's history are employed. Habitual usage appears to result from psychic dependence rather than true addiction. Bromo-seltzer® is the medication usually involved. The bromide may produce toxic effects, but it does not appear to be responsible for the habituation or the sulfhemoglobin formation.

Sulfhemoglobinemia rather than methemoglobinemia is a common result of chronic acetanilid use and probably accounts for the majority of cases of cyanosis reported following the ingestion of acetanilid containing compounds. Some unknown endogenous source of sulfur must be assumed to be present. It may be hydrogen sulfide, although constipation does not appear to be necessary for sulfhemoglobin production. Anemia and hepatosplenomegaly, both of unknown cause, may occur with chronic acetanilid poisoning.

Since this article was prepared for publication, a large number of cases of sulfhemoglobinemia, collected over an 11 year period, have been described by Brandenburg and Smith.[10] Most of their patients took bromo-seltzer.®

1200 North St.

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[7] Gross.[1a] Payne.[1b] Lundsteen.[1c] Stewart.[1d] Herrick and Irons.[1e] Barker.[2c]

[8] Gross,[1a] p. 32.

[9] Strickland, C. Y.: A Case of Chronic Acetanilid Poisoning, Penn. M. J. **11**:31, 1907. Gross.[1a] Stengel.[1c] Herrick and Irons.[1e] Stengel and White.[1h]

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CLINICAL NOTES

ADRENAL ATROPHY AND IRREVERSIBLE SHOCK ASSOCIATED WITH CORTISONE THERAPY

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Recent clinical observations and animal experiments[1] have shown that prolonged administration of cortisone produces adrenal atrophy. According to the adaptation syndrome of Selye,[2] adrenal atrophy in turn may lessen the body's ability to withstand trauma and stress. One might suspect that patients receiving cortisone would not normally withstand major surgery; yet every day patients are successfully undergoing surgery while regularly taking cortisone in both the preoperative and postoperative periods.

We wish to present the record of a patient who, while receiving a prolonged course of cortisone therapy, underwent a major operation, cup arthroplasty of the hip, and died of immediate postoperative shock, in spite of normally adequate, aggressive antishock therapy. Postmortem examination revealed marked bilateral adrenal atrophy and other conditions consistent with shock.

REPORT OF A CASE

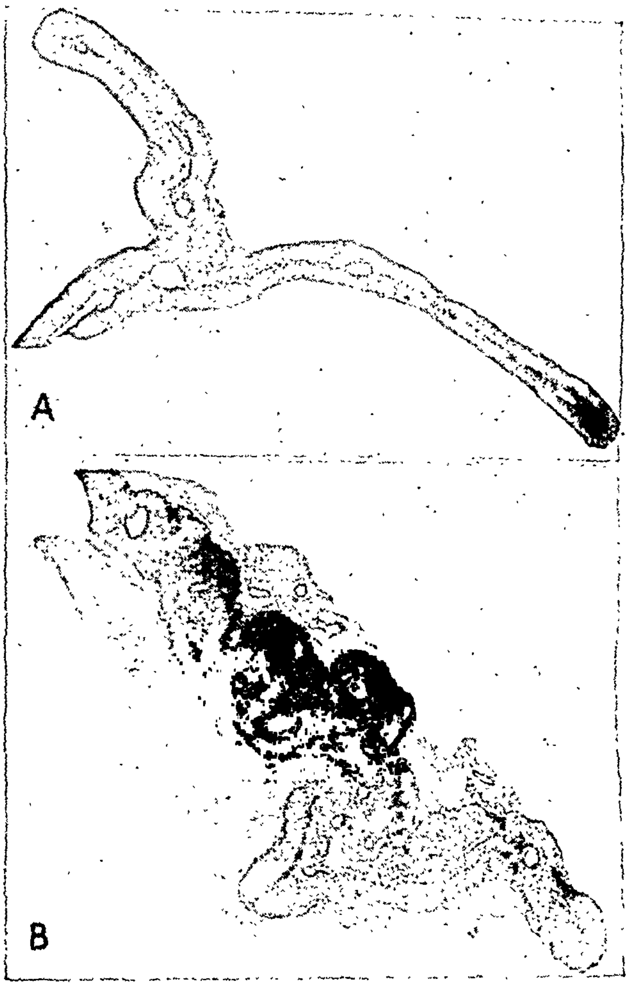
A 34-year-old man had had rheumatoid arthritis for a period of seven years. Almost every joint of his body was involved, particularly the hips, where there was a marked flexion contracture. The patient had been receiving cortisone for eight months. He first received 1,200 mg. of cortisone over a period of 10 days and then 100 mg. three times weekly. For two months prior to the present examination the patient had been taking 25 mg. of cortisone twice a day. Because of the pain in the right hip and flexion deformity, a cup arthroplasty in the right hip was advised.

Past History.—The patient had the usual childhood diseases, including smallpox, and had hay fever. Several episodes of mild skin petechiae were diagnosed as allergic purpura; the latest occurred after his use of cortisone. Bleeding, clotting, and prothrombin times were normal. The platelet count was normal.

Physical Findings.—Physical examination revealed a chronically ill-appearing, deformed man of 34 years, who was able to walk about in a stooped position with the use of crutches. He did not appear to be in acute pain. There was pronounced evidence of generalized rheumatoid arthritis. The heart and lungs were normal.

Laboratory Data.—X-ray studies revealed an essentially normal chest. The heart measured nearly 50% of the transverse diameter of the chest and was not enlarged in any one region. The red blood cell count was 4,220,000 and hemoglobin 13 gm. per 100 cc. Urinalysis disclosed no abnormalities. Serologic studies revealed nothing essentially abnormal. The white cell count was 11,000, with 72% neutrophils, 19% lymphocytes, 8% monocytes, 1 basophil, and no eosinophils.

Course in the Hospital.—The patient received cortisone up to the time of admission and two days later was taken to the operating room. Spinal anesthesia was employed, with tetracaine (pontocaine®) hydrochloride supplemented with thiopental sodium. The blood pressure at the beginning of the operation was 140/80 and the pulse rate 72. A routine cup arthroplasty of the right hip was performed. During the course of surgery the patient received one blood transfusion. His blood pressure did not fall below 110/80 during the whole procedure. After the first unit of blood was given, the second was begun. After the patient received 50 cc. of the second transfusion some muscular twitchings were noted, so that transfusion was discontinued to guard against a transfusion reaction. During the course of the surgery the patient never showed any real need for further supportive therapy. His blood pressure never went below 110/80 and his pulse rate never higher than 80. The operation extended over a period of an hour and one-half. Almost immediately after being transferred from the operating table to the bed the patient began to show some fall in blood pressure. The first postoperative blood pressure reading was 86/60, so that transfusion of another unit of blood was begun. In spite of rapid administration of 500 cc. of blood, the blood pressure continued to decline. A total of 250 cc. of plasma was given without appreciable change. It was felt at this time that there was something peculiar about this patient's reaction, because his estimated blood loss at surgery was not over 500 cc. and he had been given more than adequate replacement, but the blood pressure continued to decline. Five cubic centimeters of adrenal cortical extract was given on two occasions without benefit, and 500 cc. of plasma was given rapidly. In spite of this therapy, the blood pressure declined to about 40 systolic and the pulse became almost imperceptible. Five minims (0.3 cc.) of epinephrine was given intravenously without effect. Epinephrine was then given intracardially without effect.



A, cross section of the entire adrenal gland, showing marked adrenal atrophy (× 10). *B*, cross section of a normal adrenal gland (× 10).

From the Veterans Administration Hospital.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

[1] Antopol, W.: Experimental Observations with Massive Doses of Cortisone, Am. J. Path. **27**: 705, 1951. Sokoloff, L.; Sharpe, J. T., and Kaufman, E. H.: The Effect of ACTH and Cortisone on the Human Adrenal Gland, ibid. **27**: 706, 1951.

[2] Selye, H.: The Physiology and Pathology of Exposure to Stress: A Treatise Based on the Concepts of the General-Adaptation-Syndrome and the Diseases of Adaptation, Montreal, Acta, Inc., 1950.

Instead of showing the usual signs of vasomotor collapse and shock, the patient appeared somewhat flushed, his respirations were labored, and he appeared to have air hunger. His axillary temperature rose to 102 F. The patient lost consciousness and died three hours after surgery.

Autopsy Findings.—An autopsy was performed 21 hours after death. There was evidence of marked generalized rheumatoid arthritis. The operative area revealed a moderate amount of hemorrhage, which did not exceed that usually associated with this type of operation. There were multiple small parenchymatous hemorrhages in the lungs and a small subendocardial hematoma in the left ventricular aspect of the interventricular septum. All abdominal viscera were severely congested. The most striking observation was pronounced atrophy of both adrenals (see the accompanying figure).

Microscopic Observations.—Microscopic examination of the tissue confirmed the gross findings. The three layers of the adrenal cortex were still identifiable, but each was markedly reduced in width. The cortical cells revealed no appreciable changes. The medulla was likewise atrophic. There were also a few petechiae in the brain parenchyma. The kidneys showed no evidence of a transfusion reaction. Negative sudan IV stains of brain, lungs, and kidneys ruled out fat embolism.

COMMENT

This case is reported to add a note of warning in the immediate preoperative use of cortisone. Experience alone will tell how often this unfortunate outcome will be repeated. If so, appropriate preventive measures will be necessary. They may consist of a period of rest from cortisone therapy, or a period of adrenal stimulation with the use of corticotropin (ACTH), or possibly even the use of larger doses of cortisone through the surgical period.

SUMMARY

A patient who underwent major surgery after receiving cortisone for a period of eight months died of immediate postoperative shock, in spite of normally adequate therapy. Postmortem examination showed marked bilateral adrenal atrophy, suggesting adrenal insufficiency. Judicious use of cortisone in patients about to undergo surgery is cautioned, to prevent adrenal insufficiency.

PERITONEAL CARCINOMATOSIS FORTY-ONE YEARS AFTER RADICAL MASTECTOMY

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and
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The case reported is that of a woman in whom a radical mastectomy had been done at the age of 34 and who died of generalized peritoneal carcinomatosis at the age of 75. The carcinoma of the peritoneum was histologically typical of grade 2+ adenocarcinoma of the breast, while other primary carcinomas were not found. Four months prior to death the patient's only complaints were those related to procidentia, which possibly was due to the presence of ascitic fluid secondary to the carcinomatosis.

REPORT OF CASE

A white widow aged 75 was first seen Aug. 8, 1947. Her complaints were that she had "a growth" in the uterus, a feeling of "weight in the pelvis," urinary symptoms of frequency, nocturia, and dysuria, and protrusion of a mass from the vagina. The protrusion had been noted for years, and she had found it frequently necessary "to push the mass up" in order to urinate.

The patient's mother had died of cancer and her father of pulmonary tuberculosis. She had had two pregnancies, but both had terminated in early miscarriage. The menses had ceased at the age of 50. Her left breast had been removed in 1906 at the age of 34. She was not certain of the reason, but thought she had had cancer. The patient said she weighed 130 lb. (59 kg.) and that her weight had not varied in years. Elimination was reported as satisfactory with the aid of bran. She stated she did not have any abdominal pain, indigestion, or heartburn and had not noted any abdominal swelling. Her appetite had been poor recently.

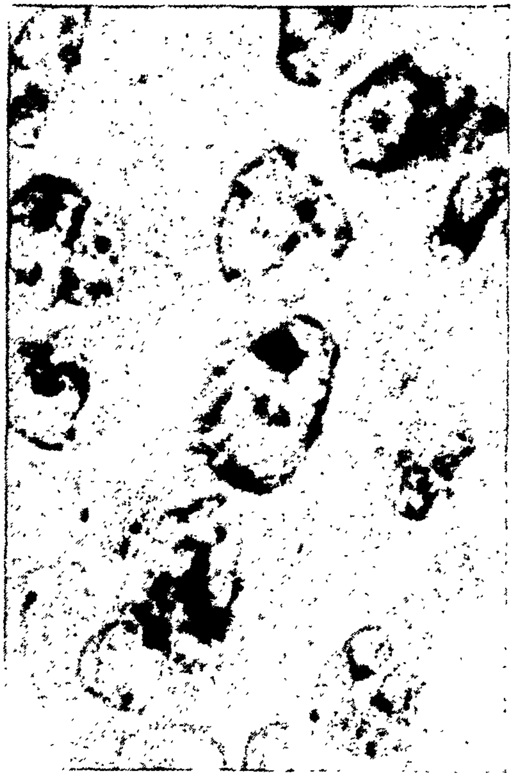

Fig. 1.—Malignant cells found in centrifuged ascitic fluid (tissue block: hematoxylin and eosin stain) (× 1,000).

When the patient was examined, her weight was 130½ lb. (59.23 kg.). Her temperature was 98.2 F (36.8 C), pulse rate 112, respirations 18, and blood pressure 140 systolic, 80 diastolic. The heart and lungs were within normal limits to auscultation and percussion, and the right breast was normal. The left breast and pectoral muscles had been removed radically. The abdomen was moderately distended and tympanitic, and a small umbilical hernia was noted with a slight tenderness in this area. The pelvic outlet was relaxed and the cervix was normal in appearance. The uterus was completely prolapsed and irregular in outline, and cystocele and rectocele, both grade 3, were present. The urine contained an occasional pus cell and one to two red blood cells per high power field. The hemoglobin was 13.6 gm., 88% (Wintrobe). The erythrocyte count was 4,500,000, the color index 0.97, and the leukocyte count 8,000.

On Aug. 12 the patient was hospitalized. A flat film of the abdomen demonstrated the presence of fluid. A vaginal hysterectomy, followed by anterior and posterior colporrhaphy with insertion of Kelly stitches, was done Aug. 14. When the peritoneum was opened, about 2,000 cc. of blood-stained fluid escaped. The oviducts and ovaries seemed normal to palpation except for their adherence to the posterior surface of the broad

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ligaments. Visualization was impossible. The uterus was enlarged and irregular in outline because of the presence of fibroids. The pelvic peritoneum was studded by scattered, discrete implants, each approximately 2 to 3 mm. in diameter, but it was not possible to remove one for biopsy. Whole blood, 500 cc., was given during surgery. The examination of the tissues removed at surgery indicated chronic cervicitis and uterine leiomyomas, although there was no evidence, either gross or histological, of malignant change.

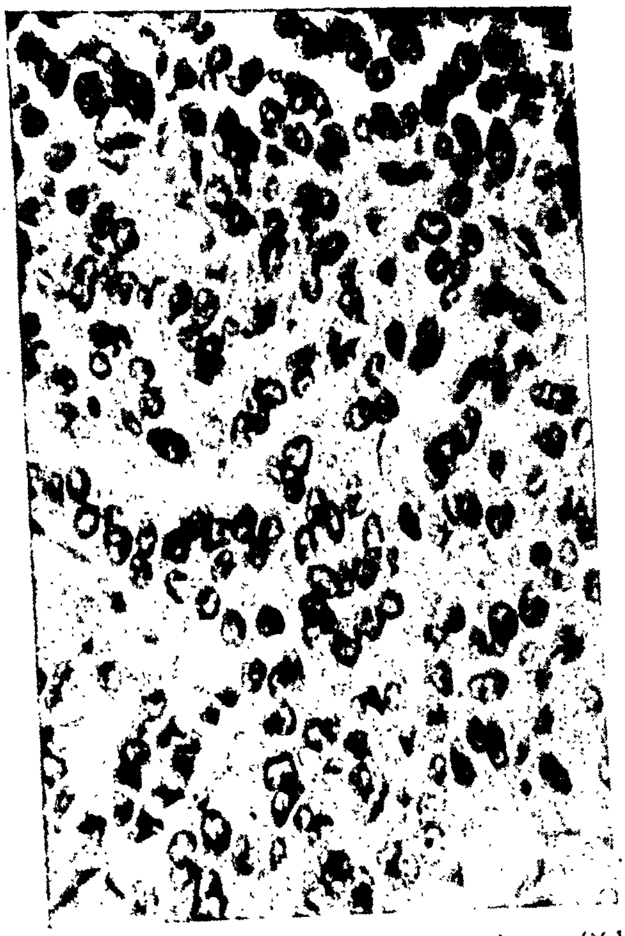

Fig. 2.—Adenocarcinoma pattern metastatic to peritoneum (× 160).

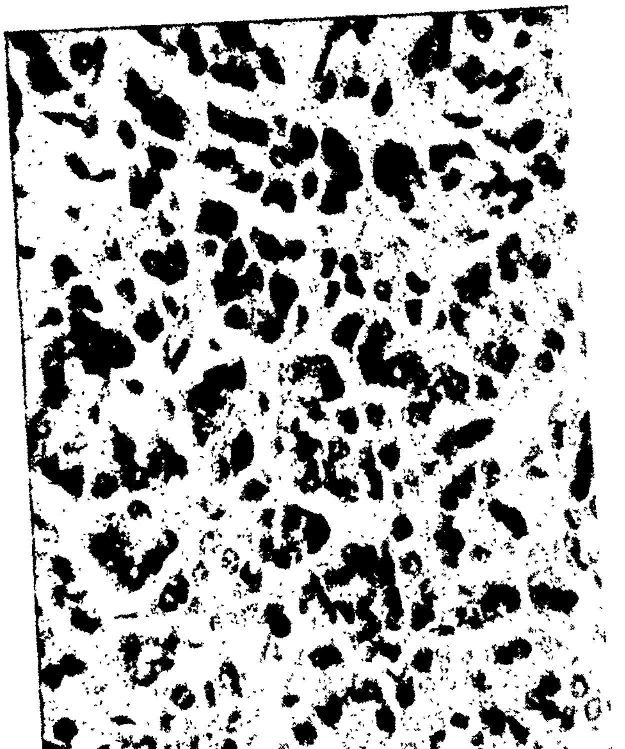

Fig. 3.—Note gland reduplication pattern of adenocarcinoma metastatic to peritoneum similar to mammary carcinoma (× 500).

The postoperative course was complicated the first few days by right-sided bronchopneumonia, possibly on the basis of a pulmonary infarct of the right lung. The patient left the hospital Aug. 30, the 15th postoperative day. During the next three months the patient had recurring episodes of ascites and paracentesis was required three times. The histological examination of centrifuged ascitic fluid on Oct. 7 indicated peritoneal adenocarcinomatosis (fig. 1). The patient lost considerable weight and complained of anorexia, nausea, and progressive weakness. She was readmitted to the hospital Nov. 23 and died Dec. 9. During this period of hospitalization, treatment was palliative.

The postmortem examination revealed generalized peritoneal carcinomatosis. The omentum had become a neoplastic mass, and the entire peritoneum, from the diaphragm to and including the pelvic peritoneum, was studded with dense collections of small and large neoplastic nodules. The ovaries had undergone senile atrophy and were rather easily separated from the small neoplastic masses of tissue studding their serosal surfaces. Neither ovary was involved with primary neoplasia. The esophagus, the thyroid, the lungs, the entire gastrointestinal tract from the cardiac end of the stomach to the anus, the pancreatic parenchyma, the kidneys, the adrenal glands, the urinary tract, and the bladder were carefully inspected for evidence of metastasis of neoplasm or of primary neoplasm. None was found. The remaining breast was carefully examined by multiple, closely spaced incisions of the parenchyma, but no neoplasia was noted. The carcinoma of the peritoneum was typical of a grade 2+ adenocarcinoma of the breast, and there was fairly good reduplication of breast acinar tissue in this peritoneal carcinoma (fig. 2 and 3). Other primary carcinomas were not found.

COMMENT

Unfortunately it has not been possible to trace the nature of the original tumor of the breast removed from this patient in 1906. The histological evidence points to adenocarcinoma of the breast as the primary neoplasm in this case. The clinical history suggests that ascites secondary to the peritoneal carcinomatosis had been sufficiently extensive to cause, in whole or in part, the uterine prolapse noted by the patient for a number of years. Four months before her death, however, she denied abdominal pain or swelling, although stating she was extremely uncomfortable. All physical observations at that time were within normal limits, with the exception of the pelvic and urinary symptoms incident to the uterine procidentia. Her weight had been unaltered for years, and her appetite had been good until about five months prior to her death.

A search of the available literature reveals only one other case in which so long a time elapsed between discovery of the original lesion and the appearance of metastases. In that case, reported by Davidson and Ratcliffe,[1] a bilateral mastectomy had been done in 1902. The diagnosis of cancer had been confirmed microscopically, although the original record and slides were not available. Early in 1942, and again in 1943, small masses in the region of the left sternomastoid muscle were removed. The patient died in 1945. At necropsy all evidence of metastases was limited to the thoracic cavity. The tissues from the neck and the thorax had considerable cytological resemblance to both the basal cell carcinoma of the skin and the intraductal carcinoma of the breast.

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[1] Davidson, W. R., and Ratcliffe, A. W.: Metastatic Carcinoma Afte Forty Years, J. Indiana M. A. 39: 165 (April) 1946.

The survival of a patient 31 years after removal of a breast for cancer was reported by Steward in 1925.[2] The patient died within a few months after the recurrence of the lesion was noted. This consisted of three small, hard nodules beneath the skin of the chest near the mastectomy scar. There was suggestive evidence of a mediastinal growth. A necropsy is not mentioned, nor is there any indication that a microscopic examination was made of any of the suspicious lesions, or of the tissue removed at mastectomy.

Kretzler's [3] patient had a mastectomy for cancer in 1902. A recurrence was first noticed in 1916, yet death from generalized metastases did not occur until 1926, 24 years after the original mastectomy. Clinically, in 1924, the metastases seemed to involve the other breast, the axilla, downward toward the navel, and up along the right side of the neck. The lung also had evidence of marked involvement. Necropsy was done, but the extent of the metastases is not given in the report. Tissue obtained at necropsy was classified histologically as adenocarcinoma of the breast, but the site of this tissue is not given. Chilko and Quastler [4] reviewed the literature on metastasis in cancer of the breast in 1942. The longest elapsed time between mastectomy and the recurrence of metastasis cited by them was 16 years.

Metastasis confined to the peritoneum below the diaphragm is another feature reported infrequently. Willis,[5] in his study of the spread of tumors, published in 1934, considered that long-delayed metastatic recurrence is by no means infrequent, especially in mammary carcinoma. In none of the data included in his study, however, does he cite a case of metastasis confined to the peritoneum.

Josephy [6] records a 23 year survival after mastectomy for cancer. The operation was done in 1927. Two years later there was metastasis to the axilla, and 19 years after, metastasis to the opposite axilla (the opposite breast not being affected). In 1950, 23 years after the original mastectomy, the patient died. The abdominal cavity contained about 4 liters of fluid. There were metastases to the omentum, the mesentery, and the pelvic and diaphragmatic peritoneum and also to the leptomeninges and the subarachnoid space. The lungs, stomach, intestines, liver, gallbladder, spleen, pancreas, suprarenal glands, ovaries, uterus, urinary bladder, and brain were searched for evidence of primary tumor, but none was found. Microscopic examination revealed a diffuse carcinomatous invasion by way of the lymphatics, with little tendency to form large nodes or masses. The cells were identical with those in the axillary lymph nodes removed in 1948.

Woolsey [7] reported a case in which the breast had been removed for cancer in 1905. Sixteen years later the patient was alive and well. In the intervening period a pelvic operation and a cholecystectomy had been done. At one time she complained of abdominal pain, gas, vomiting, loss of appetite, and headache; but a roentgenologic examination was normal, and her symptoms disappeared, so that suspicion of an abdominal metastasis was not confirmed.

SUMMARY

A case of carcinomatosis confined to the peritoneum 41 years after radical mastectomy is reported. A brief search of the literature indicates that cases in which there is either the type of metastases or the long duration recorded here are infrequently reported.

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[2] Steward, F. J.: Cancer of the Breast: Recurrence 31 Years After Operation, Brit. M. J. 1: 156 (Jan. 24) 1925.

[3] Kretzler, H. H.: Cancer of 25 Years Duration, Northwest Med. 26: 138 (March) 1927.

[4] Chilko, A. J., and Quastler, H.: Delayed Metastases in Cancer of the Breast, Am. J. Surg. 55: 75 (Jan.) 1942.

[5] Willis, R. A.: The Spread of Tumours in the Human Body, London, J. & A. Churchill, Ltd., 1934, p. 112.

[6] Josephy, H.: Carcinoma of the Mammary Gland: Death from Metastases 22 Years After Radical Mastectomy, Illinois M. J. 99: 270 (May) 1951.

[7] Woolsey, G.: Late Recurrence After Radical Operation for Carcinoma of the Breast, Ann. Surg. 80: 932 (Dec.) 1924.

PULMONARY RESECTION FOR METASTASIS FROM CARCINOMA OF THE LUNG

REPORT OF A CASE

Adrian Lambert, M.D., New York

In a recent summary of the literature concerning pulmonary resection for metastatic malignant lesions, Seiler, Clagett, and McDonald [1] found 68 cases recorded up to 1950. In the 63 cases in which the type of the primary tumor was mentioned, the most frequently occurring types were carcinoma of the large bowel (11 cases), fibrosarcoma (9 cases), hypernephroma (8 cases), and carcinoma of the ovary (5 cases). In the entire series there were no cases reported of pulmonary resection for metastasis from the lung as the primary site of the tumor. The fact that this has not been done is not because of the rarity of this type of extension. Ochsner and DeBakey [2]

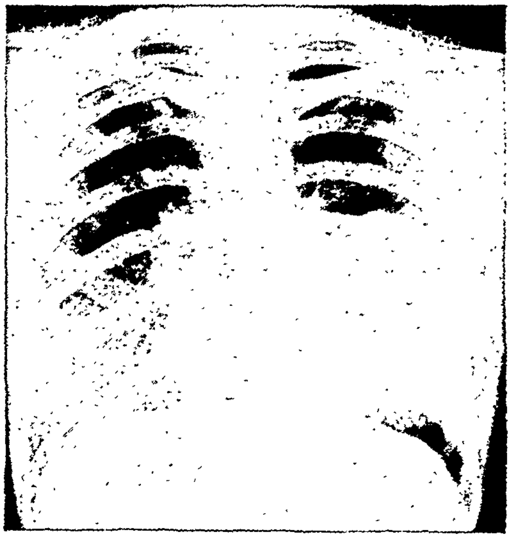

Fig. 1.—Roentgenogram (posteroanterior view) taken one month before first lobectomy was done, showing tumor in the upper lobe of the left lung.

Several illustrations have been omitted from THE JOURNAL and will appear in the author's reprints.

[1] Seiler, H. H.; Clagett, O. T., and McDonald, J. R.: Pulmonary Resection for Metastatic Malignant Lesions, J. Thoracic Surg. 19: 655-679 (May) 1950.

[2] Ochsner, A., and DeBakey, M.: Significance of Metastasis in Primary Carcinoma of Lung: Report of 2 Cases with Unusual Site of Metastasis, J. Thoracic Surg. 11: 357-387 (April) 1942.

ligaments. Visualization was impossible. The uterus was enlarged and irregular in outline because of the presence of fibroids. The pelvic peritoneum was studded by scattered, discrete implants,

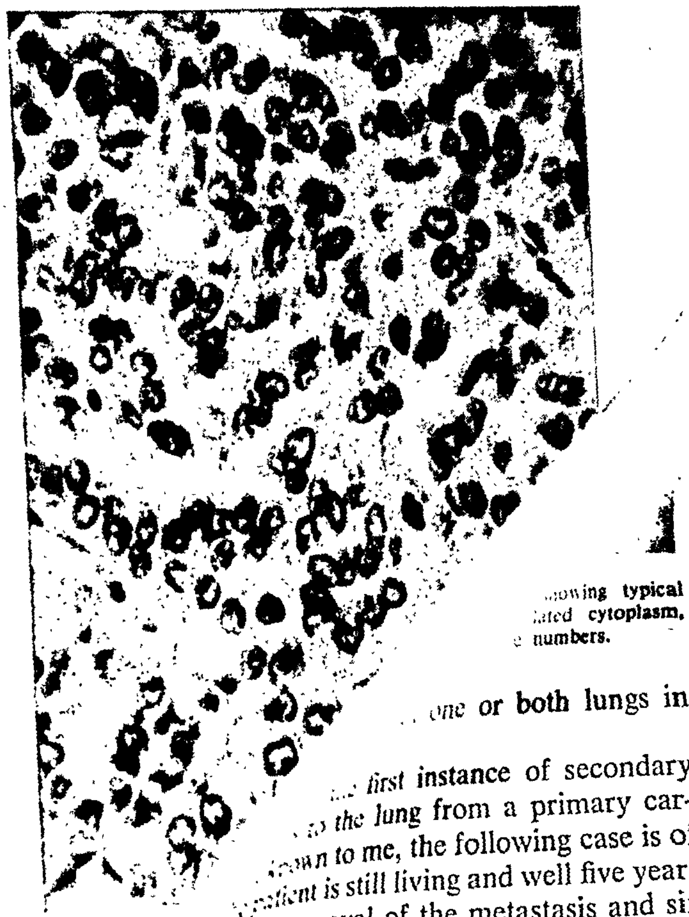

Fig. 2.—Adeno ...owing typical ...ated cytoplasm, ...e numbers.

... one or both lungs in

... first instance of secondary ... the lung from a primary car- ...own to me, the following case is of ...patient is still living and well five years ...after removal of the metastasis and six ... months after removal of the primary ...possibility of a blood-borne metastasis was ...after careful pathological study of the dis- ...ctions removed in the first operation, which may ...contributed to the successful ultimate result.

REPORT OF A CASE

A 28-year-old woman was observed during routine roentgeno-graphic examination to have a large, circular, well-circumscribed shadow in the left upper lung lobe that extended down anterior-ly almost to the diaphragm (fig. 1 and 2). The only symptoms

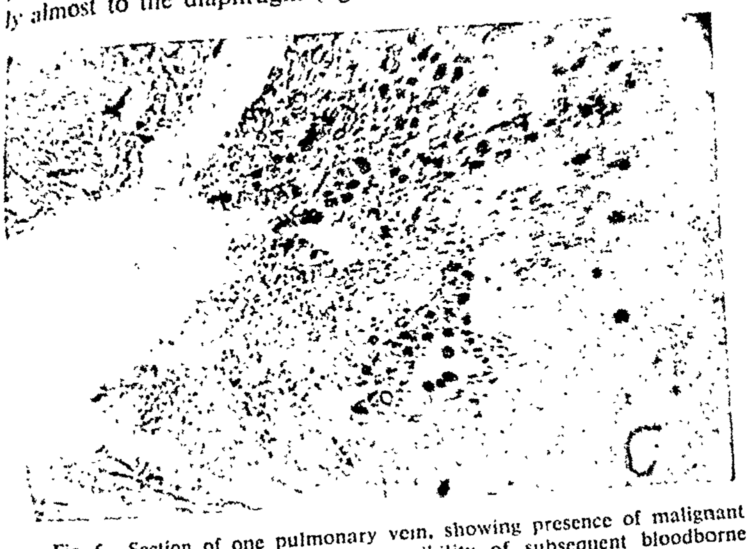

Fig. 6.—Section of one pulmonary vein, showing presence of malignant cells in the lumen, suggesting the possibility of subsequent bloodborne metastasis.

she had were fatigue and a slight cough for three weeks before her admission to Roosevelt Hospital on Jan. 25, 1946. On physical examination she appeared to be normal, except for a small amount of dullness on the anterior axillary line on the left side of her chest. Results of laboratory tests were as follows: hemoglobin, 91%; white blood cell count, 10,300 per cu. mm.,

at surgery indicated ch ... phocytes; the urine
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The postoperat ... and was found
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specimen was re... ed one pulmonary vein to con-
tain a small ... r cells (fig. 6). The patient was,
therefore, ... y for the possibility of blood-borne
metastases. ... nths later, a mass developed in her left
thigh, and it was excised as a possible metastatic lesion; it turned
out to be only fibrous tissue that was diagnosed pathologically
as a benign fibroma.

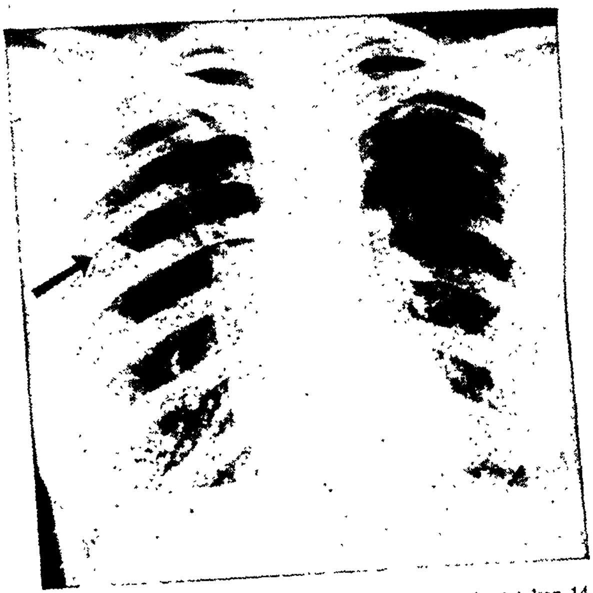

Fig. 7.—Roentgenogram (posteroanterior view) of chest taken 14 months after left upper lobectomy for primary tumor, showing (arrow) metastatic lesion, 1 cm. in diameter, in upper lobe of the right lung.

The patient remained free of symptoms until one year from her first operation, at which time she developed a slight non-productive cough. She felt well and had lost no weight. A roent-genogram made at this time showed a small, nodular, discrete shadow about one-half centimeter in diameter in the upper lobe of the right lung. It had the appearance of a metastatic nodule and in subsequent roentgenographic examination it was observed to have enlarged slightly (fig. 7 and 8). A diagnosis of metastatic carcinoma was made, and the patient was brought into Roosevelt Hospital for an exploratory operation 14½ months after her first lobectomy. At operation, a right upper lobectomy was done for a metastatic tumor in the upper lobe of the right lung (fig. 9), which on microscopic examination was seen to consist of the same type of tissue as the primary growth. The glands were atypical, columnar in type, and arranged in multiple layers. The nuclei were fairly uniform in size and rich in chromatin, with about one mitotic figure per high power field (fig. 10). The regional lymph nodes removed before lobectomy showed n malignancy.

Following the operation, the patient had an uneventful re
covery and was allowed to go home on the 14th postoperativ
day. Since that time she has been well, and, except for an oc
casional slight cough due to a cold, she has been free of symp
toms. At no time has she been conscious of getting out o
breath. A recent roentgenogram showed no evidence of furth
metastasis (fig. 11). It is now six years and five months sin
her first operation and five years and two months since h
second.

COMMENT

Isolated cases [3] of removal of solitary metastatic nodules in the lung have been reported in the literature since the first case was reported by Divis in 1927.[4] It was not until Churchill [5] reported a nine year follow-up after removal of a single metastatic lesion that it became evident that prognosis was not rendered hopeless by the presence of pulmonary metastatic carcinoma. Since that time, Alexander,[6] Blades,[7] Clagett,[1] and others have added case reports to the literature, the opinion now being that removal of a solitary malignant metastatic lesion in the lung is indicated after adequate removal of the primary tumor in patients in which there is no evidence of further metastasis. In the present patient the primary tumor was removed before any symptoms had occurred, and yet it had already undergone early microscopic metastasis via the blood stream, as so frequently occurs in adenocarcinoma.

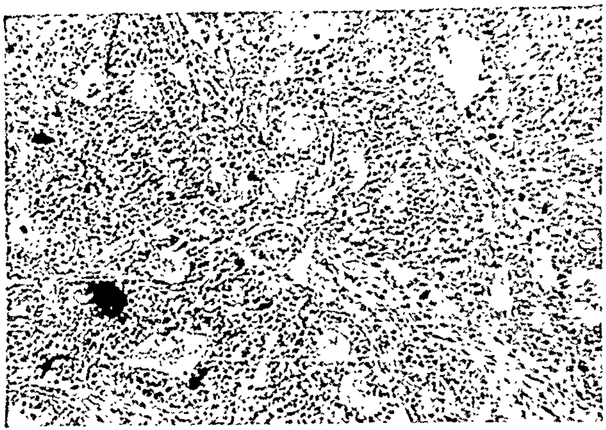

Fig. 10.—Microscopic section of secondary tumor, showing identical type of glandular epithelium as present 15 months earlier in primary tumor.

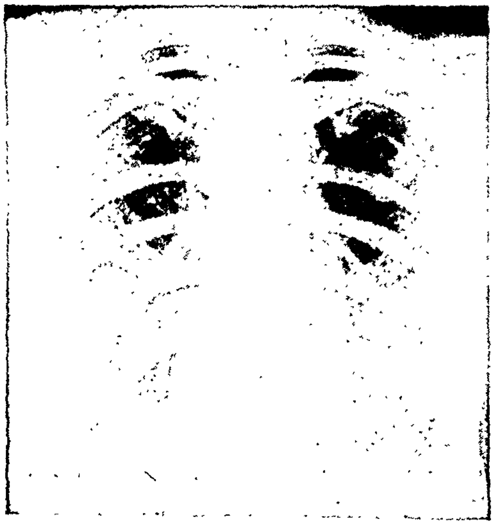

Fig. 11.—Roentgenogram (posteroanterior view) taken five years and three months after the original lobectomy and four years and one month after removal of the metastatic lesion, showing no further evidence of recurrence of carcinoma.

As early as 1903, Schmidt [8] noted how frequently cancer cells occur within the pulmonary vessels in primary carcinoma of the lung. In 1931, Fried [9] concluded that this is the essential manner of dissemination in bronchogenic carcinoma, because the condition is invariably present. This was corroborated by Boyd,[10] who noted blood vessel invasion in all patients in whom distal metastasis had occurred. Later, Koletsky [11] concluded that the adenocarcinoma type was more likely to metastasize by way of the blood stream and produce widely distributed metastatic lesions than the other types. In 1941, Gebauer [12] corroborated this conclusion in a series of 158 cases.

It is interesting to speculate on the method of extension. In the present case, the cancer cells may have travelled from the pulmonary vein through the left auricle and ventricle to the right lung by way of the bronchial artery, or they may have reached the lung by way of the pulmonary artery after traversing the general circulation. A third possible route for hematogenous metastasis is from the left pulmonary vein through the left auricle to the right pulmonary vein and into the other lung. This would depend on a tidal type of flow in the pulmonary vein, which might occur in the presence of an open chest. The route of spread can not be proved, but it is of significance in the evaluation of early metastasis.

In the present case, absence of lymph node involvement in the several glands removed from the mediastinum and the complete gross containment of the tumor in the upper lobe at the first operation were considered indications for lobectomy rather than pneumonectomy. This decision enabled further resection of the lung for metastatic involvement of the other side without making the patient a respiratory cripple. It is felt, therefore, that lobectomy is the operative method of choice in certain selected cases, such as this one, in which the patient's tumor was localized and was of the type that metastasizes first through the blood stream.

SUMMARY

A case of the removal by lobectomy of an adenocarcinoma of the upper lobe of the left lung and of a solitary metastasis to the right lung occurring 14½ months later is reported. The patient is still living and well six years and five months after the first lobectomy and five years and two months after the second lobectomy for the metastatic lesion.

768 Park Ave. (21).

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[3] Torek, F.: Removal of Metastatic Carcinoma of the Lung and Mediastinum: Suggestions as to Technic, Arch. Surg. **21**: 1416-1424 (Dec.) 1930. Lilienthal, H.: Pneumonectomy for Sarcoma of the Lung in a Tuberculous Patient, J. Thoracic Surg. **2**: 600-611 (Aug.) 1933. Ropke, W.: Mehrjahrige Hielung nach Operation eines Lungenkarzinoms, Zentralbl. f. Chir. **64**: 803-806, 1937.

[4] Divis, G.: Ein Beitrag zur operativen Behandlung der Lungengeschwülste, Acta chir. Scandinav. **62**: 329-341, 1927.

[5] Barney, J. D., and Churchill, E. D.: Adenocarcinoma of Kidney with Metastasis to Lung Cured by Nephrectomy and Lobectomy, J. Urol. **42**: 269-276 (Sept.) 1939.

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[7] Effler, D. B., and Blades, B.: Surgical Treatment of Solitary Lung Metastasis, J. Thoracic Surg. **17**: 27-37 (Feb.) 1948.

[8] Schmidt, M. B.: Verbreitungswege der Carcinome und die Beziehung generalisierter Sarkome zu den leukamischen Neubildungen, Jena, Gustav Fischer, 1903.

[9] Fried, B. M.: Primary Carcinoma of the Lungs, Arch. Int. Med. **35**: 1-41 (Jan.) 1925.

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ADENOMATOUS APPENDICULAR POLYPS

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G. J. Coloviras, M.D.
and
S. M. Wallace, M.D., Detroit

Adenomatous appendicular polyps are rare. White and Ellis[1] collected a total of 21 cases from the literature in 1945 and added a single case of their own. In 1948 Brewer and Marcus[2] described a patient in whom the polyp had caused an intussusception of the appendix. They were able to gather four other similar cases from older literature, prior to 1923. Numerous workers[3] have suggested that these polyps are precancerous lesions with potentialities similar to those of adenomatous polyps of the colon. Uihlein and McDonald[3c] reported cases studied photomicrographically showing "an adenomatous polyp" containing "regions of malignancy characterized by larger deeper-staining nuclei." They prefer to call tumors with anaplastic areas of that type "papillary adenocarcinoma grade I." In another paper Woodruff and McDonald[4] described a role that adenocarcinoma grade 1 of the appendix may play in the formation of a mucocele and of pseudomyxoma peritonei. We wish to report a case of multiple adenomatous polyps of the appendix complicated by acute appendicitis. According to Collins' review of cases,[5] adenomatous polyps of the appendix are usually single but may be multiple. They are usually found in the age group between 6 and 40 years. The majority of polyps are small, measuring from about 0.5 to 3.0 cm. in length. The polyps are usually found in the middle third of the appendix. Over half of the reported cases were complicated by appendicitis. A few cases have been associated with moderate hemorrhage into the intestinal tract.

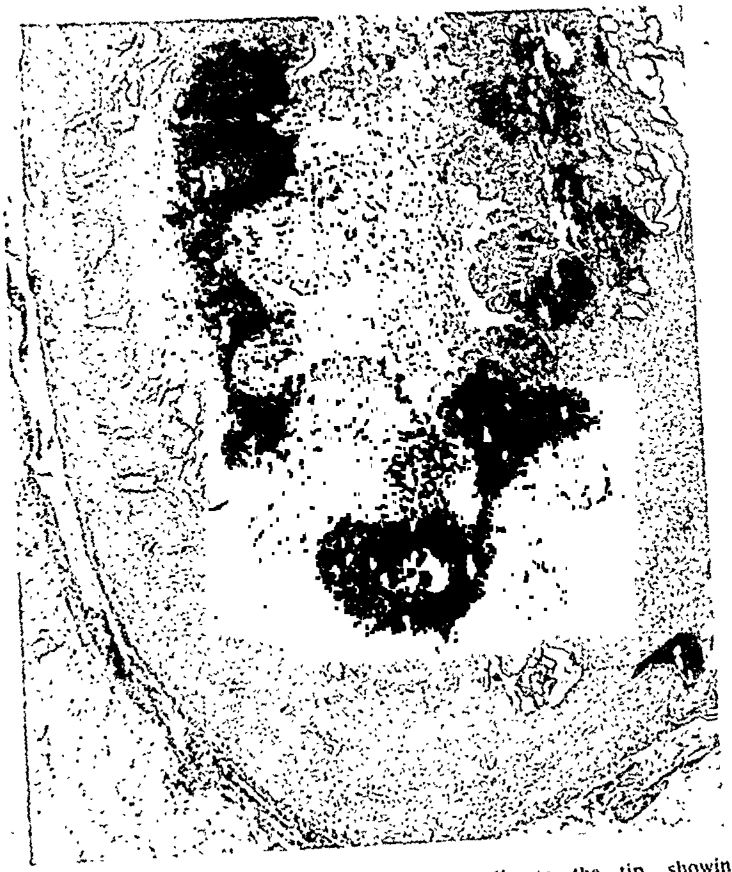

Fig. 1.—Longitudinal section of the appendix to the tip, showing multiple small microscopic polyps (hematoxylin and eosin, × 10).

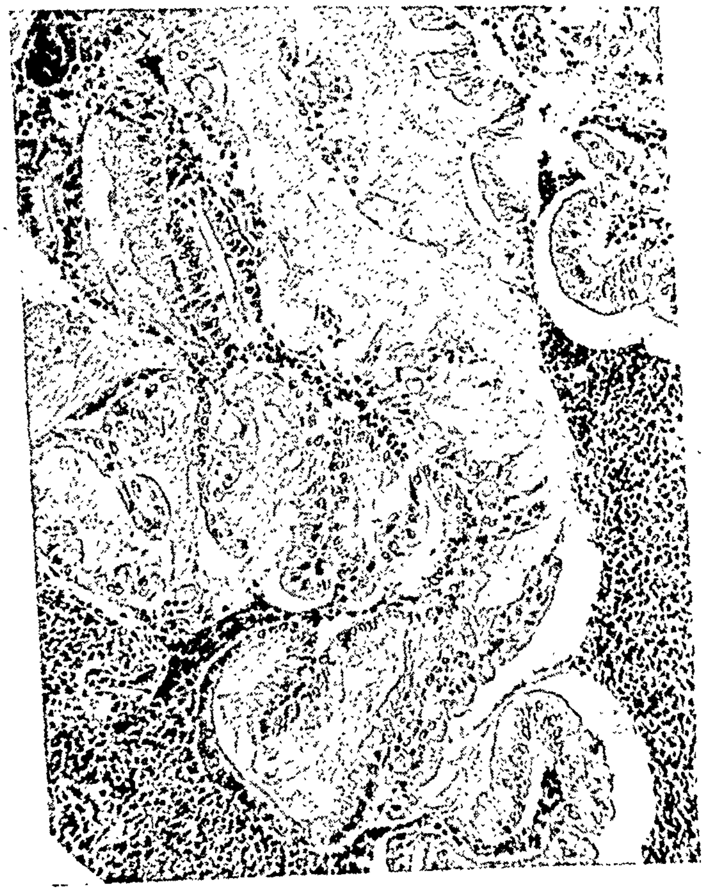

Fig. 2.—Photomicrograph of a small portion of the largest polyp, showing well-differentiated mucus-producing columnar cells and many polymorphonuclears (× 250).

REPORT OF A CASE

A 41-year-old white foundry foreman was admitted to Herman Kiefer Hospital on Sept. 17, 1951, with far-advanced tuberculosis and silicosis. While being treated with streptomycin, paraaminosalicylic acid, and bed rest for his tuberculosis, on Dec. 4, 1951, he suddenly experienced abdominal pain. The discomfort was generalized at first but became localized in the right lower abdominal quadrant within 24 hours. The pain was cramplike, and there was tenderness on palpation over the appendix. Some rebound tenderness was also noted in the right lower quadrant. The patient's appetite was unimpaired, and he had had three bowel movements since the onset of his abdominal pain. The patient had a temperature of 101 F and a pulse rate of 120. The leukocyte count was 15,400, with a differential count of 89% polymorphonuclear neutrophils, 6% lymphocytes, 3% monocytes, and 2% eosinophils.

There was no history of previous attacks similar to the present illness. An appendectomy was done on Dec. 5. The subserosal vessels were congested. There was a small amount of free fluid about the appendix. On culture this proved to be sterile. No masses were noted in the cecum or immediately adjacent ileum. The rest of the abdominal cavity was not explored. The patient's postoperative course was uneventful. At the time of writing the patient was continuing treatment for tuberculosis.

From the departments of pathology and surgery of Herman Kiefer Hospital and Wayne University College of Medicine.

[1] White, J. W., and Ellis, R. C.: Polyps of the Appendix, Am. J. Surg. **70**: 389-393 (Dec.) 1945.

[2] Brewer, A. C., and Marcus, R.: Adenomatous Appendicular Polyp Causing Intussusception, Brit. J. Surg. **35**: 434 (April) 1948.

[3] (*a*) Emge, L. A., and Cowan, C. B.: An Early Benign Adenoma of the Appendix, S. Clin. North America **3**: 765-774 (June) 1923. (*b*) Sanes, S., and Patchin, D. F.: *Polyposis* of the Vermiform Appendix: Report of a Case, Arch. Surg. **44**: 912-917 (May) 1942. (*c*) Uihlein, A., and McDonald, J. R.: Primary Carcinoma of the Appendix Resembling Carcinoma of the Colon, Surg., Gynec. & Obst. **76**: 711-714 (June) 1943.

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[5] Collins, D. C.: Adenomatous Polyp of the Vermiform Appendix, S. Clin. North America **12**: 1063-1067 (Aug.) 1932.

Gross Description.—The appendix measured 6 by 1 cm. The serosal vessels were moderately injected, but no exudate was visible on the serosa. On cut section the appendiceal wall was seen to be thickened, white, and firm. The lumen was small and stellate. At a point near the middle of the appendix there was a small polypous mass projecting into the lumen. This mass measured about 8 mm. in its greatest dimension. Its surface was finely granular. Multiple smaller polypous masses projecting into the lumen were seen out toward the tip.

Microscopic Findings.—A cross section of the appendix through the polyp showed a papillomatous arrangement of tall columnar mucus-secreting cells. The cells were arranged in single row about a central fibrous stroma with a delicate, branching, fern-like structure. The nuclei of the cells were well oriented at the base of the cell. No bizarre hypochromatic nuclei were found, and no mitotic figures were seen. Numerous crypts were formed in the polyp. The appendiceal lumen also contained many polymorphonuclear neutrophils and necrotic debris. There was ulceration of the mucosa and widespread infiltration of the submucosa and subserosa with acutely inflammatory cells. No epithelial infiltrations of the appendiceal wall were found. The polyp did not completely occlude the lumen at any point. A longitudinal section of the distal 2 cm. of the appendix showed multiple small hyperplastic areas of redundant mucosa forming microscopic polyps with broad bases.

COMMENT

The structure of the polyp present in our case was similar to that shown in photomicrographs of many previously reported cases. We agree with those authors who believe that this tumor should be regarded as similar in malignant potentialities to adenomatous polyps of the colon. We also feel that obstruction of the lumen of the appendix was an important factor in inducing acute inflammation in this case.

1151 Taylor Ave.

LONGEVITY WITH COMPLETE ATRIOVENTRICULAR BLOCK

Julien E. Benjamin, M.D., Cincinnati

and

Paul D. White, M.D., Boston

Two case reports of patients known to have complete heart block for 35 years are presented for the purpose of adding to the literature further information on the important aspect of prognosis in this disease of the heart. Atrioventricular block has always been an important clinical problem in itself and also in indicating the existence of serious disease or toxic states. Occasionally, however, and possibly more often than is generally appreciated, etiological factors may influence strikingly the course of this disease.

REPORT OF CASES

Case 1.—Miss L. W., aged 57, a white registered nurse, was known to one of us (J. E. B.) for 35 years. In 1910, when she was hospitalized for a minor surgical procedure, it was noted that she had a "slow pulse." The patient's mother told her that when she was very young the family doctor had observed her unusually slow pulse rate. She had a remarkably healthy childhood and had engaged in all the games characteristic of her play group. Her development was in every way normal. She had had no serious illness at any time and no major operations. She entered nurses' training school (Cincinnati General Hospital) at the age of 19 and passed the entrance physical examination but again was informed of her slow heart rate. Her first electrocardiogram, a pioneer record in the Middle West, taken in 1916 when she was 23 years old, showed complete heart block (fig. 1). During the training period her schedule was strenuous, involving 12 hour shifts of day and night duties, alternating every 3 or 4 months. After graduating she was assigned, in a supervisory capacity, to medical wards, and her work was exacting and strenuous. She rarely lost time due to intercurrent minor illnesses. She continued in this role for 15 years. In the following years she either worked as a private duty nurse or did nursing in the outpatient dispensary. During the past five years she worked daily in one of the nurses' dormitories supervising several activities. Her health remained astoundingly well. It is noteworthy that at no time in the years she was under observation did she have any complaints referable to her heart or circulation. She was a very stable person.

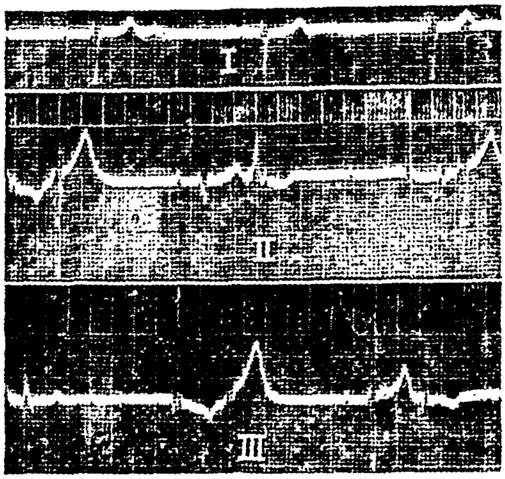

Fig. 1.—Electrocardiogram of patient L. W., taken in 1916, showing complete atrioventricular block, with atrial rate of 80 to 85 and ventricular rate of 40 beats per minute (except at the time of ventricular bigeminy).

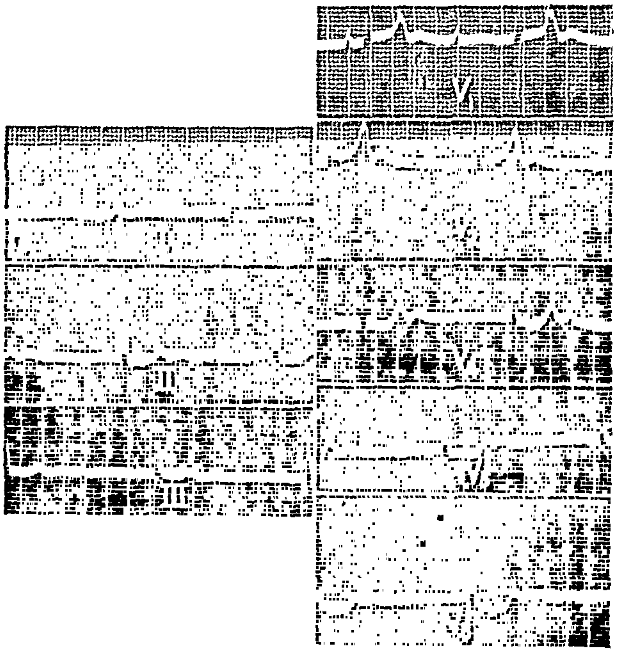

Fig. 2.—Electrocardiogram of patient L. W., taken Sept. 20, 1950, showing complete atrioventricular dissociation, with atrial rate of 60 and ventricular rate of 30 beats per minute. The atrial beats came at irregular intervals, whereas the ventricular beats came at regular intervals.

In February, 1948, the patient was examined because of a sense of exhaustion, residual from a recent upper respiratory tract infection, and it was noted that the pulse was irregular and

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Dr. Jan Schwarz performed the autopsy reported in case 1 of this article.

rapid. An electrocardiogram later revealed unmistakable atrial flutter and high-grade atrioventricular block. The patient had no complaints referable to her heart. The atrial flutter subsided in a few days with digitalis and quinidine therapy and rest in bed. It is doubtful whether medication influenced this disturbance in rhythm. There was no recurrence, and her circulation was not disturbed by this episode. Evidence of atrioventricular block was again apparent and remained so. She continued her daily activities and worked eight hours each day with no discomfort.

In a recent physical examination, the patient was about 20 lb. (9.1 kg.) overweight, in good health, and of a genial nature. She had no physical complaints. The optic fundi showed grade 1 changes in the vessels and the peripheral vessels were soft; the veins showed no gross changes. A roentgenographic examination indicated the heart was generally somewhat enlarged. There were no abnormal pulsations. The neck vessels were essentially normal. The heart rate was 32 beats per minute at the apex, changing little on exercise, and there was no pulse deficit. The sounds were clear and sharp, with A_2 considerably louder than P_2. A grade 3 rough systolic murmur was heard over the entire precordium; it was louder at the apex, was not transmitted to the axilla, and was heard distinctly between the scapulas. The blood pressure was 180 mm. Hg systolic and 65 mm. Hg diastolic; this had varied only slightly in many years. Capillary pulsations were easily demonstrable. A third heart sound was frequently audible. In fluoroscopic examination, a sclerotic aortic valve was vaguely discernible and the dissociated rhythm of the chambers that could be observed occasionally was impressive. The lungs were normal. The circulation time (arm to tongue) was 32 seconds. The venous pressure was 21 mm. of water at the level of the right atrium. The vital capacity was 69% of normal. The last electrocardiogram made before the patient died is shown in figure 2 and is exactly as the record had been for many years, except for the electrocardiogram made in February, 1948. Abdominal, pelvic, and neurological examinations were unrevealing. The results of urinalysis and hematological tests were within normal ranges.

On May 10, 1951, the patient experienced a sudden attack of dyspnea (while at rest), which persisted in varying degrees up to the time of her death. There were no other signs of heart failure; however, the venous pressure was 280 mm. of water and the circulation time was 30 seconds (dehydrocholic acid [decholin®] method). The brachial arterial pressure was 140/60 mm. Hg. The cardiac rate was 40 beats per minute. An electrocardiogram revealed an atrial flutter, with 7 or 8 to 1 heart block, the ventricular rate varying from 40 to 50 beats per minute. The patient was given 24 grains (1.55 gm.) of quinidine sulfate during the ensuing 24 hours, with return of the previous complete heart block and disappearance of the flutter. On May 11, 1951, several hours after quinidine therapy was discontinued, the patient had a generalized convulsion, following which she became extremely dyspneic and cyanotic. During the seizure the pulse could not be felt, but a few moments after her return to consciousness the former slow rate returned and there was no notable change in her general condition (Adams-Stokes attack [?]). Five hours later, a syncopal attack occurred, which lasted long enough for an emergency electrocardiogram to be taken, revealing ventricular tachycardia, which vanished with the patient's return to consciousness. For the ensuing eight hours cyanosis and orthopnea persisted, although the patient was receiving oxygen by a nasal catheter. Diuretics produced no effect, although administered early in the illness. This was also true of other medicaments. The patient stopped breathing suddenly 36 hours after the onset of the final illness.

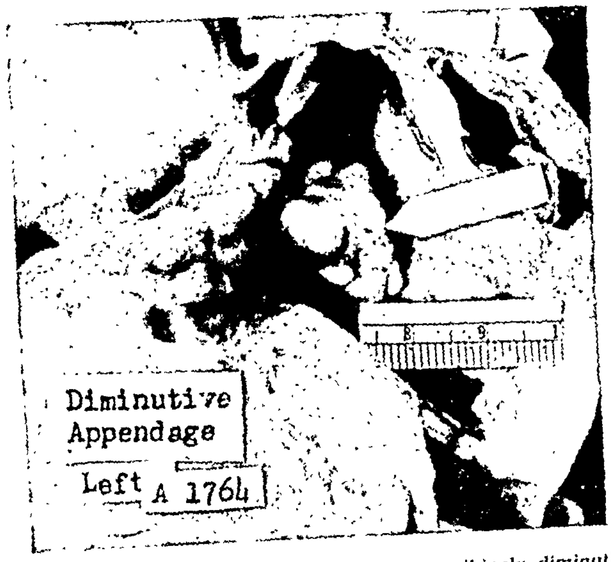

Fig. 3.—Heart of patient L. W., showing the strikingly diminutive size of the left atrial appendage (arrow), which is apparently a vestige. A developmental abnormality was also noted in the left circumflex coronary artery.

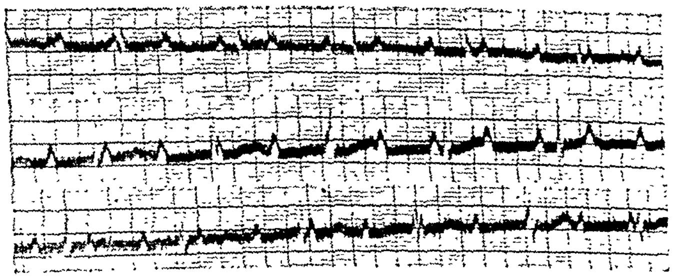

Fig. 4.—Electrocardiogram (reading from top to bottom, leads I, II, and III) of patient C. A., taken Nov. 13, 1916, showing complete atrioventricular dissociation, with otherwise normal complexes. The atrial rate is 120 and the ventricular rate 55 beats per minute. This record was taken while the patient was thyrotoxic.

Autopsy Report.—The conditions observed in the cardiovascular system, the only pertinent ones disclosed, are the only ones reported here.

On opening the chest, approximately 600 cc. of a clear straw-colored fluid was found within each pleural cavity. The pleural surfaces were smooth and glistening. The lungs were expanded and light blue in color. The pericardial sac was opened and was observed to contain approximately 25 cc. of a straw-colored fluid. The heart was enlarged. The first portion of the ascending aorta appeared slightly dilated (aneurysmal), and the wall was calcified. The diaphragm on the right rose to approximately the fifth rib and on the left to the fifth interspace. The heart weighed 590 gm. and had the following measurements:

Circumference of tricuspid ring	11.5 cm.
Circumference of pulmonic ring	6.5 cm.
Circumference of mitral ring	10 cm.
Circumference of aortic ring	6.5 cm.
Length of right ventricle	8 cm.
Length of left ventricle	7 cm.
Thickness of right ventricular wall	3 to 7 mm.
Thickness of left ventricular wall	9 to 15 mm.

The epicardial surface was smooth and glistening. A small amount of subepicardial fat was present over the coronary arteries. The heart was opened in the usual manner, and all valves appeared to be both anatomically and functionally competent. There was an increase in the amount of atheromatous material in the aortic cusp. The first portion of the aorta showed calcification of the wall and moderate dilatation. All chambers were dilated and hypertrophied. Examination of the left atrium revealed the presence of a yellow calcific material, measuring approximately 15 mm. in its greatest diameter, in an area that usually contains the remnants of the foramen ovale. No foramen ovale remnant was visible. Leading into a small left atrial appendage was a narrow orifice measuring approximately 8 mm. in diameter, which had subendocardial suffusion; no mural thrombi were noted. The walls and papillary muscles were strikingly hypertrophied. The chordae tendineae showed no remarkable changes. Examination of the coronary arteries revealed a slit-like opening for the ostium of the left coronary artery; an extremely small left circumflex artery was found. The right and left anterior descending coronary arteries were patent throughout and showed marked sclerosis of their walls. The myocardium was not sectioned because of further study to be made.

The wall of the aorta was thickened and had poor elasticity. There was dilatation of the first portion of the ascending aorta; the remainder, however, showed no aneurysmal dilatation. On opening the aorta, the intima was seen to be yellow in color.

Scattered atheromatous plaques were seen, many of which were calcified, and were most prominent in the abdominal aorta. No ulcerations or dissection was noted. The major branches were fully patent but sclerotic.

The gross pathological diagnosis was as follows:

1. Dilatation and hypertrophy of the heart (moderate).
2. Generalized arteriosclerosis (severe).
3. Ascending aortic aneurysm, arteriosclerotic in type.
4. Diminutive left atrial appendage (fig. 3).
5. Greatly enlarged right atrial appendage.
6. Vestige of left circumflex coronary artery.
7. 15 mm. area of calcification of interatrial septum, left atrial.
8. Congestive failure, lungs and liver.
9. Hydrothorax, bilateral.
10. Severe pulmonary edema, bilateral.

It should be noted that congenital abnormalities were present, namely, vestigial left atrial appendage and left circumflex coronary artery. The calcified plaque in the interatrial septum as well as the greatly enlarged right auricular appendage may also have been present at birth. The assumption that congenital heart block, mentioned in the body of this report, was the etiological factor could well be true. The task of sectioning the region of the bundle of His and its branches is to be undertaken.

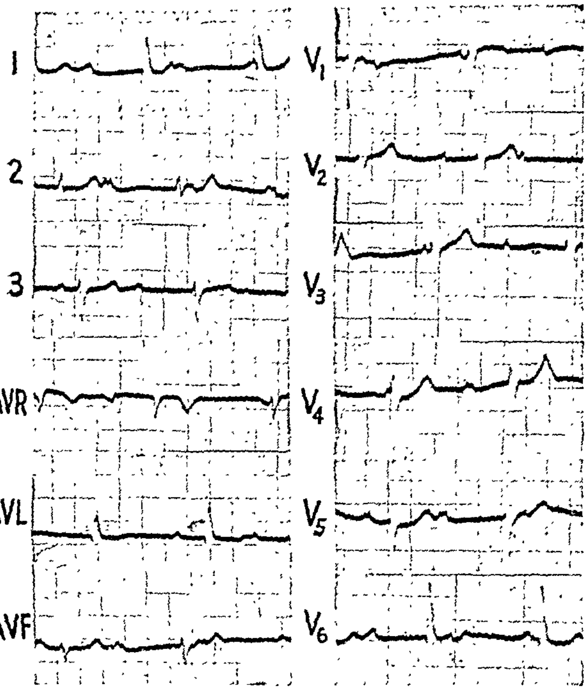

Fig. 5.—Electrocardiogram of patient C. A., taken Dec. 1, 1950, showing complete atrioventricular block, with an atrial rate of 80 to 85 and a ventricular rate of 55 to 60 beats per minute. Except for the atrioventricular block there is no gross abnormality shown in the electrocardiogram. The R waves in V_1, V_2, and V_3 are small but within the normal range.

Case 2.—Miss C. A., aged 58, a music teacher, has been known to one of us (P. D. W.) for 35 years. Her first electrocardiogram was made in November, 1916, at the age of 24. A slow pulse rate (40 beats per minute) had been noted by her family physician 12 years previously, incident to routine examination. She had had severe diphtheria at the age of 1½ years. She has been observed annually during the past 35 years and has continued to be in good health, except for exhaustion and other symptoms of neurocirculatory asthenia and functional disturbances of the peripheral circulation. Recently there have been occasional spells of bradycardia, with the heart rate ranging between 30 and 39 beats per minute.

The first electrocardiogram made in 1916 showed complete atrioventricular block with an atrial rate of 120 beats per minute and a ventricular rate of 55 (fig. 4). This record was made at the time she was taking thyroid in moderate dosage because of an erroneous diagnosis of myxedema. This diagnosis had been made hastily on the basis of the slow heart rate alone, since the heart rate did not change normally in response to exercise. The patient was thyrotoxic at that time. The electrocardiogram cleared up the mystery. There has been no change in the electrocardiogram since the first record, except for some slowing of both atrial and ventricular rates. When examined, the heart has always shown good sounds and forceful action, with a moderate (grade 3) systolic murmur over the entire precordium, loudest at the left of the mid and lower sternum. Fluoroscopic examination has shown slight enlargement of the heart that has not changed over the years.

On Dec. 1, 1950, on physical examination, the patient looked well. Her weight was 153 lb. (69.4 kg.). The pulse rate was regular at 54 beats per minute. The blood pressure was 175 mm. Hg systolic and 90 mm. Hg diastolic. The feet and ankles were thick but not edematous. The heart showed good sounds; there was a grade 2 precordial systolic murmur. Fluoroscopic examination showed a slightly enlarged heart, as before. The lungs were clear. In an orthodiagram the transverse diameter of the heart measured 11.3 cm. and the internal diameter of the thorax measured 20.8 cm. The shadow of the aorta was slightly dense. An electrocardiogram showed complete atrioventricular block, with an atrial rate of 85 and a ventricular rate of 55 beats per minute and normal T waves throughout the classical limb leads, the unipolar leads, and the six precordial leads (fig. 5).

In June, 1952, Miss C. A. was observed to be in excellent health, 35½ years after her complete heart block was first discovered.

COMMENT

Complete atrioventricular block is an easily recognized cardiac arrhythmia and one well known to clinicians; however, it is relatively rare, as was indicated in an electrocardiographic series of 10,000 cardiac patients studied at the Massachusetts General Hospital (1916 to 1930).[1] Atrioventricular block was present in 641 patients (6.4%). The block was complete in only 79 (0.79% of the total series or 12% of the 641 patients). In most instances the etiological basis is not difficult to determine, coronary artery disease and rheumatic disease accounting for the great majority of cases. Unknown factors apparently are responsible for a few. This type of disturbance of rhythm is only rarely discovered at the time of its origin, and its discovery usually occurs accidentally in the course of routine health examinations or during the study of some cardiac or noncardiac illness. It is known that atrioventricular block, even of highest grade, is compatible with good health and full activity for many years. This is particularly true in the case of patients with congenital heart block who survive early childhood, and it is highly probable that the patient in case 1 falls in this category. On the other hand, it is recognized that heart block is usually more serious in older persons, in whom the disabling symptoms of the Morgagni-Adams-Stokes syndrome are likelier to occur.

In the two cases reported here certain factors stand out prominently. The slow rhythm was present in each patient for over 35 years. The defect in no way interfered with the patients having active, full lives. The etiology in each case remains unknown, unless diphtheritic infection in childhood in the second patient is presumed to be the cause. Following this assumption, the block must be assumed to have persisted for over 56 years without causing disabling symptoms. Similar reasoning may be applied in the case of the first patient. Here a slow pulse rate was detected during early childhood, but acute infectious diseases had been conspicuously absent. Apparently, in patients in whom complete heart block is not definitely related to diffuse myocardial injury and in whom attacks of syncope have not been a factor, the disease per se may persist for a much longer time than has been hitherto suspected, producing little or no disability.

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[1] White, P. D., and Sprague, H. B.: Electrocardiography of 10,000 Patients at the Massachusetts General Hospital from 1914 to 1931, Kasan (U. S. S. R.) M. J. 27:381, 1931.

SUMMARY

Two cases are reported of complete atrioventricular block lasting for 35 years or more in women in their late fifties, who continued in good health, free of any important symptoms, during that period. One patient died suddenly last year, and the pathological diagnosis of her condition is reported. The other patient continues to be well. These two cases illustrate the fact that such a disturbance of rhythm is compatible with long life and good health.

614 Doctors Bldg. (Dr. Benjamin).

DUPLICATION OF THE STOMACH

REPORT OF THE FIFTH CASE RECORDED

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Ladd and Gross [1] defined duplications of the alimentary tract as ". . . spherical or elongated hollow structures, which possess a coat of smooth muscle, which are lined by a mucous membrane, and which are intimately attached to some portion of the alimentary tube." The most comprehensive study of these lesions thus far published was made by those authors.

These cysts have been given different names, such as "enterogenous cysts," "enteric cysts," "enterocystomas," "inclusion cysts," "ileum duplex," "giant diverticula," and "gastrogenous cysts." None of these, however, is satisfactory enough to be used as a common designation for all these similar abnormalities of common embryologic derivation or developmental aberrations, regardless of location. The term, "duplications of the alimentary tract," preferred by Ladd and Gross, seems to be the most satisfactory.

Lesions of this type have been encountered in all levels of the digestive tract, from the base of the tongue to the anus. Almost every level is represented by one or more cases in the series of 18 collected by Farber [2] at the Boston Children's Hospital. As reported in the literature, the commonest site of occurrence is the small intestine. These duplications are found in relation to the terminal portion of the ileum more commonly than in any other part of the gastrointestinal tract.

CASES DESCRIBED IN RECENT LITERATURE

The first recorded case of duplication of the alimentary tract situated along the stomach was that of Ladd and Gross in 1940.[3] The second recorded case was one in which an enterocystoma of the stomach occurred in a 6-month-old child, and was reported by Ferraro in 1942.[4] The third case, in which the lesion was called "reduplication of the stomach," was reported by McCutchen in 1949.[5] The anomaly occurred in a man 27 years old. In 1951, Høyer and Andresen [6] reported an anomaly of the stomach that never had been observed before: a communicating duct between the cardiac region of the stomach and the duodenal bulb. Since this duct had two muscular coats and its lumen was lined with gastric mucosa, it constituted a hollow structure that could be considered a duplication of the stomach. The case we are presenting is, as far as we know, the fifth recorded case of gastric duplication.

PATHOLOGICAL ASPECTS

Adequate knowledge of the pathological aspects of these interesting lesions is essential to the surgeon who is called on for operative treatment. Duplications of the alimentary tract are replicas of, and are intimately attached to, some segment of the digestive tract. They share with this particular segment, at the area to which it is contiguous, the same smooth-muscle coat. At the free surface of such a process smooth muscle also can be found in its wall. The lining of these hollow structures is formed of mucosa or epithelium, similar to that of some portion of the stomach, small bowel, or colon. The duplication is not, however, necessarily a perfect replica of that viscus to which it is contiguous. A duplication of the stomach, for instance, can have a lining similar to intestinal mucosa and vice versa. A cyst of the rectum may have gastric epithelial lining, and so forth. Variations in the epithelial lining can be caused by pressure from entrapped, clear, colorless, mucoid fluid, secreted by the membrane of the cyst. This pressure may become so great as to cause necrosis and sloughing of the epithelial lining, which would add blood to the fluid. Any communication between the cyst and the digestive tract, which is not usual, naturally would permit the passage of digestive contents into the cyst.

As was particularly well evidenced in the case to be presented, it is of considerable practical surgical importance to differentiate (1) duplications of the alimentary tract and (2) lymphatic mesenteric cysts. Cysts can be shelled out easily from the mesentery, without disturbing the neighboring segment of the gastrointestinal tract. When enterogenous cysts and duplications of the alimentary tract are encountered, however, there is, in general, no plane of cleavage into which the surgeon can dissect, in order to peel away the cyst from the adjacent viscus. Any resolute attempt at such a maneuver would

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From the Surgical Clinics of Dr. Iseu de Almeida e Silva, Hospital Geral da Santa Casa da Misericordia.

[1] Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1941.

[2] Farber, S., quoted by Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1941.

[3] Ladd, W. E., and Gross, R. E.: Surgical Treatment of Duplications of Alimentary Tract: Enterogenous Cysts, Enteric Cysts, or Ileum Duplex, Surg., Gynec. & Obst. **70**: 295-307 (Feb. [No. 2A]) 1940.

[4] Ferraro, F. P., quoted by McCutchen, G. T.: Reduplication of Stomach: Report of Case, Ann. Surg. **129**: 826-831 (June) 1949.

[5] McCutchen, G. T.: Reduplication of Stomach: Report of Case, Ann. Surg. **129**: 826-831 (June) 1949.

[6] Høyer, A., and Andresen, I.: Cardio-Duodenal Duct; Anomaly of Stomach Previously Not Observed, J. Oslo City Hospitals **1**: 225-230 (Oct.) 1951.

result in cutting into and opening either the duplication or the contiguous viscus.

Ladd and Gross [1] presented a particularly illustrative photomicrograph of the wall of a duplication attached to that of its adjacent sister viscus. Between these two walls were shown the intermixed common smooth muscle coats, without any plane of cleavage between them.

The size of the duplications varies according to location. The duplication reported by Ladd and Gross was characterized by colonic mucous membrane, and was situated at the base of the tongue; it was 1 cm. in diameter. Cysts situated along the duodenum or rectal wall may be encountered when they are still small, because the symptoms call attention to them early. Those situated along the stomach may attain a size equal to that of the stomach itself or even larger, as in our case. Those which occur along the small bowel may be very long, such as the one 38 cm. long (case 15 of Ladd and Gross [1]), but are not very wide. Those that develop along the sigmoid colon may be very large and yet cause relatively minor symptoms. In Weber and Dixon's [7] unique case the duplication involved the entire large intestine.

SYMPTOMS AND DIAGNOSIS

The symptoms caused by duplications of the alimentary tract arise from (1) obstruction, which produces colicky pain, visible peristalsis, and vomiting; (2) distention of the hollow structure, engendering a continuous sensation of fullness, made worse by the ingestion of food or actually producing pain; or (3) vascular obstruction, producing segmental infarcted areas along the alimentary tract, with consequent hemorrhage. When such duplications become large enough, they are palpable and give the examiner the impression of a rounded, smooth, fairly renitent, cystic mass, which usually is movable and nontender when not too tense.

Roentgenologic examination with contrast medium can be of considerable aid in the differential diagnosis of these structures. Roentgenograms of course cannot disclose the histological nature of the mass, but they may permit the conclusion that the mass is intramural and extramucosal. The latter diagnosis was made by Schatzki [8] when he examined the roentgenograms in our case made by one of us (N. C.).

TREATMENT

In view of the pathological data previously mentioned regarding the absence of a plane of cleavage between the involved viscus and its duplication, the surgical treatment of choice in nearly all cases is radical, entailing removal of the duplication and resection of its adjacent viscus, followed by immediate reestablishment of the continuity of the alimentary tube. Certain circumstances, however, depending on the size and location of the duplications, not to speak of the precarious condition of some patients, make this procedure too hazardous and out of proportion to the benignity of the lesions. This is especially true of lesions in the upper part of the gastrointestinal tract. Radical removal of the duplication concomitantly with resection of the adjacent viscus is feasible when the lesion is in the small intestine (which is fortunately the commonest site) and when it is in the colon. In the case of gastric duplication reported by Ladd and Gross [3] and in our own, radical resection would have entailed total gastrectomy followed by esophagojejunostomy. This would have been too formidable an operation.

Under such circumstances, other forms of surgical treatment can be carried out, such as opening and emptying of the cyst, followed by marsupialization, with or without partial excision. The cavity can then be packed tightly with gauze, and injected with a sclerosing or necrotizing solution to destroy the epithelial mucous membrane that lines the cavity.

Moreover, internal drainage such as is frequently applied to cysts of the pancreas could be established. This technique was used successfully by Gardner and Hart,[9] who established a communication between an enterogenous duodenal cyst and the contiguous duodenum. In cysts of larger size there would be the possibility of failure from inadequate drainage. With adequate knowledge of the pathological aspects of these structures, and in view of the supportive and other forms of therapy now available, the operative treatment of these lesions can be carried out with a low mortality rate and good long-range results.

REPORT OF CASE

A white woman, 33 years old, was admitted to our service on Jan. 5, 1951, where she was examined by one of us (V. B. C.). Six months prior to her admission, one morning before she had eaten, she had complained of severe epigastric pain. Subsequent ingestion of food had brought on repeated vomiting. Episodes of vomiting had occurred very frequently for the next 30 days. Her physician at that time had instituted parenteral alimentation. During the same period the patient had difficulty in bowel movements, which were accomplished only with the aid of enemas. Once there had been no bowel movement for a week, in spite of the use of laxatives. On one occasion during the month in question the patient had been unable to urinate for 24 hours. Catheterization finally relieved her of considerable distention of the urinary bladder. During the next 30 days she had felt much relieved, while taking medication prescribed by an attending physician.

The patient then noticed a small, nontender epigastric mass, which she could mobilize freely. During the next four months this mass gradually increased in size, until it finally occupied the whole left upper abdominal quadrant, became painful, and was no longer movable. The vomiting recurred, but was not so frequent as previously; small, frequent, oral feedings were possible. Once she vomited some black fluid-like material, which simulated coffee grounds. At the time of her admission, in addition to severe, continuous pain in the left upper abdominal quadrant, necessitating the use of opiates for relief, the patient complained of a dull ache in the left lumbar region.

As to pertinent data antedating the onset of the foregoing distress, the patient remarked on the occurrence of cancer in three aunts. She also said that for eight years prior to the time we saw her she had had a lesion on her face, characterized by periods of recrudescence and regression, just before and during menstrual periods, respectively. About four years prior to her admission, the patient was said to have had a gastric ulcer. She said she had vomited blood, but had been considered cured after following a medical regimen. Unfortunately the report of the roentgenologic examination of the stomach made at that time was not available. The patient also said that about two years previous to her visit she had had stones in the urinary bladder.

Palpation disclosed a large, rounded, apparently smooth bulge in the whole left upper abdominal quadrant. This bulging discretely accompanied the respiratory excursions and pulsated isochronically with the radial pulse. There was evidence of beginning venous collateral circulation over the abdominal wall.

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[7] Weber, H. M., and Dixon, C. F.: Duplication of Entire Large Intestine (Colon Duplex): Report of Case, Am. J. Roentgenol. 55:319-324 (March) 1946.

[8] Schatzki, R.: Personal communication to the authors.

[9] Gardner, C. E., Jr., and Hart, D.: Enterogenous Cysts of Duodenum: Report of Case and Review of Literature, J. A. M. A. 104:1809-1812 (May 18) 1935.

This mass was globular, with definite delimitations and a smooth surface; it was of hardened consistency, only slightly movable, and very painful. The pulsation evidently was transmitted from the underlying aorta; there was *no* expansion as in aneurysms and no abnormal sounds. Percussion showed dullness over the whole surface of the large mass, except for a narrow, transverse, elongated strip of tympanicity, apparently referable to the transverse colon, between the area of dullness over the liver and the lower portion of the mass.

Lupus erythematosus involved the skin of the face, over the sternum, and on the anterior aspects of both forearms. All other findings were normal except for chronic cervicitis found during routine pelvic examination.

Results of urinalysis and a complete hemogram were normal. A few ova of Trichuris trichiura were found in the stools. Results of flocculation tests for syphilis were reported as doubtful. Values for blood urea and sugar were within normal limits. Roentgenograms of the thorax disclosed nothing abnormal. Cholecystograms were considered to indicate a normal gallbladder. Flat roentgenograms of the abdomen, however, showed a large mass occupying the left upper abdominal quadrant. The left kidney did not appear in these roentgenograms. The right kidney appeared to be normal. Excretory urograms showed both kidneys to be functioning normally. The left kidney was slightly enlarged, and its pelvis was not distended by the contrast medium. A roentgenogram made with the aid of a barium enema (fig. 1*A*) showed the splenic flexure to be pushed downward by a large extrinsic mass of increased density, which occupied the left hypochondrium. A blastoma of the body or tail of the pancreas was suspected. Roentgenologic examination of the stomach (fig. 1*B*) revealed a voluminous extragastric tumor mass that pushed this viscus forward; the impression was that a large pancreatic cyst was responsible. Aureomycin was administered for 72 hours before the operation.

On Jan. 17, with the patient under anesthesia with nitrous oxide, ether and oxygen, exploratory laparotomy was performed by one of us (J. C. B.), through a primary upper left rectus, muscle-splitting incision. There were no adhesions to the anterior parietal peritoneum. The stomach protruded anteriorly and the transverse colon was pushed downward by a mass situated behind the stomach. This mass was made accessible by sectioning of the gastrocolic omentum. A voluminous thick-walled cystic mass was ound, firmly attached to the posterior surface and greater curvare of the stomach from the prepyloric region to the fundus. s mass was adherent to the anterior surface of the pancreas nd duodenum and splenic pedicle and to the upper surface of the transverse mesocolon. The mass was freed from the body and head of the pancreas and duodenum and also from the mesocolon with relative ease. Attempts at freeing this mass from the tail of the pancreas and splenic pedicle caused severe hemorrhage from large veins; this was finally controlled after splenectomy.

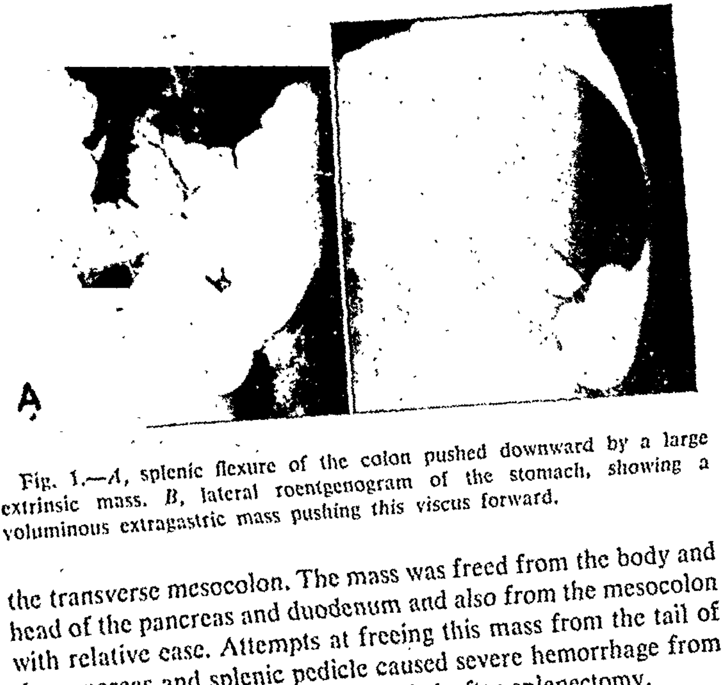

Fig. 1.—*A*, splenic flexure of the colon pushed downward by a large extrinsic mass. *B*, lateral roentgenogram of the stomach, showing a voluminous extragastric mass pushing this viscus forward.

The spleen was slightly enlarged. The cystic cavity was then opened anteriorly, and more than 2,000 cc. of clear, light yellow fluid of neutral reaction was aspirated. No communication with the stomach could be detected. At this point the walls of the cyst were completely free from neighboring structures except for the attachment to the posterior surface and greater curvature of the stomach, from which it could not be dissected free without cutting into it. This was avoided.

Fragments from different portions of the anterior wall of the cyst were given to the pathologist (Dr. E. T. Torres) for diagnosis by means of frozen section. Moments after the cavity of the cyst had been emptied, the walls contracted and became considerably thicker, measuring in certain portions about 12 mm. The cavity of the cyst then could accommodate only about 20 cc. of saline solution. The pathologist, Dr. Torres, found much fibrous tissue, intermixed with smooth-muscle fibers crossing in different directions.

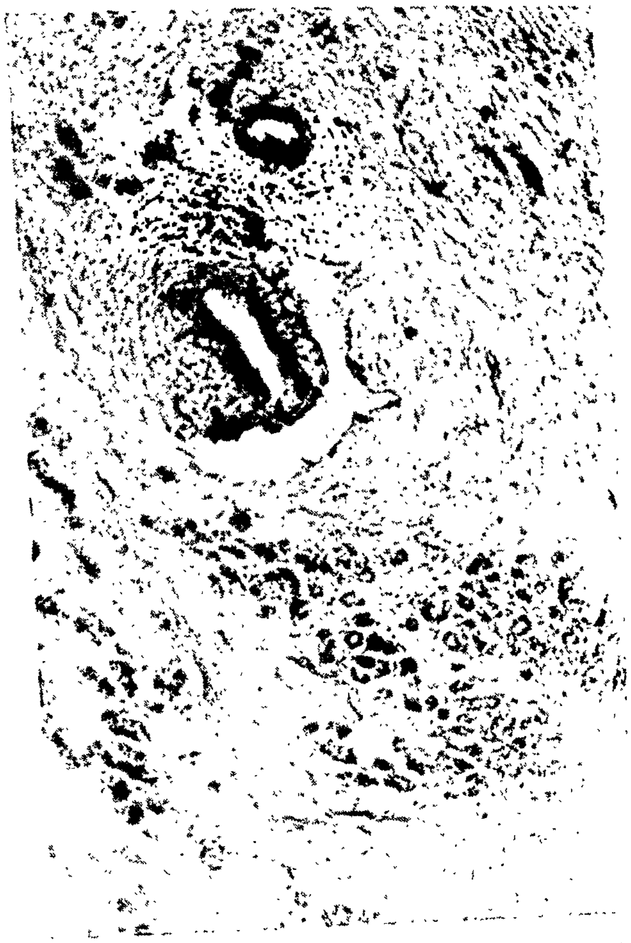

Fig. 2.—Fixed sections of fragments taken from a portion of the wall of the cystic mass, showing smooth-muscle fibers, fibrous connective tissue, and epithelial formations of small, atrophic, deep-staining epithelial cells.

An epithelial lining was searched for, but was not found at this time. Nevertheless, we realized that we were dealing with an exceedingly unusual lesion, namely, duplication of the stomach, and that to remove this lesion completely, total gastrectomy and esophagojejunostomy probably would be in order. We proceeded instead with a much less hazardous procedure, which consisted of partial hemispherical excision of the cyst, followed by its marsupialization to the anterior abdominal wall, after the lining had been painted with a 10% solution of formaldehyde. The cavity was then packed tightly with a long strip of iodoform gauze and the remaining portions of the incision were sutured with interrupted stitches of no. 0 and 1 chromic catgut in the peritoneum and fascia, respectively, with reinforcing cotton sutures in the fascia.

The pathologist reported that the fluid contents of the cys exhibited a positive (grade 4) Rivalta reaction, with a pH of 7 Microscopic examination of the fluid disclosed rare cells an numerous lymphocytes and erythrocytes. Multiple fixed section were made from fragments taken from various portions of th cyst. One of us (C. O.) finally detected epithelial formations (fig. 2), in addition to the involuntary muscle fibers and fibrous connective tissue previously detected in the frozen sections. The greater portion of the lining of the cyst had been destroyed by the great pressure from the retained fluid. The glands found were formed of small, atrophic, deep-staining epithelial cells. The nature of the cyst was thus more completely authenticated.

During the immediate 48 hours after the operation a state of shock persisted in spite of repeated use of analeptic agents and administration of blood. The extremities were cold and the systolic blood pressure oscillated between 85 and 95 mm. Hg. At the end of this period the patient improved considerably. She was up and about on the 10th postoperative day. The immediate and persistent state of shock was interpreted as being caused not so much by the operation as by absorption of the formaldehyde that was used to destroy the epithelial lining of the cyst. This provoked an "alarm syndrome" such as that described by Selye.[10]

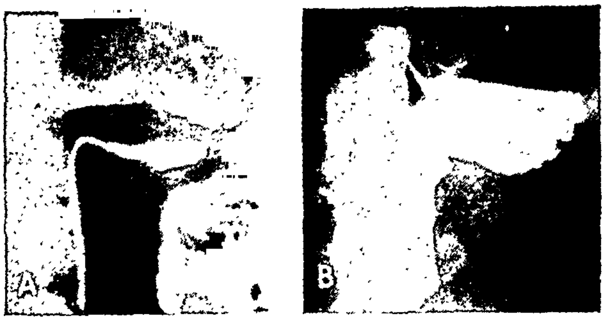

Fig. 3.—*A*, deep, narrow sinus tract at the site of marsupialization into which contrast medium was injected through a thin Nélaton catheter. *B*, the stomach, three months postoperatively, showing some deformity. The stomach, however, emptied normally.

Since the patient lived far from the hospital and needed careful daily dressings, she was purposely kept in the hospital longer than would otherwise have been necessary. The packing was removed two days postoperatively. The patient was dismissed from the hospital seven and a half weeks postoperatively. She was then taking regular meals and was feeling well, although she still had a long, narrow sinus at the site of the marsupialization (fig. 3*A*). This finally closed 30 days later. About three months later roentgenologic examination of the stomach (fig. 3*B*) showed some deformity but normal emptying. She had gained considerably in weight. At the time of this writing, 14 months postoperatively, the patient was feeling very well and complained only of occasional slight epigastric pain after a heavy meal.

COMMENT AND SUMMARY

The authors describe the fifth case of duplication of the stomach recorded in the literature. The patient was a 33-year-old white woman who complained of severe epigastric pain and vomiting and who had a large tender mass occupying the whole left upper abdominal quadrant. Roentgenologic examination of the stomach showed a voluminous extrinsic mass dislocating this viscus anteriorly. A large duplication of the stomach containing more than 2,000 cc. of fluid was partially excised and marsupialized to the anterior abdominal wall. Sections from the wall of the cyst showed much fibrous tissue, smooth-muscle fibers, and an atrophic, partially destroyed epithelial lining. The patient, except for minor symptoms after heavy meals, was well 14 months postoperatively.

Rua Debret 23 (Dr. Barbosa).

[10] Selye, H.: The Physiology and Pathology of Exposure to Stress: A Treatise Based on the Concepts of the General-Adaptation-Syndrome and the Diseases of Adaptation, Montreal, Canada, Acta, Inc., 1950.

URETERAL COMPRESSION DUE TO METASTASIS FROM CARCINOMA OF SIGMOID

George R. Krause, M.D.
and
Mortimer Lubert, M.D., Cleveland

Metastasis to retroperitoneal lymph nodes from carcinoma of the sigmoid colon or rectum may compress and obstruct the ureters, especially the left one. Two recent articles [1] mentioned this, but, unfortunately, there was not enough emphasis in either. One of the reports included cases with other sites of primary neoplasm; the other laid greatest stress on changes in position of the ureters that follow reperitonealization of the pelvic floor during an abdominoperineal resection of the rectum. Recent experiences have led us to believe that ureteral compression by metastasis from carcinoma of the sigmoid colon or rectum is of sufficient importance to deserve undivided attention.

During the past five years we have seen five patients who have had at least one ureter compressed by lymph nodes containing tumor that was metastatic from a carcinoma of the distal colon. The primary tumor had been removed from four of these patients, but they had returned to their physicians with various complaints. All described vague abdominal distress and distention. Some had actual pain; others mentioned troublesome constipation. None of the difficulties mentioned were referred to the urinary tract.

The initial investigation requested by the clinician was usually confined to a roentgen study of the site of operation, anticipating that a narrowing of the anastomosis or a local recurrence might be found to explain the symptoms. In no instance did this study answer the problem, even on repeated attempts. Studies of the upper gastrointestinal tract and the small intestine were often requested, but they did not reveal significant findings. After an intravenous urogram finally provided the answer in case 1, the other cases were evaluated correctly with less difficulty; this was done despite the fact that the clinician was frequently reluctant to consider study of the urinary tract as a possible solution of the problem. In the fifth case, routine investigation of the urinary tract prior to operation not only disclosed the presence of pressure on the left ureter but also enabled the surgeon to plan his procedure in advance and to accomplish his purpose more readily.

REPORT OF CASES

Case 1.—A 41-year-old white man was admitted to Mount Sinai Hospital on June 1, 1946, for resection of an adenocarcinoma that was about 2 in. (5.08 cm.) above the peritoneal reflection. Lymph nodes felt in the mesosigmoid were removed with a large section of the mesentery; two deeper nodes were also removed. All contained metastatic tumor. Three months later, because of abdominal cramps and distension, a barium study of the colon was done. This did not reveal evidence of significant narrowing at the anastomosis, dilatation of the colon, or stasis, even when repeated three weeks later.

From the Department of Radiology, Mount Sinai Hospital.

[1] Marshall, V. F., and Schnittman, M.: Iliac Lymphadenopathy as a Cause of Ureteral Obstruction, Surgery 23: 542-549, 1948. Spillane, R. J.; Kaiser, T. F., and Prather, G. C.: Medial Deviation of the Ureters Complicating Carcinoma of the Rectum and Sigmoid, and Proctosigmoidectomy, Surg., Gynec. & Obst. 93: 273-282, 1951.

Six months after surgery, the patient complained of constipation and severe, constant pain in the left lower quadrant. After a third normal barium enema, the medical consultant requested an intravenous urogram, which revealed no excretion of the dye by the left kidney. Ureteral catheterization (fig. 1) then demonstrated three points of obstruction in the left ureter.

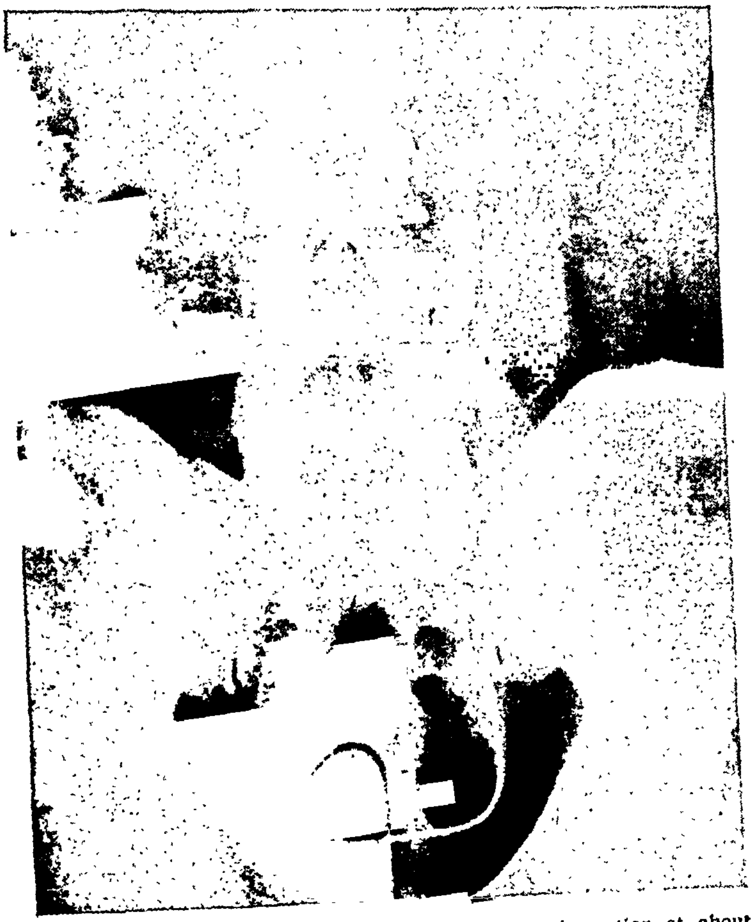

Fig. 1 (case 1).—The ureteral catheter met an obstruction at about 10 cm. There are two more distinct, narrowed areas higher up (arrows). The small amount of dye in the kidney pelvis reveals hydronephrosis. At exploration, the ureter was encased in tumor tissue at the points of constriction.

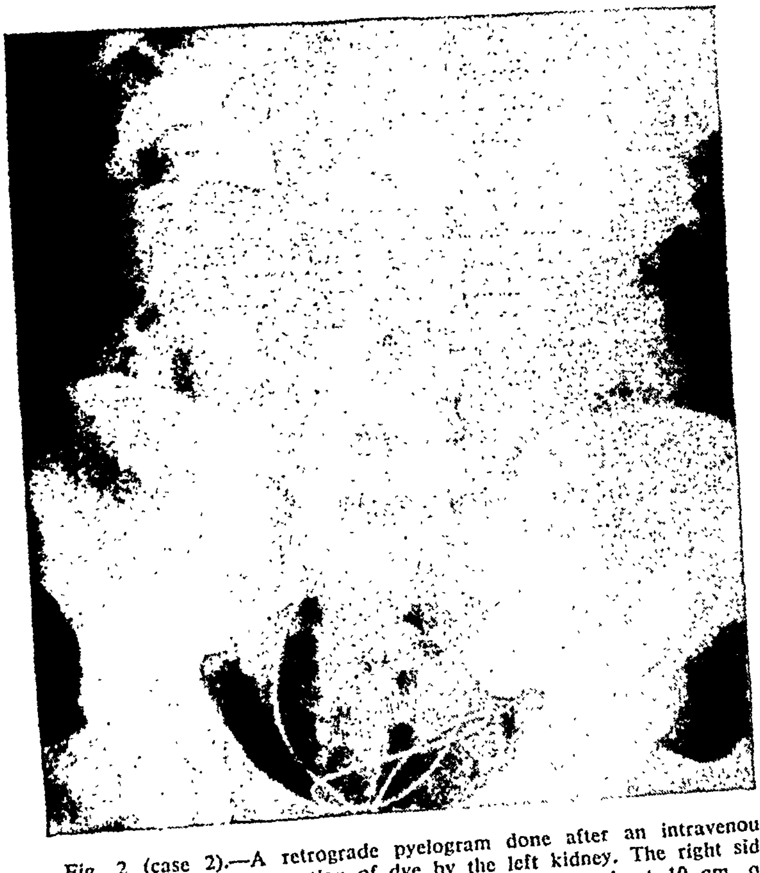

Fig. 2 (case 2).—A retrograde pyelogram done after an intravenous urogram revealed no excretion of dye by the left kidney. The right side was normal. The catheter was completely blocked at about 10 cm. on the left. Dye was extravasated when the catheter perforated the ureter.

Because of the severe pain and demonstrable infection, a left nephrectomy and a ureterectomy were done. The ureter was encased in tumor tissue at points corresponding to the constrictions seen in figure 1. The patient died in another hospital, 10 months after the original colon resection.

Case 2.—A 43-year-old white man was admitted to Mount Sinai Hospital on April 23, 1947, for resection of a carcinoma of the sigmoid, which was 1 in. (2.54 cm.) above the peritoneal reflection. A number of small nodes were present in the mesosigmoid; microscopic examination did not reveal evidence of tumor. Fourteen months later he complained of persistent, but diffuse, abdominal distress. Barium studies of the colon and the upper gastrointestinal tract did not reveal any abnormality. Because of our experience with case 1, we requested that an intravenous urogram be done; this revealed that no dye was excreted by the left kidney. Subsequent ureteral catheterization revealed a partial block of the left ureter at 10 cm., with dilatation of the urinary tract above it. Three months later (fig. 2) a catheter could not be passed up the left ureter beyond that point. Two months later (now 19 months after the original surgery) a hard mass could be palpated deep in the right lower quadrant. An intravenous urogram at this time revealed hydronephrosis and hydroureter on the right, with no dye excretion on the left. The patient died of uremia soon afterwards, 21 months after the colon resection.

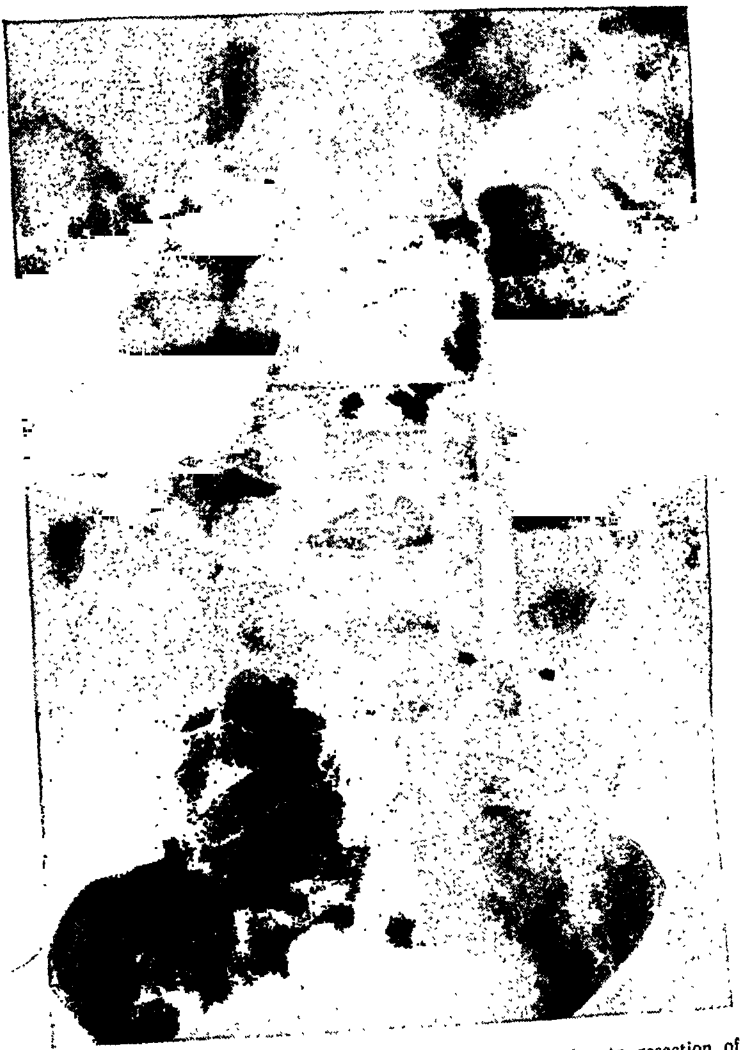

Fig. 3 (case 5).—An intravenous urogram done prior to resection of the sigmoid reveals a left hydronephrosis and hydroureter. The ureter is dilated down to the sacrum (arrows). The site of obstruction was confirmed by ureteral catheterization. At surgery it was found to be caused by metastatic tumor compressing the ureter.

Case 3.—A 52-year-old white man was admitted to Mount Sinai Hospital on Jan. 8, 1948, for resection of a small, constricting carcinoma of the sigmoid, 3 in. (7.62 cm.) above the peritoneal reflection. Nodes could be palpated deep in the mesosigmoid. Four of those removed were examined microscopically and found to contain tumor.

The patient remained in fairly good health for about 18 months but then complained of pain in the left lower quadrant, which was aggravated by standing and more severe after a bowel movement. A barium enema did not reveal evidence of narrowing at the anastomosis. An intravenous urogram, done at our request, showed that no dye was excreted by the left kidney. The ureteral catheter encountered a complete block at 10 cm. Later, repeated barium studies were again reported as normal.

Twenty-six months after the original resection, he was reoperated for intestinal obstruction. Exploration revealed many adhesions and tumor implants on the peritoneum. There were tumor masses at the root of the mesentery; the left ureter was encased in such a mass. The obstruction, largely due to the adhesions, was relieved, but the patient died at home, 30 months after the original resection.

CASE 4.—A 50-year-old white woman was admitted to Mount Sinai Hospital on Jan. 11, 1949, for resection of a carcinoma of the sigmoid 3 in. (7.62 cm.) above the peritoneal reflection. The mesosigmoid contained 17 lymph nodes; tumor was demonstrated in two of them on microscopic study. (Clearing techniques on the mesosigmoid were not done on any of these specimens.) In the ensuing months she complained only of fatigue and required mild laxatives. Later, because of pain and vague abdominal complaints, three barium studies of the colon and the small intestine were done; none revealed significant abnormalities. We suggested that an intravenous urogram be done, but none was ordered.

Thirteen months after surgery, she became lethargic and vomited repeatedly. After catheterization revealed an empty bladder, ureteral catheters were inserted. A partial, but passable block was encountered on the left at 7 cm.; a complete block was found on the right at the same level. The blood nonprotein nitrogen was 174 mg. per 100 cc. An indwelling catheter on the left side resulted in a temporary reduction in nitrogen retention, but she died of uremia 16 months after the sigmoid resection.

CASE 5.—A 62-year-old white woman was admitted to Mount Sinai Hospital on Oct. 13, 1951, for a resection of a carcinoma of the sigmoid. She complained of increasing constipation and vague abdominal distress; no symptoms were referable to the urinary tract. A routine intravenous urogram was done prior to surgery (fig. 3). The upper urinary tract was dilated; the point of obstruction appeared to be in the lower ureter, near the pelvic brim. A catheter passed beyond the point of obstruction and drained considerable pus and urine. The carcinoma was resected with the catheter in place. The left ureter, found to be compressed by a tumor mass, was dissected free, and as much as possible of the metastatic tumor in this region was removed. The immediate postoperative course was uneventful.

COMMENT

Studies of the lymphatic spread of carcinomas of the distal colon [2] have been based largely on resected specimens. As a result, there has been a concentration of interest on the nodes in the mesocolon itself, with little, if any, stress laid on the relationship of the deeper nodes to the ureter. This is in contrast to similar studies of carcinoma of the cervix, in which the intimate relationship between the lymphatic drainage of the cervix and the ureters is emphasized and widely known. Indeed, because such spread is the usual occurrence in the natural (untreated) history of carcinoma of the cervix, operations have been devised to remove these nodes as part of the primary treatment.

Since the base of the mesocolon lies in intimate relation with the lower part of the left ureter, enlarged lymph nodes in the root of this mesentery may encase or compress the ureter. Further metastasis of tumor to periureteric nodes may involve the ureter at a higher level. Spread to the other side is also possible (cases 2 and 4).

Compression of the ureter occurs gradually, because the tumor growth in these lymph nodes is slow, so that the absence of urologic symptoms is not surprising. Because at first there is usually obstruction of the left ureter only, evidence of poor kidney function is not present. Unless infection supervenes, there will be no specific clinical or laboratory evidence or urologic difficulty. The symptoms do not point to the urinary tract, and, unless the pathogenesis of the syndrome is understood, the vague complaints will not suggest the need for intravenous urograms, and unnecessary delay will result.

Because the concept of cancer surgery has become progressively more radical, important implications may be drawn from our experience. Radical surgery for carcinoma of the sigmoid colon may one day include the periureteric area on the left, somewhat in the manner of Meigs' operation for carcinoma of the cervix.

Indeed, it may be found advisable at a later time to consider removal of the left kidney and ureter a part of the radical surgical approach to this lesion. (This is not possible in surgery for carcinoma of the cervix, since lymph node spread in that lesion may be bilateral at an early stage.) Such radical extension of the surgical treatment cannot be recommended as a prophylactic procedure until statistical study has determined the incidence of spread from carcinoma of the sigmoid to the nodes along the left ureter; however, such radical surgery might well be the only hope for a patient resembling case 5 or for one in whom there is definite evidence of such lymph node metastasis at the time of surgery, even if it is not compressing the ureter at the time.

CONCLUSIONS

1. Intravenous urography is an important procedure, preoperatively and postoperatively, in the management of carcinoma of the sigmoid colon.
2. A more radical surgical approach to carcinoma of the sigmoid colon, either as a primary or palliative procedure, may prove to be necessary.

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Energy Available in Alcohol.—Alcohol has an energy food value of seven calories per gram. Approximately 90 per cent of ingested alcohol is oxidized to carbon dioxide and water, the remaining 10 per cent accounting for the smell of alcohol on the breath, and alcohol in the urine, perspiration and saliva. The body can oxidize seven to 10 grams of alcohol per hour, which corresponds roughly to 1 oz. of whiskey or 8 oz. of beer. Thus, the maximum energy available in a 24-hour period from the oxidation of alcohol alone, is about 1600 calories.—Bell, R. G., M.D., Clinical Orientation to Alcoholism, *Industrial Medicine and Surgery, The Journal of Medicine in Industry*, June, 1952.

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[2] Gilchrist, R. K., and David, V. C.: Lymphatic Spread of Carcinoma of the Rectum, Ann. Surg. **108**: 621-642, 1938. Gilchrist, R. K., and David, V. C.: Consideration of Pathological Factors Influencing 5 Year Survival in Radical Resection of the Large Bowel and Rectum for Carcinoma, ibid. **126**: 421-438, 1947. Coller, F. A.; Kay, E. B., and MacIntyre, R. S.: Regional Lymphatic Metastasis of Carcinoma of the Rectum, Surgery **8**: 294-311, 1940. Coller, F. A.; Kay, E. B., and MacIntyre, R. S.: Regional Lymphatic Metastases of Carcinoma of the Colon, Ann. Surg. **114**: 56-67, 1941.

COUNCIL ON PHARMACY AND CHEMISTRY

REPORT TO THE COUNCIL

The Council has authorized publication of the following reports.

R. T. STORMONT, M.D., *Secretary.*

STATUS OF PRIMAQUINE

1. MASS THERAPY OF SUBCLINICAL VIVAX MALARIA WITH PRIMAQUINE

Alf S. Alving, M.D., Chicago
Major John Arnold (MC), Army of the United States
Major Donald H. Robinson (MC), United States Army

This report deals with the problem in mass therapy presented by the large number of cases of vivax malaria occurring in the United Nations forces in Korea since 1950.

Because malaria is not a single disease but instead is a complex of diseases all caused by a member of the genus plasmodium the unique properties of the members of the various species and strains of the organism must be considered in any given therapeutic problem.

On morphologic grounds, four distinct species of plasmodia are recognized. One species, Plasmodium ovale, is rare and of little clinical importance; two species, P. malariae and P. vivax, have many properties in common. For therapeutic purposes, they may be considered together. The remaining species, P. falciparum, has special properties and must be considered separately.

On physiologic grounds, each species is made up of several strains, each with idiosyncrasies that modify response to treatment. It is unfortunate that differences in strains can usually be classified only by geography and can be considered in therapy only in the most general way. This means that drugs chosen for mass therapy must be sufficiently effective to cover variations in responses of strains.

These general considerations constitute only part of the problem faced in evaluation of antimalarial drugs.[1a] The following discussion emphasizes those factors that are of special importance in planning a program for the eradication of malaria in troops prior to their return to the United States.

CHOICE OF TREATMENT IN RELATION TO THE TYPES OF MALARIA

P. Falciparum.—P. falciparum appears to be the most recently acquired malaria infection in man. This is reflected in the fact that P. falciparum causes the greatest mortality and has the simplest life cycle of all malarias in the human host.

This simplicity of life cycle has proved to be of great importance in therapy. Very shortly after the parasite is introduced into man by a mosquito bite it disappears from the blood and enters the tissues of the host (fig. 1). Sometime within the first week of infection the tissue forms give rise to the easily recognized blood stages which usually are the only forms capable of producing symptoms. After producing this first brood of blood forms, the tissue stages either disappear or become nonfunctional. Blood forms arise only once in the course of any given infection with P. falciparum. For this reason, total eradication of P. falciparum can be achieved by simple eradication of the blood stages.

In practice, eradication of blood forms is usually easy, because most of the common antimalarial drugs have an action directed chiefly against the blood forms (schizonts). Such drugs, acting solely against the blood forms, are called suppressive agents. Of the newer suppressive drugs, chloroquine (aralen®), amodiaquine (camoquin®) and chlorguanide (paludrine®) have largely displaced quinacrine (atabrine®) and quinine in military use.

P. Vivax.—In contrast to P. falciparum, infections with P. vivax are much better adapted to the human host. This improved adaptation of parasite to host has led to a lower mortality rate but a higher degree of persistence, which has been achieved by the development of another stage in the life cycle of the parasite. P. vivax begins, like P. falciparum, as a tissue infection which lasts approximately one week, at the end of which time the blood forms causing the primary attack are produced (fig. 2). From this point on an important difference between P. falciparum and P. vivax appears. In the latter infection, a persisting tissue stage develops which, by giving off broods of blood forms at intervals, accounts for the periodic relapses of vivax malaria.

All the suppressive drugs effective against the blood forms of P. falciparum are effective against the blood forms of P. vivax. Thus, drug therapy of the acute disease from either parasite is usually the same, but the long-term result of such therapy is quite different. In P. falciparum infections, adequate suppressive therapy eliminates the disease for all time; in P. vivax infections, suppressive therapy eliminates symptoms of the disease until the next relapse.

If suppressive agents are taken continuously and in sufficient amounts, the blood forms produced in P. vivax infections at intervals by the persisting tissue stages are destroyed soon after they are formed and the patient remains well. Because vivax malaria is a self-limiting disease, through the development of immunity or the exhaustion of the tissue stages, continuation of suppressive medication for several years after leaving a malarious area will result in a reduction of the relapse rate. In endemic areas, continuous suppression can be carried out successfully for years provided a break in drug administration does not occur. Such long-continued suppression requires continuous vigilance. This is difficult even in endemic areas but is usually impossible for individuals or organizations returning to nonendemic areas where the stimulus for continuous therapy is lacking.

RADICAL CURE OF VIVAX MALARIA

To meet the problem produced by the constant threat of relapse from vivax infections in persons leaving an endemic area for a nonendemic area, another class of antimalarial drugs is of value. This class of drugs acts by destroying the tissue stages of the disease and thereby prevents the development of the blood form responsible for the relapse. Since they completely eliminate all trace of the infection, these drugs are called curative as opposed to the suppressive drugs which destroy only blood forms. Unfortunately, the curative drugs now known have very little effect against the blood forms and must be used in conjunction with one of the suppressive drugs when blood forms of the disease are present.

Of the thousands of drugs studied for antimalarial action, only members of the 8-aminoquinoline family closely related to pamaquine have shown a significant amount of curative activity. A number of compounds of this family possess some degree of action against the tissue stages of malaria, but only one of these compounds has shown a high enough level of activity with a low enough level of toxicity to warrant its use on a large scale. This compound, 8-(4-amino-1-methylbutylamino)-6-methoxyquinoline, now known as primaquine, was synthesized at Columbia University during the World War II antimalarial program.[1] After the wartime program was terminated, primaquine received extensive animal studies at The Christ Hospital, Cincinnati,[2] clinical tests at Stateville Penitentiary and the Federal Penitentiary at Atlanta,[3] and actual field trials in Nicaragua.[4] Large scale toxicity studies

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Report of the Army Malaria Mission to the Far East Command during the Summer of 1951.

[1a] Shannon, J. A.: Evaluation of Antimalarial Drugs, chap. 10 in Evaluation of Chemotherapeutic Agents, C. M. MacLeod ed., New York, Columbia University Press, 1949, pp. 134-151. Singh, J.: Antimalarial Drugs, Indian J. Malariology 4: 185-188 (June) 1950.

[1] Elderfield, R. C., and others: Alkylaminoalkyl Derivatives of 8-Aminoquinoline, J. Am. Chem. Soc. 68: 1524-1529 (Aug.) 1946.

[2] Schmidt, L. H.: Unpublished data.

[3] Edgcomb, J. H., and others: Primaquine, S. N. 13,272, A New Curative Agent in Vivax Malaria: A Preliminary Report, J. Nat. Malaria Soc. 9: 285-292 (Dec.) 1950. Clayman, C. B.; Arnoid, J.; Hockwald, R. S.; Yount, E. H., Jr.; Edgcomb, J. H., and Alving, A. S.: Toxicity of Primaquine in Caucasians, J. A. M. A., this issue, p. 1563. Coatney, R. G., and others: Unpublished data on clinical studies of primaquine conducted at Atlanta *Penitentiary*.

[4] Thaeler, A. D., Jr.; Arnold, J., and Alving, A. S.: Field Studies of Primaquine in Nicaragua, presented at the Annual Meetings of the National Malaria Society, Chicago, Nov. 14-17, 1951.

at Fort Knox and Fort Benning jointly conducted by Army, United States Public Health Service, and civilian investigators later established the relative safety of administration of primaquine to troops on full active duty.[5]

MALARIA IN KOREA

The first large-scale problem of relapsing malaria in nonimmunes since World War II developed as a result of the hostilities in Korea in the summer and fall of 1950. Although malaria has been known to exist in Korea, the heavy infection of United Nations troops was largely unexpected. Previous experience by the 24th Corps on occupation duty in South Korea in 1947 revealed a variable amount of the disease among the civil population. Civilian surveys gave an incidence ranging between 0.97 and 33.94%.[6] The Japanese recognized malaria in their long experience on the peninsula but had not been impressed by the incidence. Certainly, when judged by the yardstick of experience gained by military forces in the Southwest Pacific, malaria in Korea did not seem to be a serious hazard.

Even so, the United Nations command used chloroquine (or chlorguanide) in the early days of fighting and effectively suppressed most of the infections present in the field. This treatment so obscured the disease that an evaluation of the size of the malaria problem was not possible until the troop rotation program was inaugurated in 1951, and large numbers of men were returned home and discontinued chloroquine suppression.

By virtue of Korea's temperate climate it would be expected that most Korean malaria would be vivax in type and would behave like the other temperate zone strains of vivax, such as the St. Elizabeth strain in the United States.[7] Since temperate zone strains of vivax malaria do not have morphologic characteristics that distinguish them from the tropical zone strains of vivax, a determination of the temperate zone character of malaria requires a study of the strain's natural history. In figure 3 the temperate zone vivax is compared with the tropical zone vivax. As can be seen in this figure, the chief difference between the two types appears in the relative length of the interval between the primary attack and the first relapse. The temperate zone vivax has a long latent interval of eight to nine months and seems adapted to the long winters of temperate zones during which malaria is not transmitted. This characteristic modifies the monthly incidence rates of the disease caused by the two forms of plasmodium vivax. In groups chiefly infected during the summer months with temperate zone vivax the incidence rates fall sharply in the winter months, whereas in groups infected with tropical zone vivax, even when transported to a temperate zone, the relapses will hold the attack rate relatively high at any season of the year.

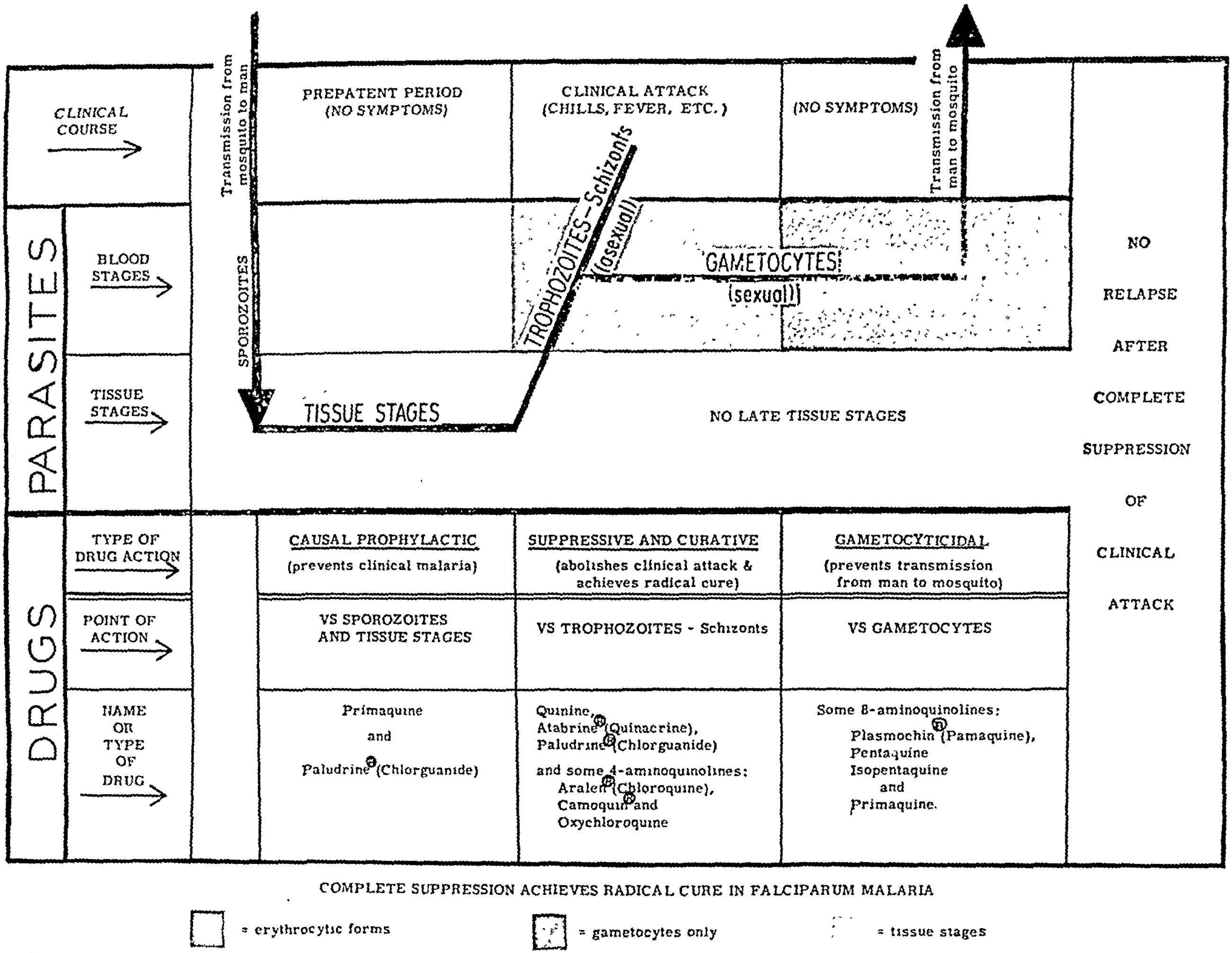
Transmission from mosquito to man
Transmission from man to mosquito
CLINICAL COURSE
PREPATENT PERIOD (NO SYMPTOMS)
CLINICAL ATTACK (CHILLS, FEVER, ETC.)
(NO SYMPTOMS)
PARASITES
BLOOD STAGES
TISSUE STAGES
SPOROZOITES
TROPHOZOITES–Schizonts (asexual)
GAMETOCYTES (sexual)
TISSUE STAGES
NO LATE TISSUE STAGES
NO RELAPSE AFTER COMPLETE SUPPRESSION OF CLINICAL ATTACK
DRUGS
TYPE OF DRUG ACTION
CAUSAL PROPHYLACTIC (prevents clinical malaria)
SUPPRESSIVE AND CURATIVE (abolishes clinical attack & achieves radical cure)
GAMETOCYTICIDAL (prevents transmission from man to mosquito)
POINT OF ACTION
VS SPOROZOITES AND TISSUE STAGES
VS TROPHOZOITES - Schizonts
VS GAMETOCYTES
NAME OR TYPE OF DRUG
Primaquine and Paludrine® (Chlorguanide)
Quinine, Atabrine® (Quinacrine), Paludrine® (Chlorguanide) and some 4-aminoquinolines: Aralen® (Chloroquine), Camoquin® and Oxychloroquine
Some 8-aminoquinolines: Plasmochin® (Pamaquine), Pentaquine Isopentaquine and Primaquine.
COMPLETE SUPPRESSION ACHIEVES RADICAL CURE IN FALCIPARUM MALARIA
= erythrocytic forms
= gametocytes only
= tissue stages

Fig. 1.—An outline of a disease mechanism which seems adequate to explain the sequence of events in falciparum malaria and the mode of action of the more important antimalarial drugs

The presence of temperate zone vivax was suggested by the experience in 1947 of the 24th Corps in Korea which reported a spontaneous drop in its attack rate after October of that year. The rate fell from a high of 50 cases per 1,000 per annum in summer and fall to 1 or 2 cases per 1,000 per annum for the rest of the year.[6] More recent experience with Korean malaria in returning American servicemen has confirmed the fact that

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[5] Toxicity studies at Fort Benning were conducted by Major Robinson and Scientist Director G. R. Coatney, U. S. Public Health Service, and at Fort Knox by Col. R. A. Radke, Capt. L. J. Polkskin, Major Arnold, (MC), Army of the United States and Dr. Alving.

[6] E.T.M.D. of the 24th Corps, 1947.

[7] Coatney, G. R., and Cooper, W. C.: Recrudescence and Relapse in Vivax Malaria, Proc. 4th International Cong. of Trop. Med. and Malaria, Vol. 1, Dept. of State, 1948, pp. 629-639.

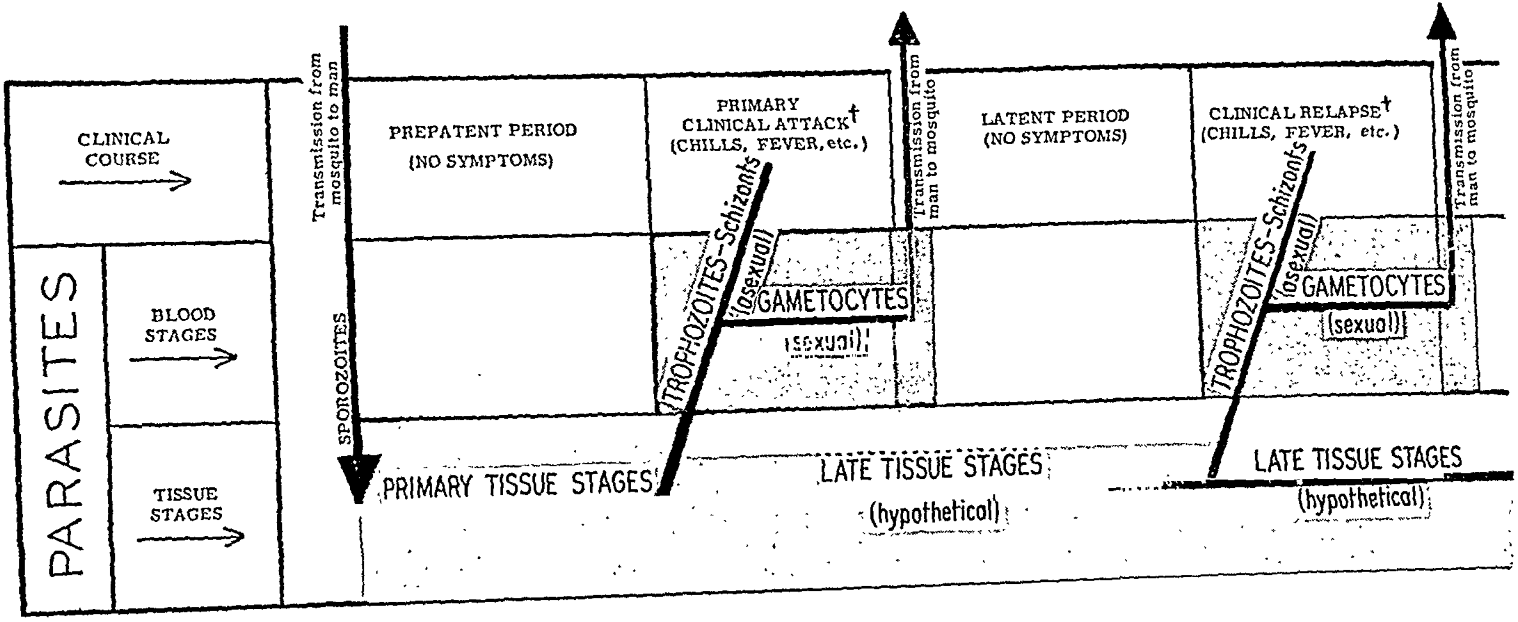
Korean malaria has a long latent interval after the first attack and thus closely resembles the American St. Elizabeth strain. Falciparum malaria is virtually unknown in Korea.

Recent Military Experience with Korean Malaria.—The characteristics of the Korean strain have been pointed out in detail because they explain why the 8th Army did not have an outbreak of malaria when it discontinued chloroquine suppression during the winter of 1950-1951. Without benefit of subsequent experience with Korean malaria, the absence of wintertime relapses then made widespread malaria infection in the Army seem unlikely. Nevertheless, the 8th Army returned to chloroquine suppression in the spring of 1951, but men evacuated to the United States from the 8th Army did not return to chloroquine. The returnees, who saw service in the summer of 1950, began to relapse throughout the spring and summer of 1951 (fig. 4). These relapses eventually suggested a very high rate of suppressed infection in the 8th Army.

The Army Malaria Mission to the Far East Command during August and September 1951 was undertaken to establish the feasibility of using primaquine for mass therapy on the rotatees from Korea.[8] Since these men had been on fairly successful chloroquine discipline until the time of embarkation from Japan it was expected that a malaria-free interval of approximately six weeks existed in which to eradicate the tissue stages of the infection and to prevent the expected clinical disease. Because it is impossible to determine which men are infected prior to a clinical attack, it is necessary to treat all men as if they have the disease.

In the light of present experience with primaquine, it was the opinion of the Mission, with the concurrence of the Malaria Subcommittee of the National Research Council, that primaquine should be given for a two week period in single daily doses of 15 mg. of base. In the beginning of this study the dose could be arrived at only by considering many factors, for no actual trials against Korean malaria with long-term follow-up had been possible. Although primaquine was carefully tested against Chesson strain vivax malaria, it is well recognized that differences in response to drugs exist among the many known strains and varieties of malaria. The most persuasive reason for choosing the 15 mg. dose was that this dosage represents the maximum that can be given to large groups without special medical supervision. The most dangerous toxic effect so far

SCHEMATIC REPRESENTATION OF THE NATURAL COURSE OF VIVAX MALARIA


CLINICAL COURSE
PARASITES
BLOOD STAGES
TISSUE STAGES
Transmission from mosquito to man
SPOROZOITES
PREPATENT PERIOD (NO SYMPTOMS)
PRIMARY CLINICAL ATTACK†
(CHILLS, FEVER, etc.)
Transmission from man to mosquito
LATENT PERIOD (NO SYMPTOMS)
CLINICAL RELAPSE†
(CHILLS, FEVER, etc.)
Transmission from man to mosquito
TROPHOZOITES-Schizonts (asexual)
GAMETOCYTES (sexual)
TROPHOZOITES-Schizonts (asexual)
GAMETOCYTES (sexual)
PRIMARY TISSUE STAGES
LATE TISSUE STAGES (hypothetical)
LATE TISSUE STAGES (hypothetical)

MODE OF ACTION OF IMPORTANT ANTIMALARIAL DRUGS

DRUGS					
TYPE OF DRUG ACTION →	PROPHYLACTIC (prevents clinical malaria)	SUPPRESSIVE(*) (abolishes clinical attack)	CURATIVE (prevents relapse)	SUPPRESSIVE(*) (abolishes clinical attack)
POINT OF ACTION →	VS SPOROZOITES AND PRIMARY TISSUE STAGES	VS TROPHOZOITES	VS LATE TISSUE STAGES	VS TROPHOZOITES
NAME OR TYPE OF DRUG →	Primaquine and Paludrine® (Chlorguanide) (†)	Quinine, Atabrine® (Quinacrine), Paludrine® (Chlorguanide) and some 4-aminoquinolines: Aralen® (Chloroquine), Oxychloroquine or Camoquin®	Some 8-aminoquinolines: Plasmochin (Pamaquine), Pentaquine, Isopentaquine and Primaquine	(Same as for primary attacks)

(†) TO ACHIEVE RADICAL CURE DURING CLINICAL ATTACK DRUGS MUST HAVE BOTH SUPPRESSIVE & CURATIVE PROPERTIES

(*) Unlike gametocytes of P. falciparum, those of P. vivax persist only a *few hours* after abolition of trophozoites. Therefore, suppressive drugs have an indirect gametocyticidal effect.

☐ = Erythrocytic forms ▨ = Gametocytes only ⬚ = tissue stages

Fig. 2.—Schematic representation of the natural course of vivax malaria and mode of action of important antimalarial drugs.

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[8] The success of this mission was due to the splendid cooperation of the Army medical officers in the Far East Command, the Japanese Log. Command, the Eighth Army, the Second Log. Command and the Navy officers assigned to the Military Sea Transport Service. The mission is especially indebted to Colonel Arthur H. Long, consultant in preventive medicine to the Surgeon, Far East Command, whose extensive knowledge and experience with the medical and administrative problems of the Far East Command proved invaluable.

recognized has been the special susceptibility of some Negroes to hemolytic reactions to primaquine in doses greater than 15 mg. of base per day.[9]

Rotation Program.—An essential part of the success of a mass therapy program depends on the administrative obstacles involved. Accordingly, the troop rotation program was analyzed in detail to find the most promising two week period in which to give primaquine. Since many exceptions exist in the routine rotation program, it was apparent that all men could not be included without overwhelming administrative difficulty. The optimum period for giving the drug to the greatest number of men under the present system of rotation appeared to be from the time of each man's arrival at the replacement depot in Japan to the end of the 14 days of treatment, which would usually occur some time at sea. Because shipboard therapy made additional complications for the routine use of primaquine, the clinical investigations reported here were made aboard ship.

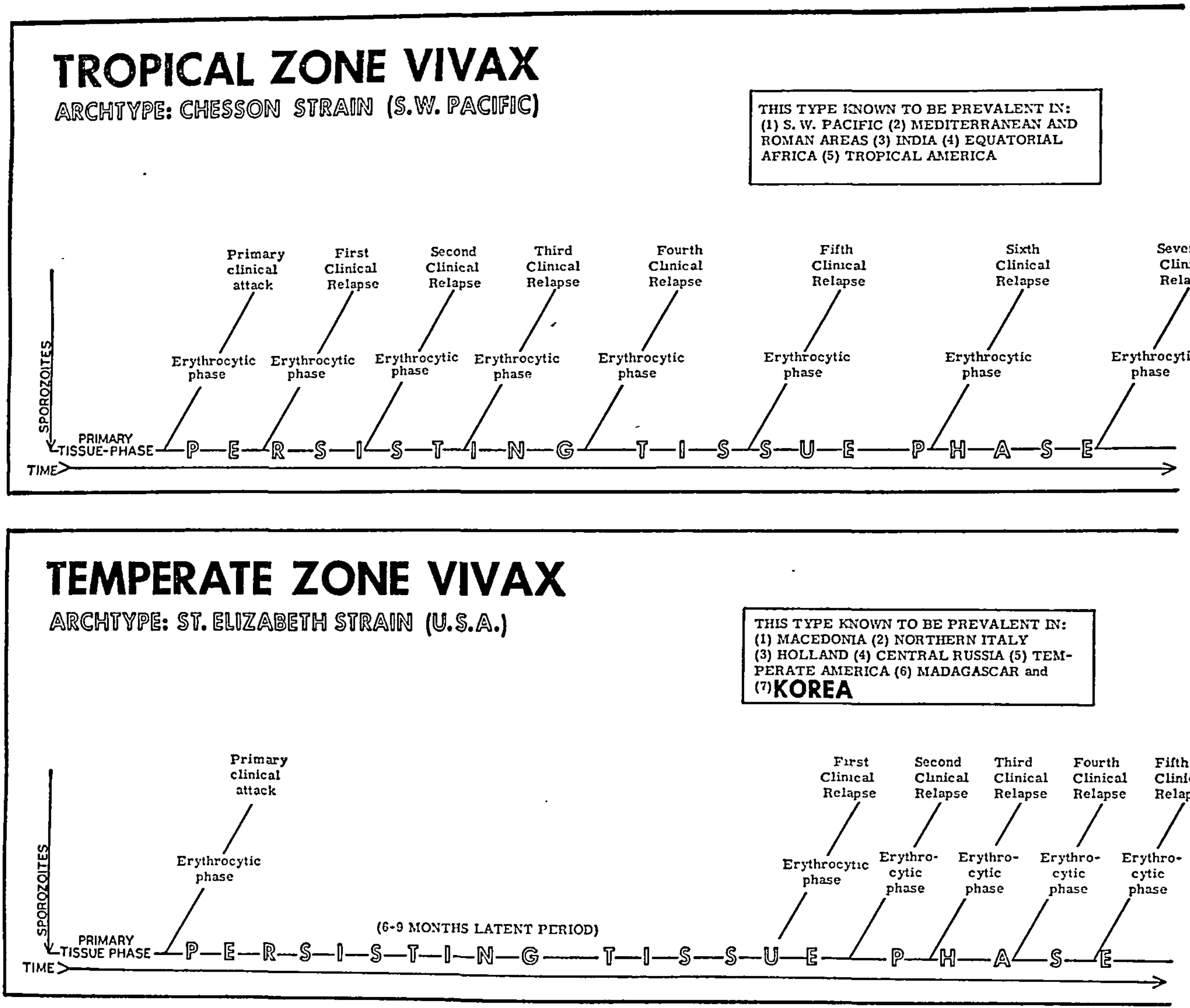

Fig. 3.—Two chief types of vivax malaria showing characteristic relapse patterns when each attack is treated by suppressive drugs.

CLINICAL TRIALS WITH PRIMAQUINE

Two independent clinical trials were performed under actual rotation conditions to determine: (1) the feasibility of continued administration of primaquine (15 mg. of base daily) to large groups of men without special medical supervision, (2) whether primaquine toxicity increased the incidence of motion sickness, (3) the effect of motion sickness on the ability to take and retain medication, and (4) whether primaquine begun on land would show enough cumulative toxicity to affect the motion sickness most common during the first four days at sea.

Studies Aboard U. S. N. S. Sgt. Sylvester Antolak.—The 1,493 officers and men on the U. S. N. S. Sgt. Sylvester Antolak, sailing from Sasebo, Japan, to Seattle on Sept. 18, 1951, were chosen for the first clinical trials. They were divided into two groups. One group of 742 officers and men, of which 16.9% were Negroes, was given primaquine (15 mg. of base daily) for 12 days immediately before the noon meal each day. The other group of 751 men received a placebo under the same conditions as the first group. Men received the pills even if they did not eat the noon meal. All cases of seasickness severe enough to send a man to sick call were evaluated and recorded. The voyage was rougher than the average Pacific run for that season.

Results of Clinical Trial.—Forty-six men in the primaquine group (742 officers and men) reported for seasickness; exactly the same number of men (46) from the control group of 751 officers and men also reported for seasickness. Only 3.9% of all men aboard missed one or more tablets. This means that 96% of all men aboard received either primaquine or placebo for 12 days without interruption. There was no sign of toxicity. None of the Negroes showed any evidence of hemolysis.

Studies Aboard U. S. N. S. Marine Phoenix.—This second study was arranged to test the effect of cumulative toxicity (which would come, if at all, 3 or 4 days after starting therapy) on motion sickness. Accordingly, after the U. S. N. S. Antolak sailed, the next 3,000 men processed at the Sasebo Replacement

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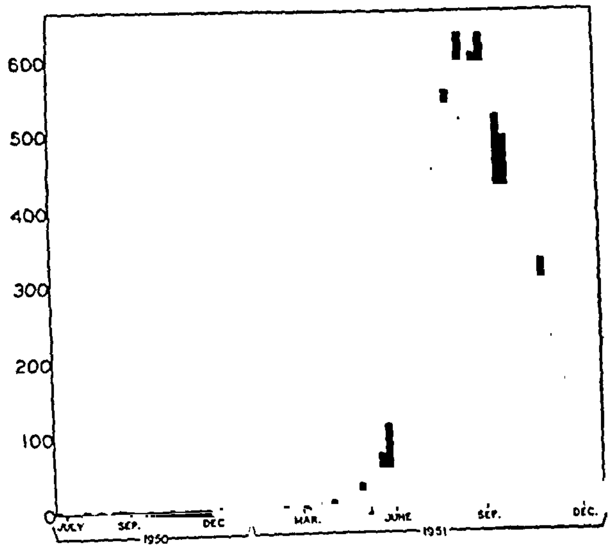
[9] Hockwald, R. S.; Arnold, J.; Clayman, C. B., and Alving, A. S.: Toxicity of Primaquine in Negroes, J. A. M. A. this issue, p. 1568.

Depot (Prov.) were started on primaquine (15 mg. of base) once daily as soon as they arrived at that installation. From this group, 2,785 eventually sailed on the U. S. N. S. Marine Phoenix bound for Seattle. Members of this group spent a variable length of time at the Replacement Depot. Twenty-nine per cent had been at Sasebo 10 days before sailing and consequently received 10 pills before embarking. The remainder spent two to nine days at the depot prior to sailing.

The U. S. N. S. Marine Phoenix sailed Sept. 23, 1951. Two thousand sixty officers and men, of which 7.6% were Negroes, were selected to continue primaquine while 725 men were given placebos. It was impossible to give either primaquine or placebo by the roster because of the complexity of shipboard living. The total number of men receiving either pill was counted each day. For other data it was necessary to resort to sampling techniques. Only men missing mess failed to receive primaquine or placebo, as both were given just prior to eating. Generally, the amount of seasickness was reflected in the number of men at mess on that day. The number of men in either group missing mess was taken as a measure of gastrointestinal distress. Every man in sick call each day was seen for possible signs of toxicity. This was an unusually rough voyage.



▲ FIRST SHIP LOAD KOREA VETERANS LANDED IN U S. MAY 5, 1951

Fig. 4.—Number of Army personnel hospitalized for malaria in the United States each week since onset of Korean conflict. (Source: Medical Statistics Division, Office of the Surgeon General, Department of the Army.)

Results of Clinical Trial.—There was no detectable evidence of toxicity among the 2,060 officers and men who received primaquine. The amount of seasickness in the primaquine group was closely paralleled by the amount among the controls. Eighty-two and one-tenth per cent of all men aboard received 13 to 20 pills consecutively. An additional 8.8% received all but one of the scheduled pills. This means that 90.9% received 12 or more pills with an interruption of not longer than one day. This is the minimum period considered necessary for adequate therapy.

CONCLUSIONS

The trials reported here demonstrate the feasibility of using primaquine as a curative antimalarial in large numbers of men without special medical supervision, even under the difficult conditions met with at sea. Because of the tremendous medical load produced by these relapsing cases of malaria, the Armed Forces adopted primaquine for the treatment of all servicemen returning from Korea after endorsement by the Subcommittee on Malaria of the National Research Council and the Armed Forces Medical Policy Council. The success of this program, however, will depend upon the administrative discipline instituted to enforce it and upon the proper indoctrination of medical officers and troops in the aims and desirability of strict adherence to the 14 day therapeutic regimen.

2. CURE OF KOREAN VIVAX MALARIA WITH PAMAQUINE AND PRIMAQUINE

Capt. Paul L. Garrison, Lieut. Daniel D. Hankey
Capt. Walter G. Coker, Lieut. Col. William N. Donovan
and Col. Bruno Jastremski, (MC), United States Army
G. Robert Coatney, Scientist Director, U. S. Public Health Service, Bethesda, Md.
Alf S. Alving, M.D., Chicago
Ralph Jones Jr., M.D., Philadelphia

In the summer of 1951, when large numbers of Korean veterans came down with vivax malaria, a study designed to compare the curative effectiveness of pamaquine and primaquine was initiated. This preliminary report presents the results of that study after 11 months of observation.

METHODS

All patients admitted to the station hospitals with vivax malaria between July 25, 1951, and May 1, 1952, were included in this study. The diagnosis was established in every case by demonstrating parasites in the peripheral blood in two nonconsecutive smears. When the diagnosis was established, patients were treated in rotation according to one of the following regimens:

Regimen A: Chloroquine.—A total dosage of 1.5 gm. of base was given in three doses of 0.3 gm. each during the first 24 hours, followed by a single dose of 0.3 gm. daily for two days.

Regimen B: Chloroquine Plus Pamaquine.—A total dose of 1.5 gm. of chloroquine base was administered as in regimen A. In addition, 27 mg. of pamaquine base was given daily in three equally divided doses for 14 consecutive days.

Regimen C: Chloroquine Plus Primaquine.—A total dose of 1.5 gm. of chloroquine base was administered as in regimen A. In addition, 15 mg. of primaquine base was given in a single dose daily for 14 consecutive days.

All patients were hospitalized for the duration of treatment and were observed daily for evidence of toxicity. A white blood count, hemoglobin concentration, and routine urinalysis were taken when the patients were admitted. These studies were repeated after one week, for patients treated with regimen B or C, and at the end of therapy. Additional studies were performed as indicated.

When treatment was completed, the patients were discharged to duty and followed as outpatients at intervals of approximately six weeks. At each follow-up visit, patients were interviewed and a thick film of peripheral blood was examined for parasites. When relapses occurred, patients were treated according to the regimen originally employed.

RESULTS

A total of 864 patients were treated. Of this group, 709 were followed for 4 to 11 months after treatment (see table). The clinical manifestations of the acute attack of malaria were rapidly controlled by chloroquine.

Sixty-four (27.6%) of 232 patients treated with chloroquine alone, and followed for more than 4 months, relapsed. Sixty-one of the 64 patients experienced a typical febrile relapse with parasitemia; three had parasitemia but no fever.

A typical clinical and parasitic relapse 54 days after the last dose of drug developed in one (0.4%) of the 246 patients treated with chloroquine plus pamaquine and followed for more than four months. This patient was treated again according to the original regimen. No further relapse has occurred during 134 days of observation.

From the Department of Medicine, University of Chicago (Dr. Alving) and the Department of Medicine, University of Pennsylvania (Dr. Jones), and the Section on Chemotherapy, Laboratory of Tropical Diseases, National Microbiological Institute, National Institutes of Health (Dr. Coatney).

This study is limited to observations conducted in the station hospitals at Fort Knox and Fort Benning under the auspices of the Research and Development Board of the Office of the Surgeon General of the Army. Important supplementary studies have been conducted by the Army in the station hospitals at Fort Dix, Fort Meade, Camp Breckenridge, and, by the Navy, at Camp Lejune.

None of the 231 patients treated with chloroquine plus primaquine and followed for more than four months has relapsed.

In the course of these studies, only minimal evidence of drug toxicity was observed. Mild generalized pruritus which subsided 4 days after the drug was stopped developed in one patient treated with chloroquine alone. A similar mild pruritus developed in one patient treated with chloroquine and primaquine. It disappeared after chloroquine was withdrawn, before therapy with primaquine was completed. Occasionally, a patient treated with chloroquine plus pamaquine complained of mild, crampy abdominal pain at some time during therapy. This was not of sufficient severity to warrant stopping the drug. There was no evidence of hemolytic anemia in any patient. Approximately 15% of the patients had a mild to moderate anemia (hemoglobin concentration of 8 to 12 gm. per 100 cc.) at the time treatment was instituted. In these patients, the hemoglobin concentration increased during therapy as the acute attack was controlled. There was a slight, but probably significant, increase in leucocyte count at the end of therapy in patients treated with pamaquine or primaquine. Methemoglobin determinations were not done; cyanosis was not observed.

Treatment of Korean Vivax Malaria

Patients	Regimen A, Chloroquine, 1.5 Gm. (Base)	Regimen B, Chloroquine, 1.5 Gm. (Base) Plus Pamaquine 27 Mg. per Day for 14 Days	Regimen C, Chloroquine, 1.5 Gm. (Base) Plus Primaquine 15 Mg. per Day for 14 Days
Total Number Followed..............	287	291	286
Number Followed 4 to 11 Months After Treatment..............	232	246	231
Number Followed 4 to 11 Months and Relapsed....................	64 (27.6%)	1 (0.4%)	0

DISCUSSION

Korean vivax malaria is the temperate zone type characterized by a long latent interval of 6 to 10 months following the initial infection.[1] All of the patients in this study acquired malaria in Korea during the summer of 1950. The attacks reported here occurred after the latent interval.

The relapse rate (27.6%) after treatment with chloroquine is lower than that reported for other strains of vivax malaria.[2] This can be explained partly by the fact that these studies were inaugurated late in 1951, near the end of the life span of the parasite.

It is apparent from these studies that 15 mg. of primaquine daily or 27 mg. of pamaquine daily for 14 days, given in combination with chloroquine, will prevent relapses in nearly all cases of Korean vivax malaria. It is reasonably certain that few, if any, of the patients treated with pamaquine or primaquine will experience a relapse during the summer of 1952, because the relapse rate was reduced from 27.6% after chloroquine to less than 1% after treatment with pamaquine and primaquine.

Both pamaquine and primaquine were well tolerated, and a significant difference in toxicity or in therapeutic effectiveness was not demonstrated. The dose of pamaquine (27 mg. per day) used in these studies was nearly twice as great as the dose of primaquine (15 mg. per day) so that an exact comparison of therapeutic effectiveness was not possible.

Primaquine is undoubtedly the drug of choice for curative treatment of Plasmodium vivax malaria because of the greater spread between the effective and the toxic dose. However, it is apparent that either pamaquine or primaquine in doses which do not produce significant toxicity will cure most vivax infections of Korean origin. Because primaquine and pamaquine are relatively ineffective in destroying the erythrocytic stages of the malaria parasite, these drugs should be given simultaneously with chloroquine for the treatment of an acute attack. Because of the greater toxicity to pamaquine exhibited by Negroes, this drug should not be used for treatment of an acute attack unless the patients are under close medical supervision and unless the hemoglobin is followed daily.

SUMMARY

In a study of the effect of pamaquine and primaquine on the relapse rate of vivax malaria acquired in Korea, 709 patients were treated at the time of an acute attack and were followed for 4 to 11 months after treatment. Of 232 patients treated with chloroquine alone, 27.6% relapsed at least once after treatment. In a group of 246 patients treated with chloroquine and 27 mg. of pamaquine daily for 14 days, one patient (0.4%) relapsed.

Relapses did not occur among 231 patients treated with chloroquine and 15 mg. of primaquine daily for 14 days. Significant toxicity was not observed during this study.

3. TOXICITY OF PRIMAQUINE IN CAUCASIANS

Capt. Charles B. Clayman, Major John Arnold
Capt. Robert S. Hockwald, Capt. Ernest H. Yount Jr., and
Capt. John H. Edgcomb, (MC), United States Army,
Alf S. Alving, M.D., Chicago

Primaquine, 8-(4-amino-1-methylbutylamino)-6-methoxyquinoline,[1] a new curative agent for vivax malaria, has recently been extensively tested in the Armed Forces. Since mid-December, 1951, veterans returning from Korea have received 15 mg. of primaquine base once daily for two weeks aboard Military Sea Transport ships after termination of weekly chloroquine (aralen®) suppression at the time of embarkation.[2] Chloroquine remains in the blood in effective concentration for several weeks; it suppresses clinical symptoms by eliminating the erythrocytic forms of the malaria parasite but does not prevent subsequent relapses. Relapses can be prevented, however, by the administration of primaquine, which acts primarily against the exoerythrocytic parasites. Thus, combined action of the two drugs has been exploited during the transpacific voyage to achieve radical cure of vivax infections before symptoms have developed. Primaquine, combined with chloroquine, has also been employed extensively in treating clinical attacks of vivax malaria in Army installations in the United States.[2]

Curative dosages of primaquine may vary from 10 to 30 mg. of base administered daily for two weeks. The ideal dose within this range depends on several factors, including the species and strain of the infecting parasite, the degree of natural or acquired immunity of the individual, and the density of the sporozoite inoculum. Ten milligrams may produce radical cure in persons having great immunity.[3] Limited experience with the Korean strain of vivax malaria indicates that 15 mg. is probably sufficient to cure over 99% of patients [2] (table 1). Twenty-two and one-

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[1] Coatney, G. R., and others: Studies in Human Malaria. XVIII. Life Pattern of Sporozoite-Induced St. Elizabeth Strain Vivax Malaria, Am. J. Hyg. **51**: 200-215 (March) 1950.

[2] Most, H., and others: Chloroquine for Treatment of Acute Attacks of Vivax Malaria, J. A. M. A. **131**: 963-967 (July 20) 1946. Gordon, H. H., and others: Treatment of Plasmodium Vivax Malaria of Foreign Origin; Comparison of Various Drugs, Arch. Int. Med. **79**: 365-380 (April) 1947. Coatney, G. R., and others: Studies in Human Malaria: X. Protective and Therapeutic Action of Chloroquine (SN 7618) Against St. Elizabeth Strain Vivax Malaria, Am. J. Hyg. **49**: 49-59 (Jan.) 1949.

From the Army Malaria Research Unit, University of Chicago.

The authors wish to acknowledge the invaluable technical assistance of Shirley Mock Swanson, R.N., and Miss Ann Flint, and to express their appreciation to the inmate nurses, technicians, and volunteers whose participation and cooperation permitted these studies to be made.

The investigations covered in parts 2 and 3 of this report were carried out under contract between the University of Chicago and the Medical Research and Development Board, Office of the Surgeon General, Department of the Army and were, in part, supported by a grant-in-aid from the United States Public Health Service. The studies would not have been possible except for the valuable cooperation and help given by Warden Joseph E. Ragen of Stateville Penitentiary and other administrative officials of the State of Illinois.

[1] The authors are indebted to Dr. C. E. Roach of Eli Lilly and Company, Indianapolis; Dr. Marlin Leffler of the Abbott Laboratories, North Chicago, Illinois, and Dr. Justus B. Rice of Winthrop-Stearns, Inc., New York, for providing supplies of *primaquine*.

[2] Returning Korean Veterans to Receive New Anti-malarial Drug, Department of the Army, Office of the Surgeon General, Technical Information Office, Technical Release dated Oct. 19, 1951. Coatney, R. G., and others: Unpublished data on clinical studies of primaquine conducted at Atlanta Penitentiary

TABLE 1.—*Curative Treatment During Clinical Attack of Vivax Malaria Acquired in Korea*[2]

Day		Drug: Chloroquine (Base)	Drug: Primaquine (Base)*
1st day	Initial dose	0.3 gm.	0.015 gm.
	After 6 hours	0.3 gm.	None
2nd, 3rd, and 4th day	In a. m.	0.3 gm.	0.015 gm.
5th to 14th day inclusive	In a. m.	None	0.015 gm.
Total dose	....	1.5 gm.	0.210 gm.
Daily dose	....	....	0.015 gm.

* Primaquine should be given with meals. The third dose of chloroquine should be administered approximately 24 hours after the initiation of treatment. The blood hemoglobin *concentration* should be determined for any patient whose urine is of extremely dark or brown color.

half milligrams cures practically all persons heavily infected with the Chesson (Southwest Pacific) strain of vivax malaria under experimental conditions.[4] Thirty milligrams may conceivably be necessary to cure nonimmune persons infected by unusually large sporozoite inocula. Therapy during a clinical attack should include concurrent administration of quinine or chloroquine, of which the latter is recommended.

STUDIES AT STATEVILLE PENITENTIARY

Since early 1948, 699 light-skinned Caucasian adult male volunteers have received primaquine at the Illinois State Penitentiary (Stateville Branch). The men varied in weight from 130 to 260 pounds, the mean being 161 pounds. Three hundred and thirty-five were either noninfected or were treated in the latent interval between clinical attacks of malaria. The remaining 364 volunteers were treated during a clinical attack (table 2).

TABLE 2.—*Dosage Schedules Employed in Primaquine Administration to 699 White Inmate Volunteers During Clinical Investigations at Stateville Penitentiary*

	Treatment During Clinical Malaria								Treatment in Absence of Clinical Malaria							
	Mosquito Induced				Blood Induced				Prophylaxis				Interim Therapy or Toxicity Study			
Daily Dosage of Primaquine and Schedule of Administration to White Subjects	*Alone*	With Quinine	With Chloroquine	With Other Drugs	Alone	With Quinine	With Chloroquine	With Other Drugs	Alone	With Quinine	With Chloroquine	With Other Drugs	Alone	With Quinine	With Chloroquine	With Other Drugs
10 mg. in 6 divided doses for 8 days	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..
12 mg. in single daily doses for 14 days	..	6	..	..	..	..	..	..	..	..	..	..	..	..	..	..
15 mg. in single daily doses for 14 days	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..
15 mg. in 6 divided doses for 14 days	10	33	24	2	..	..	..	..	..	..	..	..	..	..	..	..
18 mg. in 6 divided doses for 14 days	..	13	..	..	..	..	..	..	..	..	..	..	..	..	..	..
20 mg. in single daily doses for 14 days	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..
22.5 mg. in single daily doses for 14 days	11	..	..	6	..	..	..	..	..	..	..	..	..	..	..	..
22.5 mg. in 6 divided doses for 14 days	5	48	..	6	..	..	..	..	..	..	..	..	..	..	..	..
30 mg. in single daily doses for 14 days	20	4	19	27	1	..	13	..	..	..	..	..	45	..	123	24
30 mg. in single daily doses for 6 to 8 days	..	..	..	..	..	..	..	3	18	..	..	..	34	..	7	..
30 mg. in 3 divided doses for 14 days	..	..	..	4	..	..	..	..	..	..	..	..	..	..	..	..
30 mg. in 6 divided doses for 14 days	..	8	..	1	4	..	..	..	..	..	..	..	..	..	..	..
30 mg. in 6 divided doses for 6 to 10 days	..	..	..	..	6	..	..	2	..	..	..	..	..	..	..	..
30 mg. in single doses twice weekly for 4 weeks	..	..	..	..	..	..	..	..	19	..	..	..	..	..	..	..
30 mg. in single weekly doses for 14 weeks	..	..	..	..	..	..	..	..	5	..	10	..	..	..	..	..
30 mg. in single weekly doses for 52 weeks	..	..	..	..	..	..	..	..	..	..	..	..	..	..	20	..
30 mg. in single doses twice weekly for 52 weeks	..	..	..	..	..	..	..	..	..	..	..	..	..	..	20	..
45 mg. in single daily doses for 14 days	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..	8
45 mg. in 6 divided doses for 14 days	5	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..
60 mg. in 6 divided doses for 14 days	..	4	..	..	7	..	..	..	..	..	..	..	..	..	..	..
60 mg. in 3 divided doses for 6 days	..	..	..	..	4	..	..	..	..	..	..	..	..	..	..	..
60 mg. in 6 divided doses for 6 days	..	..	..	..	..	..	..	4	..	..	..	..	..	..	..	..
90 mg. in 3 divided doses for 14 days	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..
90 mg. in 3 divided doses for 6 days	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..
90 mg. in 6 divided doses for 14 days	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..
120 mg. in 6 divided doses for 14 days	..	..	..	8	17	6	..	12	..	..	..	..	..	..	..	..
240 mg. in 6 divided doses for 14 days	..	..	..	1	..	2	..	1	..	..	..	..	..	..	..	..
240 mg. in 6 divided doses for 5 to 10 days	1	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..
	52	116	44	60	46	8	13	25	43	0	10	1	79	0	170	32
	272				92				54				291			
	364								335							
	Total - 699															

The following is a report of the toxicity of primaquine observed during these studies.[5] Parallel studies of pamaquine were used as a control. General procedures during these observations followed those outlined by Alving and co-workers.[6] Details of drug regimens and laboratory procedures are described below.

DRUG REGIMENS

Primaquine was administered orally as the diphosphate salt containing 56.9% of base. *Dosages of all drugs are expressed in terms of base weight.*

Primaquine was administered in programs utilizing single or divided daily dosage schedules. Daily dosage ranged from 10 to 240 mg., administered for 5 to 14 days. In addition, single doses of 30 mg. of primaquine were given weekly and semiweekly for 52 weeks together with chloroquine.

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[3] Thaeler, A. D., Jr.; Arnold, J., and Alving, A. S.: Field Studies of Primaquine in Nicaragua, presented at the Annual Meetings of the National Malaria Society, Chicago, Nov. 14-17, 1951. Garrison, P. L., and others: Cure of Korean Vivax Malaria with Pamaquine and Primaquine, J. A. M. A., this issue, p. 1562. In addition to the major studies at Fort Knox and Fort Benning, important trials of primaquine are being conducted at Camp Breckenridge, Fort Dix, and Fort Mead under the auspices of the Army and at Camp Lejune in cooperation with the Navy.

[4] Edgcomb, J. H., and others: Primaquine, S. N. 13,272, A New Curative Agent in Vivax Malaria: A Preliminary Report, J. Nat. Malaria Soc. 9:285-292 (Dec.) 1950.

[5] Earlier toxicity studies were performed in primates by Dr. Leon H. Schmidt, The Christ Hospital Institute of Medical Research, Cincinnati.

[6] Alving, A. S., and others: Procedures used at Stateville Penitentiary for Testing of Potential Antimalarial Agents, J. Clin. Investigation 27: 2-5 (May) 1948.

Pamaquine was administered on similar schedules in dosages of 15, 31.5, and 63 mg. for 14 days.

The suppressive action of pamaquine, even at the maximum tolerated dose of 90 mg. daily, is inadequate to terminate attacks of malaria quickly or consistently.[7] Therefore, quinine was used as the standard concurrent suppressive antimalarial drug in testing potential curative agents during World War II. Although better suppressive agents have since been developed, quinine has been retained to make results of current studies comparable with earlier investigations. When chloroquine was substituted as the concurrent suppressive drug, clinical malaria was almost invariably terminated during the first two days.

During the standard two week treatment schedule, 180 men received 15 or 30 mg. of primaquine daily, together with 1 gm. of chloroquine, administered in two divided doses during the first 24 hours of treatment (table 2). One hundred fifteen men were given 1.64 gm. of quinine (2 gm. of quinine sulfate) in six divided doses daily, concurrently with varying doses of primaquine. Primaquine was given alone to 220 men. Forty-three received four times the maximum therapeutic dose for 14 days and 7 men received eight times that dose for 5 to 14 days, either alone or concurrently with other drugs. One hundred eighteen men received the following potential antimalarial agents concurrently with varying doses of primaquine: methylene blue, nicotinamide, thionine, Bindschedler's green, toluidine blue, theobromine, aminophylline, ephedrine sulfate, chloramphenicol, or daraprim® (2,4-diamino-5-*p*-chlorophenyl-6-ethylpyrimidine).[8]

PROCEDURES

Drug Administration for 14 Days.—Hemoglobin and methemoglobin determinations[9] were made on all men before they received any drug. During treatment of clinical malaria these determinations were usually made every other day until the last 5 days, when they were performed daily. White blood cell counts were done routinely every other day with differential counts every fourth day; both were checked daily if significant variation from control values occurred. Daily urinalyses were performed on subjects treated during clinical attacks of malaria or whenever toxicity was anticipated. During interim therapy, hemoglobin and methemoglobin levels and white blood cell counts were usually done on the first, seventh, tenth, and fourteenth days of drug administration.

Plasma concentrations of primaquine and pamaquine were determined every other day by the method of Brodie, Undenfriend, and Taggart,[10] which measures only the heptane extractable, diazotized fraction of the drug.

Determinations of direct and indirect serum bilirubin,[11] 30-minute sulfobromophthalein (bromsulphalein®) retention,[12] thymol turbidity,[13] and total plasma proteins[14] were performed on 57 subjects. Control tests were made prior to drug administration and were repeated on the 3rd, 7th, and 14th days.

Weekly and Semiweekly Drug Administration.—Hemoglobin estimations and red and white blood cell counts were performed every other week in the groups receiving chloroquine concurrently with primaquine semiweekly or weekly for 4, 14, and 52 weeks.

EVALUATION OF TOXIC MANIFESTATIONS

Malaria produces anemia, abnormalities in white blood cell counts, and symptoms that may be confused with toxic manifestations of a drug. Consequently, evaluation of toxicity was begun following elimination of the febrile attack. The validity of this method of interpretation of toxic symptoms is supported by the fact that the toxicity of primaquine, when given in the absence of clinical malaria, seldom becomes apparent for three or four days except at extremely high doses (0.12 and 0.24 gm.).

Detailed results are reported only for groups receiving primaquine or pamaquine alone and concurrently with quinine or chloroquine for two weeks. Toxicity observed during shorter or unusual regimens is discussed only when results contradict or modify the conclusions drawn from the standard programs.

TOXICITY AT THERAPEUTIC DOSES

Fifteen Milligrams (Table 3).—Doses of 15 mg. or less produced abdominal symptoms of no greater incidence or severity than those encountered with a placebo. Methemoglobin levels did not rise above 7.8% of the total hemoglobin. One of 67 subjects (1.5%) lost 1.3 gm. per 100 cc. of hemoglobin; the variation in hemoglobin was within ± 1 gm. per 100 cc. in the remaining volunteers. Four subjects (5.9%) had leukocytosis ranging from 11,500 to 13,700 per cubic millimeter. Leukopenia did not occur.

Twenty-Two and One-Half Milligrams.—Seven of 57 subjects (12.3%) had mild to moderate abdominal symptoms during administration of 22.5 mg. of primaquine. No hemoglobin loss exceeded 1 gm. per 100 cc. Twelve volunteers (21%) manifested leukocytosis from 12,100 to 16,400 per cubic millimeter. Leukopenia was not observed.

Thirty Milligrams.—Twenty-two of 214 persons (10.3%) reported mild to moderate abdominal cramps, including six who also complained of epigastric distress which was relieved by antacids. Thirty-two persons (14.9%) lost up to 2 gm. per 100 cc. of hemoglobin. Leukocytosis of 10,000 to 17,000 per cubic millimeter occurred in eight subjects (3.8%). Leukopenia did not occur.

TOXICITY AT HIGH DOSES

Sixty Milligrams (Table 3).—Five of seven volunteers receiving daily doses of 60 mg. during clinical attacks of malaria experienced abdominal symptoms. Complaints ranged from mild and transient to moderate and repeated, or severe and persistent, abdominal cramps. Other abdominal symptoms were anorexia, nausea, occasional vomiting, and mild burning epigastric distress. Cyanosis caused by methemoglobinemia was present in the three subjects receiving primaquine alone. The subjects given quinine concurrently with primaquine did not become cyanotic. One volunteer had a leukocytosis of 13,500 per cubic millimeter. Hemoglobin loss or leukopenia was not observed.

One Hundred Twenty Milligrams. — Abdominal symptoms were present in all volunteers receiving primaquine alone or in combination with quinine. Symptoms were so severe in one man receiving quinine concurrently that it was necessary to discontinue the drugs. The five men receiving only primaquine had methemoglobinemia severe enough to produce cyanosis, while only two of six volunteers receiving quinine concurrently with primaquine appeared cyanotic. Hemoglobin variation was within ± 1 gm. per 100 cc. of the control values in the men receiving primaquine alone. Two of the five men receiving quinine concurrently with primaquine lost between 1.1 and 2 gm. per 100 cc. of hemoglobin. Two cases of leukopenia were observed. Agranulocytosis was not encountered.

Two Hundred Forty Milligrams.—Daily doses of 240 mg. further accentuated abdominal symptoms. This group has not been expanded since the preliminary report of Edgcomb and coworkers[4] who discussed in detail the toxic manifestations.

EFFECT OF CONCURRENT ADMINISTRATION OF OTHER DRUGS

Quinine.—Within the therapeutic dosage range, concurrent administration of quinine with primaquine did not increase abdominal symptoms beyond the amount clearly attributable to cinchonism. At higher dosages of primaquine, however, the concurrent administration of quinine increased the subjective manifestations of toxicity.

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[7] Berliner, R. W., and others: Studies on Chemotherapy of Human Malarias: VII. The Antimalarial Activity of Pamaquine, J. Clin. Investigation **27**: 108-113 (May) 1948.

[8] The authors are indebted to Dr. G. H. Hitchings of Burroughs-Wellcome and Company, Tuckahoe, N. Y., for providing supplies of daraprim.®

[9] Evelyn, K. A., and Malloy, H. T.: Microdetermination of Oxyhemoglobin, Methemoglobin, and Sulfhemoglobin in a Single Sample of Blood, J. Biol. Chem. **126**: 655-662 (Dec.) 1938.

[10] Brodie, B. B.; Udenfriend, S., and Taggart, J. V.: Analysis of Basic Organic Compounds in Biological Tissues: 4. Coupling with Diazonium Salts, Fed. Proc. **5**: 125 (Feb.) 1946.

[11] Malloy, H. T., and Evelyn, K. A.: The Determination of Bilirubin with the Photoelectric Colorimeter, J. Biol. Chem. **119**: 481-490 (July) 1937.

[12] Rosenthal, S. M., and White, E. C.: Clinical Application of Bromsulphalein Test for Hepatic Function, J. A. M. A. **84**: 1112-1114 (April 11) 1925.

[13] Shank, R. E., and Hoagland, C. L.: Modified Method for Quantitative Determination of Thymol Turbidity Reaction of Serum, J. Biol. Chem. **162**: 133-138 (Jan.) 1946.

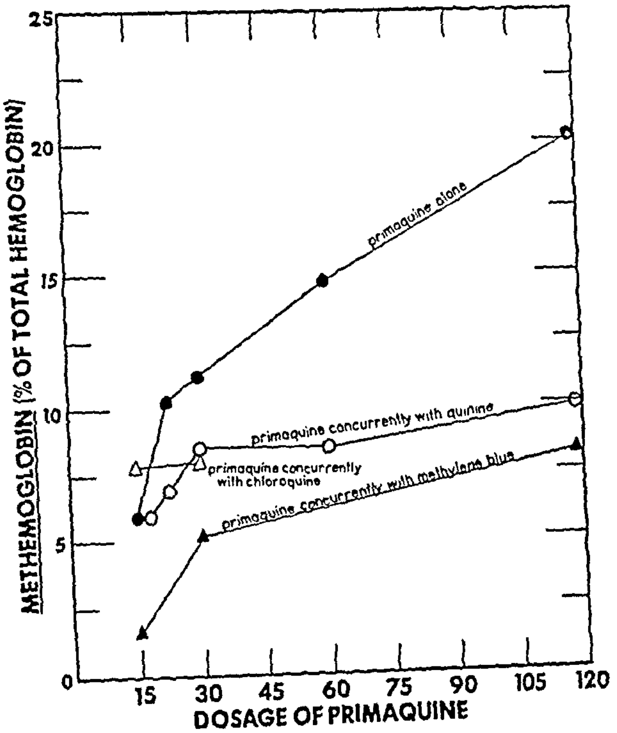
[14] Phillips, R. A.; Van Slyke, D. D.; Dole, V. P.; Emerson, K., Jr.; Hamilton, P. B., and Archibald, R. M.: Copper Sulfate Method for Measuring Specific Gravities of Whole Blood and Plasma, Bull. U. S Army Med. Dept. 71 (Dec.) 1943, pp. 66-83.

Above 15 mg. of primaquine, methemoglobin concentrations were lower when quinine was administered concurrently. This effect of quinine became more apparent as the therapeutic range of primaquine was exceeded (fig. 1).

Within the therapeutic range, the average concentration of primaquine in the plasma was unaffected by concurrent administration of quinine. In the small number of patients treated at very high dosages, the average plasma concentration of primaquine was approximately 50% lower in the group receiving quinine concurrently.

Chloroquine.—In the therapeutic range, chloroquine did not increase the toxicity of primaquine. The symptoms and toxic manifestations after the first two days were the same as those encountered during drug administration to well persons.

Miscellaneous Drugs.—Methylene blue, nicotinamide, thionine, Bindschedler's green, and toluidine blue were tested at widely varying doses as concurrent drugs. Methemoglobinemia during their administration in sufficiently high dosages was lower than when primaquine was administered alone. Methylene blue appeared to be the most effective in reducing methemoglobinemia (figure). Other toxic manifestations of primaquine were not increased by the concurrent administration of these drugs.



Effect of concurrent administration of quinine, chloroquine, and methylene blue on the methemoglobin concentration of white subjects treated with primaquine at various dosages for two weeks.

Ephedrine, theobromine, and aminophylline were used concurrently with primaquine following the observation that intravenous aminophylline relieved abdominal cramps for a short time. Each of these drugs, when administered orally, reduced the incidence and severity of symptoms at the higher dosages of primaquine.

Three subjects were treated with 25 mg. of daraprim® concurrently with primaquine at therapeutic doses for 14 days. No adverse reactions were noted but the series was too small to warrant further conclusions.

TOXICITY OBSERVED DURING LONG-TERM PRIMAQUINE-CHLOROQUINE ADMINISTRATION

To test chronic toxicity of primaquine given concurrently with chloroquine, two groups of 20 normal white men were studied for 52 weeks. The first group received 30 mg. of primaquine plus 300 mg. of chloroquine in a single dose once weekly, simulating the current use of chloroquine in field suppression. The second group received 30 mg. of primaquine and 150 mg. of chloroquine simultaneously in a single dose twice weekly. Abnormalities in the red and white blood cell counts were not observed. One volunteer in each group requested to be withdrawn from the study after six months. Their symptoms were mild, transient abdominal cramps, epigastric distress, and anorexia for one to three days following ingestion of each dose of drug. Symptoms began after the fourth month of participation in the program. None of the other volunteers consistently complained of untoward effects.

An additional 10 volunteers were bitten by infected mosquitoes. On the day following infection, these men began to take single weekly doses of 30 mg. of primaquine concurrently with 300 mg. of chloroquine for a total of 14 weeks. Drug was well tolerated by all.

MISCELLANEOUS OBSERVATIONS

Gastric Secretion.—The effect of primaquine on gastric acidity was studied because the epigastric distress associated with the ingestion of primaquine was relieved by food and, in some respects, was similar to that encountered in patients suffering from peptic ulcer. A series of nocturnal hourly gastric aspirations were done as a control in six normal men, and then identical aspirations were continued during administration of single doses of 30 or 60 mg. of primaquine daily for seven days. Variations of free or total acidity were within the limits of control observations.

Liver Function.—Evidence of liver damage was not obtained in liver function studies performed on 57 men receiving 15, 22.5, 30, and 120 mg. of primaquine alone or concurrently with quinine or chloroquine. Three patients hospitalized with infectious hepatitis were given 30 mg. of primaquine for 10, 14, and 18 days. All patients improved; primaquine did not appear to affect the clinical course.

Comparison of Toxicity of Primaquine Given in Single and Divided Dosages Daily.—No difference could be distinguished between the toxic manifestations of primaquine at therapeutic doses administered once a day and the same total dosage administered in three or six equal parts. Abdominal symptoms were of greater incidence and severity when primaquine was administered in single daily doses of more than 30 mg. than they were when the dose was divided. It was impossible to administer single doses of 120 mg. because of the severity of the gastrointestinal symptoms.

Effect of Meals.—The ingestion of primaquine at meal time alleviated abdominal distress even in subjects receiving high dosages.

COMPARISON OF THE RELATIVE TOXICITY OF PAMAQUINE AND PRIMAQUINE

The greater curative effect of primaquine compared to that of pamaquine logically led to a more intensive study at lower doses of the former and higher doses of the latter drug. This discrepancy prevented precise evaluation of their relative toxicity where differences between the two drugs were either not great or not consistent at all dosages.

When primaquine or pamaquine was administered alone or concurrently with quinine the abdominal symptoms were of approximately the same order of magnitude and incidence for both drugs at 30 mg. per day or less. It was not necessary to discontinue drug administration to any subject receiving primaquine in doses below 120 mg., whereas, three subjects receiving 63 mg. of pamaquine daily could not complete the course (tables 3 and 4).

Abnormal laboratory findings after primaquine was administered differed from those encountered during administration of pamaquine as follows: (1) Methemoglobinemia at all dosages was greater with primaquine than with pamaquine. Quinine did not diminish the methemoglobinemia produced by pamaquine. (2) Plasma primaquine concentrations were lower than plasma concentrations of pamaquine at similar dosage schedules. (3) The hemolytic effect of primaquine seemed to be slightly less than that of pamaquine. Two men receiving 15 mg., and four receiving 63 mg. of pamaquine concurrently with quinine lost between 2 and 3 gm. per 100 cc. of hemoglobin. This degree of hemoglobin loss was not observed in any person receiving primaquine at any dose, alone or with a suppressive drug, but some of the groups studied were small. (4) Abnormalities in the white blood cell count occurred less frequently during the administration of primaquine.

DISCUSSION

Toxic symptoms and abnormal laboratory findings resulting from primaquine may be divided into two groups: first, those that are not dangerous but may be annoying; second, those that may constitute a serious threat to life, examples of which are severe neutropenia and acute hemolytic crisis.

The toxic symptoms observed in this study were anorexia, nausea, epigastric distress, abdominal cramps, and occasionally, vomiting, vague chest pain, and weakness. However, symptoms were not present at 15 mg., and symptoms at 30 mg. were mild. At high dosages, weakness was common. In dosages of less than 120 mg. per day, the cramps were not severe enough to warrant discontinuation of drug in any case.

In some subjects, particularly those with mild abdominal cramps, symptoms disappeared after the first week of treatment, while in other persons with more severe cramps, the symptoms increased during the two weeks of drug administration and continued a few days after treatment. Rarely, symptoms of anorexia and mild abdominal cramps persisted up to two weeks after completion of treatment.

TABLE 3.—*Toxicity of Primaquine Administered for Fourteen Days*

Daily Dose of Primaquine, Mg. of Base	Number of Subjects Treated	Number of Subjects Having Abdominal Symptoms*					Plasma Primaquine Concentration, Gamma per Liter		Methemoglobin,† % of Total Hemoglobin			Number of Subjects with a Change in Hemoglobin		Number of Subjects with a Change in White Blood Count‡	
		0	1+	2+	3+	4+	Mean	Range	Mean	Standard Deviation	Range	Change Within ± 1 Gm. per 100 Cc.	Decrease of 1.1 to 2 Gm. per 100 Cc.	Leukocytosis 10,000-17,000	Leukopenia 2,500-5,000
TREATMENT DURING CLINICAL MALARIA															
Primaquine Alone															
15	10	10	0	0	0	0	....	....	6.1	1.74	3.5- 9.1	9	1	0	0
22.5	16	15	0	1	0	0	6.4	4-10	10.2	3.64	6.0-17.5	16	0	1	0
30	24	23	1	0	0	0	16.0	9-23	11.2	3.57	7.4-18.0	24	0	2	0
60	3	2	1	0	0	0	....	....	14.7	4.21	10.5-21.0	3	0	0	0
120	5	0	3	2	0	0	210.0	144-308	20.1	1.15	18.3-21.7	5	0	0	1
Primaquine Concurrently with 2 Gm. of Quinine Sulfate Daily															
15	33	31	2	0	0	0	....	....	6.1	2.89	2.2-12.4	33	0	3	0
22 5	41	35	3	3	0	0	6.7	2-25	6.8	2.12	1.9-12.2	41	0	11	0
30	8	5	1	2	0	0	11.0	7-14	8.4	2.39	4.5-12.1	8	0	0	0
60	4	0	1	2	1	0	37.0	21-58	8.3	0.96	6.8- 9.2	4	0	1	0
120	6	0	1	1	3	1	102.0	74-132	9.9	3.29	5.5-15.5	4	2	0	1
Primaquine Concurrently with 1 Gm. of Chloroquine Base in 1st 24 Hours															
15	24	24	0	0	0	0	....	....	7.8	2.44	3.3-12.0	24	0	1	0
30	28	26	1	1	0	0	....	....	7.9	4.01	2.5-19.9	27	1	0	0
TREATMENT IN ABSENCE OF CLINICAL MALARIA															
Primaquine Alone															
30	31	28	2	1	0	0	....	....	7.9	3.09	3.5-18.8	23	8	4	0
Primaquine Concurrently with 1 Gm. of Chloroquine Base in 1st 24 Hours															
30	123	110	11	2	0	0	....	....	7.4	2.96	1.6-17.5	100	23	2	0

* Although anorexia was the most common symptom, abdominal cramps most accurately reflected the severity and therefore were chosen for quantitative evaluation, as follows: 0 = no symptoms; 1+ = mild, transient abdominal cramps; 2+ = moderate, repeated abdominal cramps; 3+ = severe, persistent abdominal cramps; 4+ = intolerably severe abdominal cramps.

† The methemoglobin values tabulated are the average of the last five days of drug administration. The mean methemoglobin concentration of 196 normal subjects was 1.8% of the total hemoglobin; standard deviation was 1.10 and the standard error of the mean 0.08.

‡ The incidence of leukocytosis during drug administration was probably lower than that expressed in the table because all cases except those unequivocally caused by acute intercurrent infection have been included.

TABLE 4.—*Toxicity of Pamaquine Administered for Fourteen Days*

Daily Dose of Pamaquine, Mg. of Base	Number of Subjects Treated	Number of Subjects Having Abdominal Symptoms*					Plasma Pamaquine Concentration, Gamma per Liter		Methemoglobin,† % of Total Hemoglobin			Number of Subjects with a Change in Hemoglobin			Number of Subjects with a Change in White Blood Count‡	
		0	1+	2+	3+	4+	Mean	Range	Mean	Standard Deviation	Range	Change Within ± 1.0 Gm. per 100 Cc.	Decrease of 1.1 to 2.0 Gm. per 100 Cc.	Decrease of 2.1 to 3.0 Gm. per 100 Cc.	Leukocytosis 10,000-23,000	Leukopenia 3,300-4,500
TREATMENT DURING CLINICAL MALARIA																
Pamaquine Alone																
31.5	12	11	1	0	0	0	40.1	16-124	4.4	3.68	2.1-15.8	11	1	0	8	0
63	6	2	2	2	0	0	105.8	78-145	12.8	4.08	7.2-21.4	6	0	0	2	1
Pamaquine Concurrently with 2 Gm. of Quinine Sulfate Daily																
15	10	7	3	0	0	0	23.6	17- 33	2.5	0.69	1.3- 3.6	7	1	2	7	3
31.5	27	17	6	4	0	0	62.9	21-135	4.5	2.09	1.7- 9.2	20	7	0	14	0
63	76	25	18	19	11	3	111.1	27-253	10.7	4.88	2.9-24.0	68	4	4	22	4

* Although anorexia was the most common symptom, abdominal cramps most accurately reflected the severity and therefore were chosen for quantitative evaluation, as follows: 0 = no symptoms; 1+ = mild, transient abdominal cramps; 2+ = moderate, repeated abdominal cramps; 3+ = severe, persistent abdominal cramps; 4+ = intolerably severe abdominal cramps.

† The methemoglobin values tabulated are the average of the last five days of drug administration. The mean methemoglobin concentration of 196 normal subjects was 1.8% of the total hemoglobin; standard deviation was 1.10 and the standard error of the mean 0.08.

‡ The incidence of leukocytosis during drug administration was probably lower than that expressed in the table because all cases except those unequivocally caused by acute intercurrent infection have been included.

In this study, all cases of leukocytosis not clearly caused by intercurrent disease have been included to avoid underestimation of the toxic effect of primaquine. While the frequency of leukocytosis may thereby have been exaggerated, it seems probable that primaquine, at therapeutic dosages, produces a mild stimulation of the bone marrow: at very high doses, depression may result. Though leukocytosis is not dangerous, it may complicate diagnostic problems involving intercurrent infections or the evaluation of acute abdominal pain. Severe neutropenia has not been observed within the therapeutic range in normal persons. However, primaquine should not be given in dosages greater than 15 mg. per day to patients having concurrent diseases that are characterized by bone marrow depression. Brennecke and coworkers[15] have found that arthritic patients exhibiting leukopenia have increased susceptibility to granulocytopenia during primaquine therapy.

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[15] Brennecke, F. E.; Alving, A. S.; Arnold, J.; Bergenstal, D. M., and DeWind, L. T.: A Preliminary Report on the Effect of Certain 8-aminoquinolines in the Treatment of Rheumatoid Arthritis, J. Lab. and Clin. Med. 38: 795-796 (Nov.) 1951.

Although mild decrease in hemoglobin occasionally has occurred at both high and low doses, severe hemolytic anemia has not been encountered during this study involving 699 white adults receiving primaquine in dosages as high as 240 mg. daily. Severe hemolytic anemia, however, has been observed in Negroes after dosages as low as 30 mg.[16] Because of the potential hemolytic activity of the drug, primaquine should not be administered to persons having complicating diseases characterized by a hemolytic tendency.

Primaquine, at 15 mg. daily, can be administered safely for two weeks without special medical supervision. At 30 mg. daily, primaquine may occasionally produce minor symptoms but can be safely administered to white patients under observation on an out-patient basis. At higher dosage schedules, hospitalization is desirable.

Quinacrine (atabrine®) potentiates the toxicity of pamaquine[17] and pentaquine.[18] Until the effect of quinacrine on primaquine toxicity has been determined, it would seem inadvisable to institute primaquine treatment until several weeks have elapsed following termination of treatment with this suppressive agent. Suppressive agents, at present, should be restricted to quinine and chloroquine, of which the latter is the drug of choice.

A comparison of primaquine and pamaquine can be complete only if the toxicity of the two drugs is correlated with their relative curative action on an equal weight basis; in this respect primaquine is at least four times as effective.

CONCLUSIONS

Primaquine has been administered to 699 adult white subjects in a dosage range of 10 to 240 mg. per day. On the basis of symptoms and laboratory findings it is concluded that, at dosages of 15 mg., primaquine may be safely administered daily for two weeks without special medical supervision. Adult white patients may be followed on an ambulatory basis when receiving doses as high as 30 mg. per day. If it is ever used at higher dosages or administered to patients having diseases known to affect the bone marrow or blood, primaquine should be given only during hospital supervision.

Weekly and semiweekly administration of primaquine for as long as 52 weeks in doses of 30 mg. to 50 subjects did not reveal dangerous toxicity when combined with 0.3 gm. of chloroquine. (All dosages have been expressed in terms of base weight.)

[16] Hockwald, R. S.; Arnold, J.; Clayman, C. B., and Alving, A. S.: Toxicity of Primaquine in Negroes, J. A. M. A., this issue, p. 1568.

[17] Earle, D. P., Jr.; Bigelow, F. S.; Zubrod, C. G., and Kane, C. A.: Studies on Chemotherapy of the Human Malarias: IX. Effect of Pamaquine on the Blood Cells of Man, J. Clin. Investigation **27**: 121-129 (May) 1948.

[18] Atchley, J. A., and others: Reactions Observed During Treatment with Pentaquine, Administered with Quinacrine (Atabrine®), Metachloridine (S. N. 11,437), and with Sulfadiazine, J. Nat. Malaria Soc. **7**: 118-124 (June) 1948.

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4. TOXICITY OF PRIMAQUINE IN NEGROES

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Capt. Charles B. Clayman, (MC), United States Army
Alf S. Alving, M.D., Chicago

Hemolytic reactions during antimalarial therapy have proved to be a limiting factor in the use of 8-aminoquinolines for the cure of vivax malaria. The most severe type of reaction is clinically identical to blackwater fever, and is characterized by massive, explosive, intravascular hemolysis with hemoglobinemia and hemoglobinuria, which constitutes a medical emergency necessitating immediate transfusions. Acute hemolysis has been observed during the therapeutic use of a number of 8-aminoquinolines, for example, pamaquine,[1] pentaquine,[2] and isopentaquine.[3] The incidence of hemolytic reactions is higher in the dark-skinned races. According to Earle and co-workers,[1] 5 to 10% of Negroes contract acute hemolysis when given 30 mg. or more of pamaquine daily. Only 1% of white subjects studied by him contracted hemolysis which, however, was not acute.

PROCEDURES

Dosage Schedules.—Primaquine[4] 8-(4-amino-1-methylbutylamino)-6-methoxy quinoline, 30 mg. of base daily, was administered orally in single or divided doses to 110 normal male Negro volunteers at Stateville Penitentiary. The men ranged in weight from 130 to 240 lb.; the average being 165 lb. Primaquine was given, alone or concurrently with quinine or chloroquine, for 14 days. This dose is slightly larger than that usually recommended for therapy.

The following four schedules were used:

1. Fifty men were given 30 mg. of primaquine base in a single daily dose for 14 days.
2. Twenty-five men were given 30 mg. of primaquine base daily concurrently with 2 gm. of quinine sulfate (1.64 gm. of base) daily for 14 days (both drugs were administered in six divided doses).
3. Twenty-five men were given 30 mg. of primaquine base in a single daily dose for 14 days concurrently with single doses of chloroquine base, 0.6 gm. on the first day and 0.45 gm. on the second day.
4. Ten men were given 30 mg. of primaquine base for 14 days concurrently with chloroquine base, 0.6 gm. on the first day, 0.45 gm. on the second day, and 0.3 gm. on each of the next 12 days. Both drugs were administered in single daily doses.

The men contracting severe anemia while receiving the 30 mg. dosage subsequently were given 15 mg. of primaquine base orally once daily for 14 days to determine whether the reduced dosage would also produce hemolysis.

Later, the same subjects were given pamaquine orally, 30 mg. of base once daily, to compare the hemolysis with that produced by 30 mg. of primaquine base.

Primaquine, 15 mg. of base, was administered orally, once daily, for 14 days to a second group of 50 normal male Negro volunteers. These men ranged in weight from 127 to 211 lb., the average being 161 lb. The primaquine was given alone or concurrently with chloroquine, 0.6 gm. of base on the first day and 0.45 gm. on the second day. This dose of primaquine has been adopted for routine interim treatment of malaria in Korean veterans.[5]

Special Observations and Laboratory Studies.—Each man was questioned daily to detect unusual symptoms. Methemoglobin and hemoglobin levels were determined daily, three and seven days after drug administration was completed. A white blood cell count was taken initially, every two days while the drug was being given, and three and seven days afterward. Differential white counts were performed initially and thereafter if any significant change in the total white count occurred. A urinalysis was performed before the drug was given, and was later repeated if a marked drop in hemoglobin occurred. The following liver function tests were performed on 32 of the men receiving the 30 mg. dosage: total plasma proteins,[6] 30-minute bromsulphalein retention test,[7] serum bilirubin (direct and indirect),[8] thymol turbidity.[9] Red blood cells from men who developed severe anemia were tested for osmotic fragility and sickling.[10]

Severity of hemolytic reactions was evaluated in the following manner:

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[1] Earle, D. P., Jr.; Bigelow, F. S.; *Zubrod*, C. B., and Kane, C. A.: Studies on Chemotherapy of Human Malarias: IX. Effect of Pamaquine on the Blood Cells of Man, J. Clin. Investigation **27**: 121-129 (May) 1948.

[2] Coatney, G. R., and others: Studies in Human Malaria: XXVII. Observations on Use of Pentaquine in *Prevention* and Treatment of Chesson Strain Vivax Malaria, J. Nat. Malaria Soc. **9**: 222-232 (Sept.) 1950. Atchley, J. A., and others: Reactions Observed During Treatment with Pentaquine, Administered with Quinacrine (Atabrine®), Metachloridine (S. N. 11, 437), and with Sulfadiazine, J. Nat. Malaria Soc. **7**: 118-124 (June) 1948.

[3] Brown, J. B.: personal communication to the authors.

[4] The authors are indebted to Dr. Marlin Leffler of the Abbott Laboratories, North Chicago, Illinois; Dr. C. E. Roach of Eli Lilly and Company, Indianapolis, and Dr. Justus B. Rice of Winthrop-Stearns, Inc., New York, for providing supplies of primaquine.

[5] Returning Korean Veterans to Receive New Anti-malarial Drug, Department of the Army, Office of the Surgeon General, Technical Information Office, Technical Release dated Oct. 19, 1951.

[6] Phillips, R. A.; Van Slyke, D. D.; Dole, V. P.; Emerson, K., Jr.; Hamilton, P. B., and Archibald, R. M.: Copper Sulfate Method for Measuring Specific Gravities of Whole Blood and Plasma, Bull. U. S. Army Med. Dept. 71 (Dec.), 1943, pp. 66-83.

[7] Rosenthal, S. M., and White, E. C.: Clinical Application of Bromsulphalein Test for Hepatic Function, J. A. M. A. **84**: 1112-1114 (April 11) 1925.

[8] Malloy, H. T., and Evelyn, K. A.: Determination of Bilirubin with the Photoelectric Colorimeter, J. Biol. Chem. **119**: 481-490 (July) 1937.

[9] Shank, R. E., and Hoagland, C. L.: Modified Method for Quantitative Determination of Thymol Turbidity Reaction of Serum, J. Biol. Chem. **162**: 133-138 (Jan.) 1946.

[10] Wintrobe, M. M.: Clinical Hematology, Philadelphia, Lea and Febiger, 1942, pp. 82 and 407.

No anemia: A variation in hemoglobin from the initial determination of 1 gm. per 100 cc. or less. (Some men had a small rise in hemoglobin during the 14 days.)

Mild anemia: A decrease in hemoglobin of 1.1 to 4 gm. per 100 cc. during 14 days.

Moderate anemia: A decrease in hemoglobin of more than 4 gm. per 100 cc. during 14 days.

Severe anemia: A decrease in hemoglobin which necessitated discontinuation of the drug before 14 days.

Acute intravascular hemolysis: Explosive hemolysis with gross hemoglobinemia and hemoglobinuria, resembling malarial blackwater fever, frequently complicated by acute renal shutdown.

RESULTS

Group Receiving 30 Mg. of Base Daily.—One hundred ten Negroes received 30 mg. of primaquine daily, 105 of whom were able to complete the 14 day schedule. No acute intravascular hemolysis occurred, but mild anemia developed in 17 men. For these 17 men, the mean decrease in hemoglobin was 1.8 gm. per 100 cc. and the standard deviation was ±0.72. Five men contracted severe anemia, which necessitated discontinuation of the drug after four to nine days (fig. 1-5). In the latter group, hemolysis commenced after three to five days. These five men had elevated serum bilirubin and four of them passed dark-colored urine during one or two days before the drug was stopped. The urine of one of the four was burgundy-colored. This man received quinine concurrently with the primaquine (fig. 5). None of the men had red-colored plasma. Red cells from the five men with severe anemia were tested for osmotic fragility and sickling. The results were normal. There were no abnormalities in the other laboratory examinations of any of the 110 men studied, and none of the men had subjective complaints of clinical importance. Leukopenia did not develop. The mean change in methemoglobin concentration was +0.4 gm. per 100 cc. There was no correlation between the degree of methemoglobinemia and hemolysis.

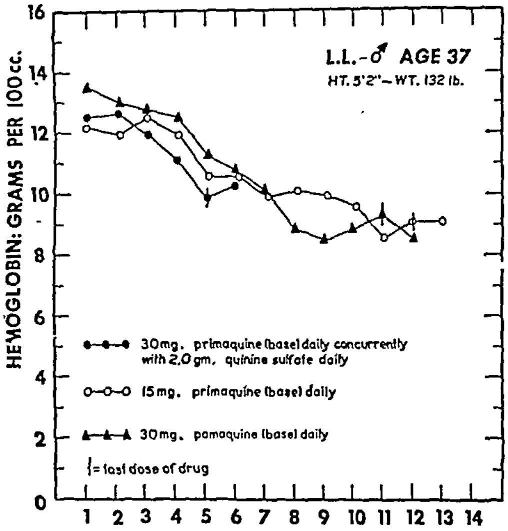

Fig. 1.—Comparison of the hemolytic effects of primaquine and pamaquine in one of five Negroes who had severe anemia. This man had the least severe reaction, and was the only one who never passed grossly discolored urine.

After complete recovery, the five men whose medication had been stopped because of severe anemia were given 15 mg. of base, one-half the original dose of primaquine, daily for 14 days to determine if the reduced dosage would also produce hemolysis. Four of the men contracted mild anemia (fig. 1, 3, 4, 5) more slowly than during the higher dosage of drug. One man did not contract anemia (fig. 2). In a single subject, the drug was discontinued before 14 days, and this was probably done unnecessarily (fig. 1).

Later these five men were given pamaquine, 30 mg. of base in a single dose daily. All five became severely anemic which necessitated discontinuation of the drug (fig. 1-5). The urine of four of the men became dark-colored. In only one subject was the urine benzidine positive; he was the man whose urine had become burgundy-colored while he was on the 30 mg. dosage of primaquine (fig. 5). Methemoglobin formation was slight and leukopenia did not develop. None of the men had red-colored plasma.

All men who had severe anemia recovered spontaneously in 2 to 3 weeks; transfusions were not necessary.

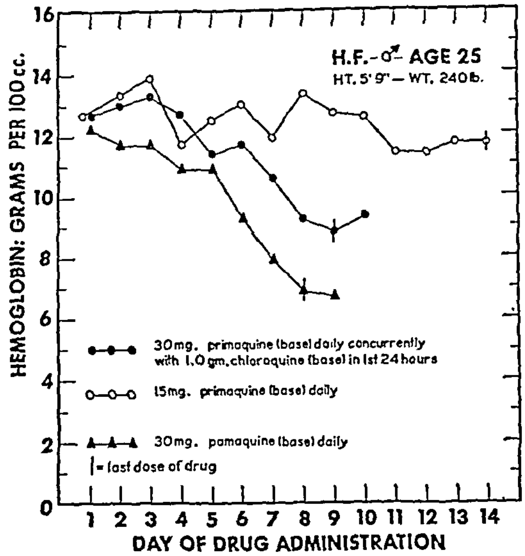

Fig. 2.—Comparison of the hemolytic effects of primaquine and pamaquine in one of five Negroes who had severe anemia. This man had a moderately severe reaction.

Group Receiving 15 Mg. of Base Daily.—In another group of 50 men who received 15 mg. of primaquine base once a day for 14 days, 12 had mild anemia. In these 12 men the mean decrease in hemoglobin was 2 gm. per 100 cc. and the standard deviation was ±0.75. Anemia of any greater degree was not produced. The mean change in methemoglobin concentration was +0.2 gm. per 100 cc. All men were able to complete the 14 day course of drug administration. There were no abnormal findings in the other laboratory examinations of the 50 men studied, and none of the men had complaints of clinical importance.

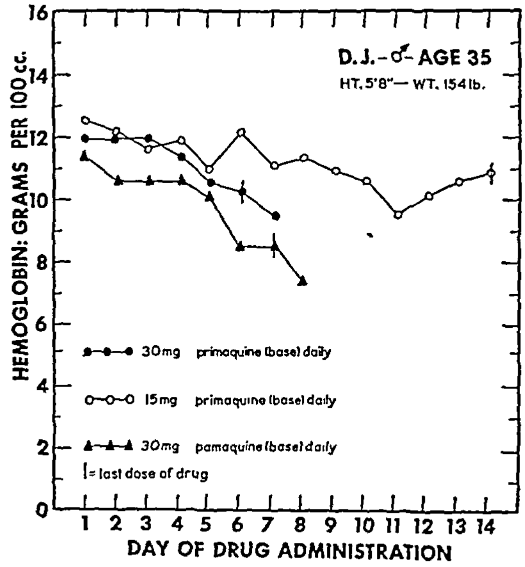

Fig. 3.—Comparison of the hemolytic effects of primaquine and pamaquine in one of five Negroes who had severe anemia. This man had a moderately severe reaction.

DISCUSSION

Primaquine, 30 mg. of base, was administered daily to 110 Negro volunteers at the Illinois State Penitentiary (Stateville).

Severe anemia developed in five of the men, which necessitated discontinuation of the drug before 14 days of administration was completed. The five men were later given pamaquine, 30 mg. of base daily, and anemia of a similar magnitude developed.

Within the group of five volunteers who had severe reactions, it is possible to subdivide the degree of hemolysis further. In the man having the least severe reaction (fig. 1), the urine remained of normal color, although the serum bilirubin was elevated. It was subsequently noted during administration of 15 mg. of primaquine that, after an initial drop, the hemoglobin level became relatively stabilized. In retrospect, it therefore seems that medication was stopped during both periods of primaquine administration before it was urgent. This opinion is further strengthened by the fact that this man was later able to tolerate pamaquine in daily doses of 30 mg. for 11 days, even though the initial rate of fall during administration of pamaquine was almost identical to the decrease when 30 mg. of primaquine had been given to him. The other four men demonstrated more rapid hemolysis (fig. 2-5) and in one of them the reaction nearly approached the severity of blackwater fever (fig. 5). He passed burgundy-colored urine during administration of primaquine and brown urine, which was benzidine positive, during administration of pamaquine.

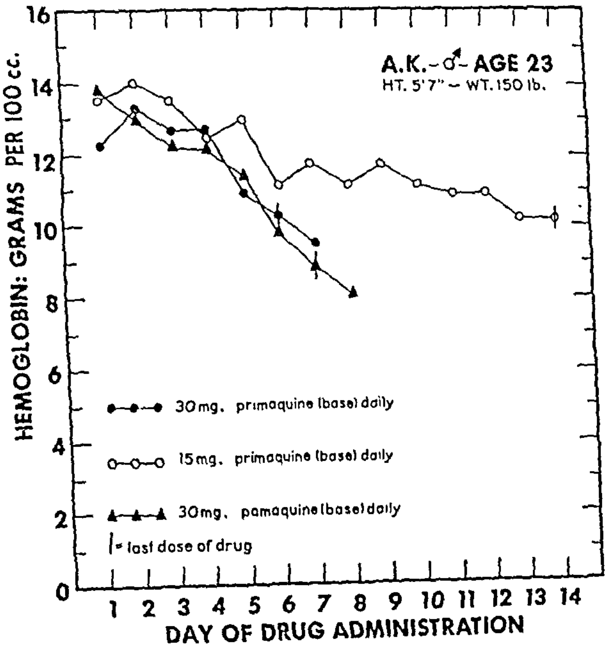

Fig. 4.—Comparison of the hemolytic effects of primaquine and pamaquine in one of five Negroes who had severe anemia. This man had a moderately severe reaction.

Although all of the men had highly colored (yellow to brown) plasma, none had red-colored plasma; and the hemolysis was not explosive as in malarial blackwater fever.

Fifteen milligrams of primaquine base was well tolerated by the five men in whom severe anemia developed and by 50 other Negro volunteers.

These studies, while in progress, served in part as a basis for choosing 15 mg. of primaquine for use in extensive field trials to cure vivax malaria acquired in Korea. The relative safety of administering this dose to Negroes was confirmed by studies on 52 Negroes from the enlisted personnel at Fort Knox, Kentucky, with the cooperation of the United States Army,[11] by studies on 268 Negro veterans from Korea on shipboard between Japan and the United States with the cooperation of the United States Army and Navy and the Military Sea Transport Service,[12] and by studies on 317 Nicaraguan natives who had a mixture of Indian and Negro bloods.[13]

Blake [14] reported that pamaquine and quinine act synergistically to cause increased hemoglobin catabolism. In these studies, 30 mg. of primaquine base was administered alone or with quinine or chloroquine. Each series, however, was too small to determine whether either of these suppressive drugs increased the hemolytic tendency of primaquine.[15]

There was no correlation between methemoglobin formation and hemolysis.

Except for the hemolysis, the men tested showed no evidence of toxicity, and their status during drug administration was similar to that of light-skinned Caucasians given identical doses.

The tendency to hemolytic reactions during administration of primaquine is probably not limited to Negroes, but represents an intrinsic danger to several racial groups. More deeply pigmented persons may have a special predilection to hemolysis during treatment with primaquine, as they have during administration of pamaquine.[1] Taggart [16] has observed a hemolytic reaction of moderate severity in a Caucasian, a Hindu, who received 30 mg. of primaquine base daily concurrently with quinine.

Because of reports that quinacrine increases the toxicity of 8-aminoquinolines,[17] its concurrent use with primaquine should be avoided until more information has been accumulated.

Primaquine should not be used to cure vivax malaria in persons having a concurrent disease characterized by a hemolytic tendency. Thaeler [13] has found, however, that primaquine may be administered safely to mestizoes (persons having a mixture of white, Indian, and Negro blood) in the presence of severe anemia caused by accompanying malnutrition and infestation with intestinal parasites.

CONCLUSIONS

1. Primaquine in doses of 30 mg. of base is *too toxic to be used in Negroes without close medical supervision.* Although subjective complaints were negligible, 5% of 110 volunteer subjects contracted severe and progressive anemia. The anemia was of the same order of magnitude as that developing after th administration of pamaquine, 30 mg. of base daily.

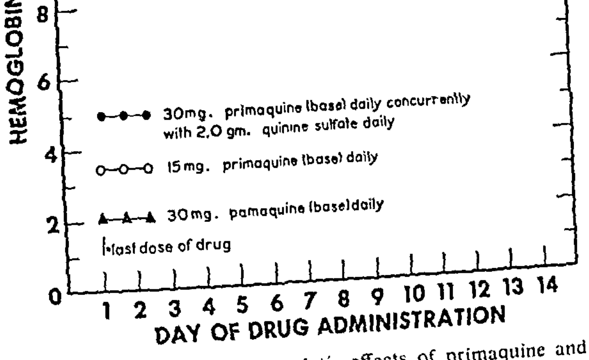

Fig. 5.—Comparison of the hemolytic effects of primaquine and pama quine in one of five Negroes who had severe anemia. This man had th most severe reaction.

2. Studies herein reported indicate that primaquine, 15 mg. c base, can be given safely to adult Negroes for 14 days withou special medical supervision. These findings have been confirme in expanded field studies in military and civilian installation

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11. Radke, R. A.; Polskin, L. J.; Alving, A. S., and Arnold, J.: Unpublished data.

12. Alving, A. S.; Arnold, J., and Robinson, D H.: Unpublished data.

13. Thaeler, A. D., Jr.; Arnold, J., and Alving, A. S.: Field Studies of Primaquine in Nicaragua, presented at the Annual Meetings of the National Malaria Society, Chicago, Nov. 14-17, 1951.

14. Blake, W. D.: Methemalbumin: II. Effect of Pamaquine and Quinine in Pathways of Hemoglobin Metabolism, J. Clin. Investigation **27**: 144-150 (May) 1948.

15. Four of the five men who contracted hemolysis with each program of drug administration, including 30 mg. of pamaquine base alone, were later given pamaquine, 27 mg. of base, concurrently with quinine, 1.64 gm. of base, daily to determine if there was any increased hemolysis. These men had severe hemolysis but the rate was approximately the same as that developing with pamaquine alone. The gastrointestinal complaints were greatly increased when quinine was given, and the hemolysis continued for a longer period after the drug was discontinued.

16. Taggart, J. V.: Personal communication to the authors.

17. Findlay, G. M.: Recent Advances in Chemotherapy (Volume II), Philadelphia, The Blakiston Company, 1951, pp. 226-227.

COUNCIL ON FOODS AND NUTRITION

ACCEPTED FOODS

The following products have been accepted as conforming to the rules of the Council.

JAMES R. WILSON, M.D., *Secretary.*

The Bib Corporation, Lakeland, Fla.

Bib Brand Orange-Apricot Juice for Babies.

Ingredients: Single strength orange juice prepared from Florida oranges specially selected for their natural high ascorbic acid content; fresh apricot puree processed from mature fresh Blenheim—variety apricots and minimal addition of dextrose for standardizing flavor to maintain the brix solids in this product at approximately 20°.

The percentage of apricot and orange used in this product may vary slightly from time to time, depending upon the natural ascorbic acid in the oranges used, so as to maintain a minimum of no less than 30 mg./100 cc. in each 4-ounce can when packed, as well as to maintain uniformity of flavor.

Use.—For use where babies and young children do not take orange juice or in situations where flavor changes might be desirable.

Bib Brand Prune-Orange Juice for Babies.

Ingredients: Prune juice 20 to 21° solids is processed from prune puree produced from Santa Clara variety prunes; Orange juice—single strength prepared from Florida oranges specially selected for their natural high ascorbic acid content, and a minimal addition of dextrose for standardizing flavor to maintain the brix solids in this product at approximately 20°.

The percentage of prune juice and orange juice used in this product may vary slightly from time to time, depending upon the natural ascorbic acid in the oranges used, so as to maintain a minimum of no less than 30 mg./100 cc. of natural ascorbic acid in each 4-ounce can when packed, as well as to maintain uniformity of flavor.

Use.—For use as a valuable supplementary source of natural ascorbic acid for infants and young children when a change from orange juice is desired or prescribed by the doctor for its mild laxative effect.

The Baker Laboratories, Inc., Cleveland, Ohio

Varamel.

Ingredients: A highly nutritious formula base made from grade A (U.S.P.H.S.) cows' milk in which the milk fat has been replaced by coconut oil, destearinated beef fat, and corn oil; with the addition of Vitamin A palmitate, activated 7-dehydrocholesterol, thiamine, niacinamide, and iron ammonium citrate.

Analysis (submitted by manufacturer)—Total solids 20%, moisture 80%, ash 1.2%, fat 5.8%, protein 4.9%, carbohydrate 8.3%, calcium 0.19%, phosphorus 0.15%, iron 0.0016%.

Vitamins	Amounts per Quart*
Vitamin A	2500 U.S.P. Units
Vitamin D	800 U.S.P. Units
Vitamin C	none
Thiamine	0.6 mg.
Niacin	5.0 mg.
Riboflavin	1.2 mg.

*When diluted with an equal part of water.

Calories.—32 per fluid ounce.

Use.—For use in infant feeding.

Winthrop-Stearns, Inc., New York

Essenamine® Powder.

Ingredients: Lactalbumin and carboxymethylcellulose.

Analysis (submitted by manufacturer).—Total solids 96.0%, moisture 4.0%, protein (N x 6.62) 90%, sodium 0.23%, chlorides 0.3%, ash 2.0%.

Calories.—4.0 per gram; 113.4 per ounce.

Use.—For conditions that require an increased intake of protein.

Essenamine® Compound Powder—Vanillin Flavored.

Ingredients: Essenamine® Powder; carob gum, powdered; sucrose, U.S.P. powder; saccharin sodium, U.S.P. powder; vanillin, U.S.P., and coumarin, N.F.

Analysis (submitted by manufacturer).—Total solids 97.0%, moisture 3.0%, ash 1.4%, protein (N x 6.62) 63%, carbohydrate (from cane sugar) 25.0%, sodium 0.16%, chlorides 0.2%.

Calories.—3.5 per gram; 99.2 per ounce.

Use.—For conditions that require an increased intake of protein.

Essenamine® Compound Granules—Vanillin Flavored.

Ingredients: Essenamine® Powder; dextrose, anhydrous; vanillin, U.S.P. and coumarin, N.F.

Analysis (submitted by manufacturer).—Total solids 97.0%, moisture, 3.0%, ash 1.4%, protein (N x 6.62) 63%, carbohydrate (dextrose) 30.0%, sodium 0.16%, chlorides 0.2%.

Calories.—4 per gram; 113.4 per ounce.

Use.—For conditions that require an increased intake of protein.

Beech-Nut Packing Company, Canajoharie, N. Y.

Beech-Nut Junior Creamed Spinach (Chopped).

Ingredients: Spinach, milk, cornstarch, cream, salt, dehydrated onions, and sufficient water for preparation.

Analysis (submitted by manufacturer).—Total solids 9.9%, moisture 90.1%, ash 1.25%, fat 1.69%, protein (N x 6.25) 2.06%, crude fiber 0.44%, carbohydrates other than crude fiber (by difference) 4.1%.

Vitamins and Minerals	Per 100 Gm.
Vitamin A	2,830 I.U.
Thiamine Hydrochloride	0.01 mg.
Riboflavin	0.12 mg.
Nicotinic acid	0.25 mg.
Ascorbic acid	5.7 mg.
Calcium	110 mg.
Phosphorus	44 mg.
Total iron	1.6 mg.
Available iron	1.3 mg.
Copper	0.05 mg.

Calories.—0.45 per gram; 12.8 per ounce.

Use.—For use in the feeding of older infants and young children.

The Cream of Wheat Corporation, Minneapolis.

Zing! Wheat Germ.

Ingredients: Wheat germ and malt syrup.

Analysis (submitted by manufacturer).—Total solids 93.3% to 92.0%, moisture 6.7% to 8.0%, ash 4.2% to 4.8%, fat (ether extract) 10.6% to 13.4%, protein 28.8% to 33.4%.

Vitamins and Minerals	Mg./100 Gm.
Thiamine	2.2—3.4
Riboflavin	0.8
Nicotinic acid	4.4
Pantothenic acid	1.6
Tocopherols	44.0
Phosphorus	1,128
Iron	7.8

Calories.—4.02 per gram; 114 per ounce.

Use.—A food supplement providing the nutrients of wheat germ as indicated above. May be eaten alone as cereal or in combination with other foods.

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SOCIALIZED MEDICINE— THE INTERNATIONAL WAY

During the past several years the American medical profession, together with thousands of national, state, and local organizations, representing a cross section of all Americans, have communicated their definite opposition to "socialized medicine" to their state and federal Senators and Representatives. As a result it has become apparent to members of state legislatures and the Congress that compulsory health insurance would not only destroy the present high quality of medical care in this country but that it would endanger the basic American freedoms.

Although the battle against the proponents of political medicine has been won, at least temporarily, in our state legislatures and in the Congress, we are in danger of losing the war through an international approach. In our enthusiasm to espouse such principles as "fundamental human rights," "social progress," and "international peace and security," we have aided and abetted the activities of these forces. Through the wholesale approval of treaties, conventions, and executive agreements, our international representatives have placed not only American medicine but our national sovereignty and our Constitution in jeopardy.

Socialism by treaty is now a greater threat than socialism by domestic legislation, principally because the possibility of political and economic regimentation from an external source is not widely recognized. The public is unaware of the dangers inherent in the treaty-making power because, during the first 150 years of our republic, treaties concerned only the relationship between the United States and other sovereign states. Their attempted use to define the relationship between American citizens and their own government is a recent development—a development, however, which can result in the subtle realization of the socialist's dream of cradle-to-the-grave security.

A recent covenant, entitled "Minimum Standards of Social Security," approved by the International Labor Organization in Geneva in June, 1952, envisions government benefits in nine fields of social security—medical care, sickness benefits, unemployment benefits, old age pensions, employment injury benefits, family allowances, maternity benefits, invalid benefits, and survivors' benefits. While the medical benefits in the covenant are carefully distributed through the document, when they are considered together they constitute "socialized medicine."

Statements on the I. L. O. have appeared recently in THE JOURNAL.[1] In this issue (page 1574) are additional statements that should interest all who are interested in the prevention of the deterioration of medical care through domination by the federal government.

PLACEMENT ASSISTANCE ON RELEASE FROM MILITARY SERVICE

The American Medical Association has recently initiated a new program designed to acquaint physicians being released from active military service with existing medical opportunities, and to assist State Selective Service Medical Advisory Committees in replacing deferred physicians classified in priority 1 under the "Doctor Draft Law."

This program, which was inaugurated by the Council on National Emergency Medical Service with the cooperation of the Department of Defense and the Selective Service System, has the enthusiastic endorsement of the state medical societies and the chairmen of Selective Service Medical Advisory Committees.

The names of physicians to be released from service and other necessary information will be supplied to the Council on National Emergency Medical Service by the offices of the three Surgeon Generals. The Council will communicate with each of these physicians to determine in general their post-service plans, their medical qualifications and specialty, as well as the state in which they prefer to work.

The names of physicians interested in receiving additional information will then be broken down by states and sent to State Selective Service Medical Advisory Committees. While these names are being forwarded primarily as names of potential replacements for deferred priority 1 physicians, further action, if any, with respect to them will depend entirely on the wishes and desires of the advisory committees. This same information will be made available to the state medical societies through the Physicians' Placement Service of the Council on Medical Service in accordance with current procedures.

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[1] The World Medical Association, Organization Section, J. A. M. A. **149:** 488 (May 31) 1952. Statement of Dr. Louis H. Bauer Re International Labor Organization and World Medical Association and Resolutions on Amendment to Constitution of the United States (S.J.R. 130), Organization Section, ibid. **149:** 869 (June 28) 1952. Resolutions on International Labor Organization Attempt to Socialize Medicine, ibid., **140:** 938 (July 5) 1952

It is the earnest hope of the Association that the program will be of real assistance to individual physicians and to the state medical societies, and that it will be successful in the satisfactory placement of many physicians upon their release from active duty.

PRIMAQUINE FOR VIVAX MALARIA

Elsewhere in THE JOURNAL (p. 1558) are four reports on malaria and drugs used in control of this disease. These reports provide much practical information for the medical profession and at the same time raise some points not previously appreciated by those not close to this field of investigation. Studies undertaken by the authors of the Council-sponsored reports and some of the results of studies on malaria therapy jointly undertaken by the Medical Research and Development Board of the United States Army, the United States Public Health Service, and the Subcommittee on Malaria of the Committee on Medicine of the National Research Council bring to the physicians of the United States a very practical problem.

When our forces entered Korea in the summer of 1950, chloroquine was administered routinely for the suppression of malaria. Chloroquine is an excellent suppressive drug, but it does not prevent infection with the malaria parasite. In the spring of 1951, when large numbers of troops were returned to the United States and use of suppressive chloroquine was discontinued, attacks of malaria began to occur in Korean returnees and increased in frequency, reaching a peak of more than 600 attacks per week. Acute attacks treated with chloroquine were promptly terminated, but relapses occurred after treatment with the same frequency as in other types of vivax malaria. This prompted the inauguration of a program to evaluate the effectiveness of primaquine, a new 8-aminoquinoline, reported to be superior to pamaquine as a curative agent for vivax malaria. Up to the present time the limited supply of primaquine has necessitated restricting its use to military installations and to other groups included in the studies carried out by the several participant agencies. Because of improved methods of synthesis moderate quantities of the drug will shortly become available. Studies of the toxicity and therapeutic effectiveness of primaquine have been in progress at several military installations. Final evaluation of the therapeutic studies will not be possible until the fall of 1952; however, because the incidence of malaria in Korean returnees is increasing rapidly and because primaquine is now available at military installations, this preliminary summary is presented.

It has been demonstrated that a single dose of 15 mg. of primaquine (base) daily for 14 consecutive days will reduce the relapse rate of Korean malaria to less than 1%. In a control group of more than 450 patients, treated with chloroquine alone, and followed to date, the relapse rate is 30% and is still increasing; in a similar group treated with the same dose of chloroquine, plus primaquine in doses of 15 mg. (base) daily for 14 days, there has been only one relapse.

In a detailed study of toxicity, primaquine was administered to over 300 natives in a jungle area of Nicaragua, in the United States to 1,500 persons at full duty on land, and to 2,700 persons returning from Korea by sea, without evidence of toxicity. In more than 500 patients with malaria who were treated with primaquine in combination with chloroquine, no untoward reactions have been observed. While it is apparent that the toxicity of primaquine in a dosage of 15 mg. (base) per day for 14 days is minimal, it should be pointed out that this agent has all the potential toxicity of pamaquine (hemolytic anemia, bone marrow depression, methemoglobinemia, and abdominal pain), when given at higher doses. However, the maximum tolerated dose of primaquine in white subjects is more than twice the maximum tolerated dose of pamaquine, and the minimum curative dose of primaquine is less than one-fourth that of pamaquine. Like pamaquine, primaquine is more toxic for Negroes than for Caucasians. It has been shown that primaquine, in doses of 30 mg. (base) per day for 14 days, will occasionally produce hemolytic anemia in Negroes. This has not been observed in military installations where dosages of 15 mg. (base) per day have been employed. Data available at this time clearly indicate the following conclusions.

1. The recommended regimen for the treatment of an acute attack is chloroquine, three doses of 0.3 gm. (base) during the first 24 hours, followed by 0.3 gm. (base) daily for two days. Primaquine, in doses of 15 mg. (base) daily, is started simultaneously with chloroquine and continued for 14 consecutive days.

2. Primaquine in doses of 15 mg. (base) per day for 14 days will produce radical cure of Korean vivax malaria, in most persons, by destroying the tissue phases of the parasite. Because primaquine is relatively ineffective in destroying the blood stages of the parasite, it should be used in combination with chloroquine in the treatment of acute attacks of vivax malaria.

3. When primaquine is used at this dosage, no significant toxicity is to be expected in the lightly pigmented races, and it may be administered to the deeply pigmented races under the minimal medical supervision available on troop transport ships. The possibility of a hemolytic reaction should be kept in mind if the patient is deeply pigmented, and the blood hemoglobin concentration determined if extremely dark or brown urine is observed.

ORGANIZATION SECTION

The May 31 issue of THE JOURNAL, *page 489, carried the statement that the World Medical Association had prepared on medical aspects of social security in view of the International Labor Office's proposed international standards for cradle-to-grave social security. The May 31 issue also described the efforts that W. M. A. had made and would make to present these views before this international body with treaty formulation authority.*

The I. L. O. conference was held in June. Despite a vigorous contest over whether the social security standards should be a recommendation or a convention (international treaty) binding on ratifying members, the social security standards were adopted as a convention by the required two-thirds vote, including three of the four American votes.

Membership obligation under the I. L. O. constitution requires that the instrument be submitted by its members, including the United States, to their proper authorities for ratification or rejection and for appropriate legislation. Thus there is a series of questions respecting the international and domestic significance of this new instrument, especially its provisions relating to social security medical programs.

The views of W. M. A. were presented to the Social Security Committee of the I. L. O. The report of that committee to the Plenary Session on the proposed social security standards states:

"The Conference added to the committee as a technical expert without power to vote, Dr. Maystre, a representative of the World Medical Association. Dr. Maystre submitted to the committee a statement of the views of the World Medical Association concerning the provision of health services under social security schemes."

The report also states, "The Committee . . . rejected by a very large majority a proposal to delete Part II altogether . . ." Part II contains the broad medical care social security program. The report further states:

"An employer member submitted a new Article establishing general principles to be observed in the provision of medical care . . . The author explained that this amendment was based on the statement made by the World Medical Association . . . It seemed reasonable that the proposed instrument should contain not only quantitative but also qualitative provisions . . . The Committee, however, by a very large majority . . . did not deem it advisable to do so. . ."

The "employer member" who submitted and fought for both of the above amendments was Leonard J. Calhoun, Washington attorney and social security expert. His observations as to the background of the I. L. O., its actions respecting social security medicine, and the international and domestic significance of the adoption of the new convention are of such interest that these are given below.—ED.

MEDICAL CARE AND THE INTERNATIONAL SOCIAL SECURITY TREATY

Leonard J. Calhoun, Washington, D. C.

In the domestic drive for socialized medicine, defeat of the Wagner-Murray-Dingell bill (the cradle-to-the-grave medical and cash benefits proposal) proved to be only an opening skirmish. The final outcome may be importantly affected by actions taken in far-away Geneva, Switzerland, at the International Labor Conference. This conference has just exercised its authority of formulating international treaties in its adoption of the so-called "Social Security Minimum Standards Convention of 1952."

Minimum Medical Standards.—The principal provisions of the treaty relating to medical care are found in "Part II, Medical Care." The scope of the medical benefits is specified in articles 8, 10 and 12, which read as follows:

"Article 8. The contingencies covered shall include any morbid condition, whatever its cause, and pregnancy and confinement and their consequences."

"Article 10. The benefit shall include at least
- (a) In case of a morbid condition
  - (i) general practitioner care, including domiciliary visiting;
  - (ii) specialist care at hospitals for in-patients and out-patients, and such specialist care as may be available outside hospitals;
  - (iii) the essential pharmaceutical supplies as prescribed by medical or other qualified practitioners; and
  - (iv) hospitalisation where necessary; and
- (b) in case of pregnancy and confinement and their consequences
  - (i) pre-natal, confinement and post-natal care either by medical practitioners or by qualified midwives; and
  - (ii) hospitalisation where necessary."

"Article 12. The benefit . . . shall be throughout the contingency . . . except that, in case of a morbid condition, its duration may be limited to 26 weeks in each case, but . . . provision shall be made to enable the limit to be extended for prescribed diseases recognized as entailing prolonged care."

On first consideration it might seem difficult to formulate any great broadening of the above provisions. The worker block proposed an amendment adding dental care. The American employer representative raised a point of inquiry. The convention was dealing with "minimum standards." If dental care were added, would there be a great difference between the "minimum standards" and the "advanced standards"? This amendment was defeated.

The scope of the minimum standards, covering not only general medical care but eight other programs, was such that the Social Security Committee did not have time to consider the advanced social security standards treaty, and it had to be postponed. The tentative text of the "advanced standards" adds the following to the medical care required under the minimum standards treaty: (1) specialist care without the minimum standards limitation; (2) dental care and supplies; (3) nursing care at home as well as in hospitals; (4) "maintenance in hospitals, convalescent homes, sanatoria or other medical institutions"; (5) "prosthetic appliances, kept in repair, and eyeglasses," in addition to pharmaceuticals; and (6) care of professionals "legally recognized as allied to the medical profession" as well as physicians.

Questions Concerning the I. L. O. Treaty

What is this International Labor Organization? What authority does it have for formulating treaties? Is the United States a member, and did it participate in formulating and vote for adoption of these medical and other social security standards as a treaty? Does this treaty affect us? These are inquiries deserving answers.

Background of I. L. O. Treaty Formulation.—For many decades preceding the formation of I. L. O. in 1919 as an adjunct to the League of Nations, there had been a series of international conferences on labor. These, however, with the exception of an unproductive conference of governments called by Kaiser Wilhelm, had no authority to frame conventions (international treaties). Instead, they were conferences of labor unions and, at times, of employers, who could only pass resolutions and recommendations, which were by and large ignored.

With the establishment of I. L. O. came the authority for framing international labor conventions (treaties). Its unique organization—with 60-odd governments as members but each providing a representative of its employers and a representative of its employees, as well as two government representatives—afforded labor and employer representatives the opportunity of participating in the framing of instruments in the nature of international treaties, binding on all ratifying countries. The United States has been a member since 1934, and reaffirmed its membership after World War II and the broadening of the I. L. O. constitution.

Basis of Social Security Standards Treaty.—Besides what might be classed as normal subject matter for international labor treaties, such as the treatment by countries of seafarers and of non-nationals, matters such as hours of work for women and children have been considered appropriate by conferences preceding I. L. O. and by I. L. O. itself. The general theory has been that treaties were essential if nations were to have humane legislation of this type without suffering the probably catastrophic consequences of loss of foreign markets to competitors with less humane laws.

The present theories supporting the promulgation of documents such as the social security standards in the form of international treaties appear to stem from a very broad base indeed, afforded by the language found in the preamble to the constitution of I. L. O. and the declaration annexed to the constitution. This preamble and declaration are to the effect that "social justice," including, among other things, all-out social security, is essential to the establishment of lasting peace, and that failure of any nation to act is an obstacle hindering other nations. This lays a broad predicate for international treaties governing domestic programs of the ratifying countries.

Chapter I, article I, of the constitution states, "A permanent organization is hereby established for the promotion of the objects set forth in the preamble . . . and in the declaration . . . which is annexed . . ." The declaration annexed states, "The Conference recognizes the solemn obligation of the I. L. O. to further among the nations of the world programs which will achieve: . . . (f) the extension of social security measures to provide a basic income to all in need of such protection and comprehensive medical care." The United States Government, incident to its I. L. O. membership, has ratified this constitution and the annexed declaration.

It is difficult to demonstrate, therefore, that the social security minimum standards treaty is outside the scope of the I. L. O. constitution or that in reaffirming its I. L. O. membership after adoption of the preamble and declaration the United States was not put on notice that a treaty of this type might be promulgated. A point that might have been raised when the amended charter was up for consideration is whether the United States did in fact subscribe to the statements and assumptions of the preamble and the declaration. Certainly this was a pertinent question, particularly with respect to medical care.

I. L. O. Emphasis on Treaties.—While I. L. O. has a much wider field of action that has great potential value, the thrust and drive of some governments and the solid labor block has been in the direction of treaties in achieving I. L. O. purposes. If one subscribes to the philosophy that the broad I. L. O. social security and other objectives can be achieved by national legislation, and only by that process, and that these national programs are essential to preserve peace, in other words, if one subscribes to the interpretation of I. L. O. made by labor and many governments, it must be admitted that there is some logic back of the demands for international treaties dealing with the international standards for state medical care and other social security programs.

Our Philosophy vs. Welfare State Philosophy

There is no question as to the desirability of social justice and of the economic security of the individual. But neither, at least from our viewpoint, is there any question of the paramount desirability of individual freedom—including freedom from dependence of an all-powerful state. Nor do we question the fact that such freedom can be achieved only under what is termed free enterprise. Thus, unless we are in time to surrender to the philosophy back of the new social security treaty, it would seem essential that we spell out and actively press for the philosophy of free enterprise.

That this task is not easy, in view of much current international thinking, is manifest when a sample or so taken from the speeches made in Geneva respecting the new social security standards is considered. Perhaps the statement made at the International Labor Conference by Mr. Leao de Vasconcelos of Brazil might be taken as typifying a good bit of thinking regarding social security by some governments. He stated:

"One of the most important characteristics of the modern world is the constant interest shown in social problems and in social security. The introduction of effective measures of social security and public assistance is considered one of the best means of alleviating misery and of avoiding the emergence of seditious forces.

"We therefore feel it is essential to achieve international understanding in the social field and to establish international standards . . . Now that we are conscious that labour legislation, including that relating to social security and social welfare, will have a very great influence on economic life, sacrifices must be made to enable the workers to enjoy a decent standard of living within the economic framework."

The general attitude of workers in demanding treaties is probably that indicated by Mr. Texier of France:

"To meet human needs is one of the essential purposes of social security . . . It was to give concrete expression to this indispensable concept of social security that . . . the I. L. O. adopted recommendations . . . concerning income security and medical care . . . Either by means of valid legal documents we sanction the principles . . . or we take on ourselves the heavy responsibility of bringing to a halt our progress towards the achievement of social security and greater social justice."

Notwithstanding past experience as to ratifications and the arguments advanced against adoption of the social security standards in the form of a convention (treaty), the workers' monolithic position on social security standards ran true to pattern. A principal answer to all arguments against the form of the instrument was to impugn the motives of those advancing the arguments. This answer was summed up by an American worker representative in the following statement: ". . . employers generally are against conventions because conventions carry with them the obligation to act, to do something, to carry out objectives in support of the workers throughout the world."

United States Government Votes for the Treaty

Under the I. L. O. constitution, for an instrument to be adopted either as a convention (international treaty) or a recommendation, a two-thirds record vote is required. If it fails to secure the necessary vote as a convention, on motion of any member it may be adopted as a recommendation and revised accordingly. Thus, even if a member believes that the instrument sets fair standards, he still has the choice of voting whether the instrument should be advisory to members or mandatory on all ratifying members.

In the Social Security Committee two votes indicated that less than two-thirds wanted a convention. One vote was taken at the beginning of its sessions to determine the basis on which the text would be drafted, the other at the end of its sessions. In both instances the vote in favor of a convention fell substantially short of two-thirds, although the worker representatives voted solidly for a convention. Thus there is every reason to believe that in plenary session the instrument would not have been adopted as a convention except for a shift in position by government delegates who had previously voted against it as a convention.

In the light of these facts it is difficult to evaluate a United States government representative's statement in plenary session, before the final vote: "The United States Government delegation is going to vote for the convention . . . The United States Government supported the amendment to change its form to a recommendation. Nevertheless, while believing that a recommendation would be preferable, we will not withhold our support for a convention since the Conference prefers that form." It would appear obvious that an announced intention of this nature influenced the final adoption of the minimum social security standards in the form of an international treaty.

Whatever may be an administration's social security policies or our nation's current social security programs, there is an important question as to whether instruments like the new social security convention should be ratified. The solemn nature of treaties is recognized in our Constitution. Article VI, Clause 2, provides as follows: ". . . all treaties made, or which shall be made, under authority of the United States, shall be the supreme law of the land; and the judges in every State shall be bound thereby, anything in the constitution or laws of any State to the contrary notwithstanding."

Regardless of legal refinements as to the exact legal situation on ratifying a treaty by virtue of the foregoing provision, it seems obvious that voting in favor of provisions and their adoption as treaties should be matters of great thought and caution by representatives of the United States. In our current social security laws Congress has specifically reserved the right to amend or repeal them. This is obviously inconsistent with a permanent obligation to maintain social security programs meeting international specifications. Being a "good fellow" or "going along with the majority" affords no basis for voting for a convention containing provisions inconsistent with national policy, evidenced by congressional action, or of placing the government in the awkward position of refusing ratification of an instrument that its representatives have helped formulate and voted for as a treaty.

Of course, the fact that our government representatives voted for this treaty does not mean that our social security is automatically changed to include socialized medical care and other benefits in conformity with the provisions of any of its nine programs. Nor does it mean that the Senate will ratify it, or that Congress will pass implementing social security legislation establishing medical care, compensation for sickness, invalidity, maternity, family allowance, or other programs within its purview. Even ratification does not require adherence to more than three programs.

But beyond question, from an international viewpoint, the United States government is in the position of having voted in favor of the adoption of these social security minimum standards and of having voted that these standards shall be in the form of an international treaty. Under our international obligation assumed under the I. L. O. constitution as an I. L. O. member, this document must be considered for ratification and enactment of implementing legislation contemplated. The same situation may again arise in connection with the presently postponed advanced social security standards.

With these considerations in mind, let us examine further some of the major medical care aspects and the basic principles involved and the political maneuvers employed in getting the instrument adopted as a convention.

Ratification on the Basis of Token Programs.—In the case of the United States and other "developed" countries, under the treaty the medical benefits scheme must, at a minimum, cover (*a*) at least half of all employees in the country plus their wives and children; or (*b*) "economically active" persons constituting at least one-fifth of the total population, plus their wives and children; or (*c*) half of the population. Other programs require correspondingly broad coverage.

Obviously a very large percentage of the 60-odd nations belonging to I. L. O. could not possibly provide medical programs of this extent, and would not vote for the instrument as a convention unless they could ratify it. The tentative text had escape clauses, permitting ratification by these nations with little more than token coverage. Medical care, in their cases, need cover only half the "employees in industrial work places employing 20 persons or more, and also their wives and children." In many undeveloped countries such persons would constitute only a negligible proportion of the population.

This "practical" provision has very obvious political advantages. The token program that was permitted for countries like India secures their vote for a convention. They can have the prestige of ratifying an international treaty on social security standards, even though millions of their people may be starving. Without this concession, adoption of the convention desired by labor and some of the governments of "advanced countries" would not have been politically possible.

How Long Is Temporary?—Article 3, the "escape clause" above referred to, applies to any "member whose economy and medical facilities are insufficiently developed"—a substantial majority of I. L. O. voting strength. The theory in the text was that this was a mere temporary provision. Obviously the "escape clause" did not get labor in those countries very far. Amusingly enough, when "temporary" was defined by a solid labor vote and many government votes as not more than 10 years a great deal of furor arose among the governments concerned.

At the plenary session, Mr. Stark, Workers' Advisor of Austria and leader of the worker block, recognized that, if "temporary" remained defined as 10 years, these countries' votes would be lost and there would probably be no convention. Hence he stated, respecting a floor amendment deleting the 10 years limit, "In the interest of making ratification possible, and in the interest of frankness, as far as ratification is concerned, the workers' group has decided, however, to vote in favor of this amendment."

Thus a purely political maneuver, indefensible under I. L. O. principles, insured that I. L. O. minimum social security standards could be adopted as an international convention.

World Medical Association Recommendations

In the May 31 issue of THE JOURNAL, page 488, it was stated with respect to W. M. A. recommendations as follows:

"The only section in the document that may puzzle American physicians is that pertaining to Principle XI, which reads: 'Compulsory health insurance plans should cover only those persons who are unable to make their own arrangements for medical care.' In the United States physicians do not believe that compulsory plans are necessary for anyone. It must be remembered, however, that many countries do have compulsory plans, but the doctors in all countries that are members of the WMA are in agreement that if such plans do exist they should be limited as expressed in the quoted statement."

Section XI might be described as an attempt to limit social security medical care to what is sometimes described as the "medically needy." It is very understandably the position of physicians who are faced by the grim reality of a compulsory medical care program. To the extent that patients are left free to make private arrangements, physicians are free to enter into such arrangements with them.

Great practical and political difficulties attend any effort to have a nation "half slave and half free" medically. The whole thrust and drive of the philosophy of compulsory social security is in opposition to making the source of medical care dependent on the means of the patient. This is regarded as "degrading." Financing is very frequently by special taxes called "contributions" and these "contributions" of higher paid persons may be in part used for the benefit of lower paid persons, just as in our old-age and survivors insurance system. Obviously the cost distribution machinery of such a system is crippled if the higher paid are exempt, and there are political difficulties in connecting "compulsory contributions" for medical care denied some contributors. Obviously section XI would raise a host of side issues to the main issue of "medical freedom."

The "Medical Freedom" Amendment.—Without consultation with the W. M. A. I decided to introduce a specific amendment embodying broad principles that it had advanced. Rightly or wrongly, to avoid issues such as those indicated above, I decided to omit the "medically needy" coverage provision and certain others of its 12 points.

The rejected amendment was as follows:

"Add a new Article 64 bis, as follows:

In the provision of medical care referred to in Parts II and VIII the following general principles shall be observed:

1. There shall be freedom of choice of physician and hospital by the patient except in cases of emergency.
2. There shall be no intervention of a third party between the physician and the patient.
3. There shall be no restriction as to medication or mode of treatment prescribed by the physician except in case of abuse.
4. Physicians shall have the right to participate or refuse to participate in the medical care system and their participation shall not be conditioned on their moving to or from any area or becoming employees of the government or engaging in any special kind of practice.
5. Physicians shall be appropriately represented on administrative and advisory bodies concerned with the provision of the medical care.
6. There shall be no exploitation of the physician or the physician's services."

Action on the Amendment.—This amendment was introduced with the statement that any clarifying amendments were welcome, and it was an amendment designed to secure the adoption or rejection of broad principles and to be discussed on that basis, and not on the basis of minor matters that could be corrected by subamendments. A brief description of some of the discussion is contained in the committee report on the new social security instrument. This statement is as follows:

"An Employers' member submitted a proposal to add a new Article establishing general principles to be observed in the provision of medical care. These included notably free choice of doctor and hospital by the patient; non-intervention of a third party between physician and the patient; freedom of medication and mode of treatment by the physician except in case of abuse; right of the doctor to participate in the service; appropriate representation of physicians in administrative and advisory bodies, and a guarantee that there should be no exploitation of the physician or the physician's services.

"The author explained that this amendment was based on the statement made by the World Medical Association, which represented medical associations in 43 countries and some 700,000 doctors. It seemed reasonable that the proposed instrument should contain not only quantitative but also qualitative provisions; as this had been done in the Medical Care Recommendation, No. 69, adopted in 1944, there seemed to be no reason for not including such an important matter in the instrument under consideration.

"The Committee, however, by a large majority comprising members of all three groups, did not deem it advisable to do so, more particularly in view of the fact that, to many members, the principles formulated in the amendment would not be acceptable without modification. Moreover, this subject would require careful examination and was, in any case, not within the competence of this Committee."

The assigned "reasons" for rejecting the amendment were mere smoke screens. No modifications of the amendment were suggested by anyone opposing it. The matter was fully in the competence of the committee, as evidenced by the fact that a "freedom of doctor" provision was adopted in the revised maternity convention also voted out by the conference this year. The English government representative stated that England could not ratify a convention containing the amendment.

Fundamental Conflicts Between "Social Security" Medicine and Private Medicine

Adoption of the amendment would probably have meant little more than an empty gesture. There are fundamental and probably irreconcilable conflicts between the underlying philosophy and practices of "compulsory health insurance" and the individual freedoms called for in the amendment.

The philosophy of "health insurance" calls for universal coverage of people and medical services. The practical situation is that freedom of a medical practitioner may be akin to the freedom to stop practicing; his position in not participating is that of dependence on persons who have already paid for their medical care who may exercise their "freedom" by coming to him and paying again. Under the philosophy of a compulsory system, the state assumes responsibility to have practitioners and hospitals available for everyone covered. Such a responsibility is inconsistent with any prohibition against coercing physicians to move to places where the state must have them in meeting its obligations.

The medical care is a "benefit," conceptually a matter of right, and its denial, whether because of medical judgment or other cause, is, under specific provisions of the treaty, a matter of administrative or court action and decision. This concept is hardly compatible with the principle of nonintervention of third parties. Being operated by government, the system is by nature subject to politics. What practical meaning would be given the prohibition against exploitation of physicians or physicians' services? If physicians are underpaid—whether on a fee, salary, or per capita basis—and equitable adjustment means raising "contributions" on a hundred times the number of underpaid physicians, the obvious political answer is to "protect" the "contributors."

The foregoing is merely illustrative of a host of fundamental considerations surrounding "social security medicine," however designated and however described. Fundamental considerations of this kind must be used in evaluating such statements respecting medical freedom as that made on the floor of the I. L. O. Conference by an American labor representative:

"Now, speaking solely as an American, I would like to make a few brief remarks on the problem that was raised by the Employers' representative of the United States when he spoke about 'socialised medicine.' He said that as an employer he could not support this social security Convention because one of its branches, medical aid, was socialised medicine. I just want to say that the implication of that remark is that those of us in the United States who are supporting what we call medical assistance, or a national health insurance programme, do not call that socialised medicine—nor is it. The programme advocated in the United States by the trade union movement is one which ensures freedom of choice of doctor, freedom of choice of hospitals, and freedom of choice of medical assistance in any form, and does not in any way interfere with the normal patient-doctor relationship which Mr. Calhoun, the Employers' representative of the United States, has talked about.

"I would like this Conference to know that, as far as the United States is concerned, we do believe in health insurance and we hope that before long, through the strength of our trade union movement and the representatives of our Federal Government who support the national health insurance programme, we will be able to have such a programme."

American medicine must stand ready to answer whether or not we can have the medical utopia indicated by this statement, and its answer must deal clearly and understandably with fundamental facts and principles.

THE INTERNATIONAL LABOR ORGANIZATION—ITS HISTORY, PURPOSE, AND PLANS

This article has been prepared in the office of the Committee on Legislation of the American Medical Association because of the increasing interest of the medical profession in the International Labor Organization.—Ed.

The International Labor Organization, meeting in Geneva in June, 1952, approved a Convention on Minimum Standards of Social Security in nine fields: medical care, sickness benefits, unemployment benefits, old age pensions, employment injury benefits, family allowances, maternity benefits, invalidity benefits, and survivor benefits.

While the medical benefits in the convention are carefully distributed throughout the document, considered together they constitute what is commonly referred to as "socialized medicine." The approval of this convention, with an affirmative vote by the majority of the United States delegates, in the face of a clear-cut repudiation of compulsory health insurance by the American people and their representatives in Congress, is tangible evidence of a determination to provide socialized medicine even in the face of objections by citizen voters.

At the meeting of the House of Delegates of the American Medical Association in June of this year a resolution was adopted that, in part, advocated "an amendment to the Constitution of the United States which will provide that no Treaty or Executive Agreement shall be made which conflicts with any provision of the Constitution or which operates, or may operate, to regulate any of the purely domestic affairs of the United States." Every physician should acquaint himself with the details of the organization, the history, and the program of the United Nations and the I. L. O. The purpose of this article is to present, in detail, pertinent facts concerning the I. L. O. and to discuss the treaty as an instrument of legislation.

History of the I. L. O.

The charter that brought the United Nations into being on Oct. 24, 1945, provided for a General Assembly, a Security Council, an Economic and Social Council, an International Court of Justice, a Trusteeship Council, and a Secretariat. In addition, more than a score of commissions and specialized agencies were created. The I. L. O. was the first such agency with which the United Nations established such a relationship. This relationship was agreed on by the I. L. O.'s general conference and by the U. N.'s General Assembly in 1946.

Simply stated, the I. L. O. is an association of nations. It was established on April 11, 1919, by the adoption of Part XIII of the Treaty of Versailles, and during the period between World War I and World War II was affiliated with the League of Nations. The charter members were the original members of the League of Nations. Countries joining the League later automatically became members of the I. L. O., whereas nonmembers of the League could be admitted by order of the International

Labor Conference. The United States, although a signatory of the League Covenant, was not a charter member and did not join until 1934.

The I. L. O.'s present constitution recognizes as members those governments that were members on Nov. 1, 1945. It also provides that any member of the United Nations can become a member of the I. L. O. simply by accepting the obligations of membership. Countries that are not U. N. members must, on the other hand, file applications, which have to be approved by the general conference. Today the membership numbers 65 countries. Twenty-two are European, 20 are American, 19 are in the Asian and Far Eastern region, and 4 are African.

Purposes

The Commission on Labor Legislation, which drafted the original constitution of the I. L. O., was composed of representatives of the United States, Great Britain, France, Italy, Japan, Belgium, Czechoslovakia, Cuba, and Poland. Samuel Gompers, President of the American Federation of Labor, was chairman of that commission, and Arthur Fontaine of France, who later was to preside for 10 years over the I. L. O.'s governing body, was its general secretary. Its assistant general secretary was H. B. Butler, who subsequently became director of the International Labor Office.

The commission was assigned the task of inquiring "into conditions of employment from the international aspect, and to consider the international means necessary to secure common action on matters affecting conditions of employment, and to recommend the form of a permanent agency to continue such enquiry and consideration. . . ."

One of the first basic decisions to be made concerned the powers that the new organization should have. It is indeed interesting to note that one suggestion was that the new body be given the right to adopt legislation that would automatically become binding on all the member countries. This suggestion, which would have given to an international agency the power to legislate for member governments in matters vitally affecting their domestic economy, was rejected. It was agreed then, and apparently needs to be reemphasized now, that such a plan would result in an invasion of national sovereignty.

It was finally agreed that the organization would draft conventions similar to treaties. These would not automatically become binding on the member countries, but the members would be required to consider accepting or ratifying them. It was agreed that once a country ratified a convention it must give effect to it and submit reports on the way in which it was doing so.

When the general conference of the I. L. O. met at Philadelphia in 1944, a redefinition of the organization's aims and purposes was agreed on. In addition to spelling out the responsibility of the I. L. O. to examine and consider international economic policies and measures, the statement set forth a number of specific objectives. It declared that it shall be a permanent obligation of the I. L. O. to further among the member nations the achievement of the following 10 propositions.

(1) full employment and raising of standards of living; (2) the employment of workers in the occupations in which they can give the fullest measure of their skill and attainments and make their greatest contribution to the common well-being; (3) the provision of facilities for training and the transfer of labor, including migration for employment and settlement; (4) policies in regard to wages and earnings, hours and other conditions of work, and a minimum living wage to all employed and in need of such protection; (5) the effective recognition of the right of collective bargaining, the cooperation of management and labor in the continuous improvement of productive efficiency, and the collaboration of workers and employers in social and economic measures; (6) the extension of social security measures to provide a basic income to all in need of such protection and *comprehensive medical care*; (7) adequate *protection for the life and health of workers* in all occupations; (8) provision for *child welfare and maternity protection;* (9) the provision of adequate nutrition, housing, and facilities for recreation and culture; and (10) the assurance of equality of educational and vocational opportunity. (Emphasis added in 6, 7, and 8 above.)

Organization

The I. L. O. has three basic parts—the general conference, which is also called the International Labor Conference; the governing body, and the International Labor Office. The general conference, which is called together once a year by the governing body, has the task of drafting the International Minimum Social and Labor Standards that later must be considered for ratification by the member countries. Every three years the conference is the scene of the election of members of the governing body. Each year it adopts a budget to finance the organization's work in the succeeding year. It also considers major questions relating to the organization's policy and program. In line with the "tripartite" principle, each of the member countries is represented by four delegates. Two of these represent the government, one represents the workers, and one the employers.

The governing body, or executive council, has authority to act on its own responsibility over a wide range of subjects. Of its 32 members, 16 are government representatives, 8 are workers, and 8 are employers. Eight of the 16 government seats are occupied by representatives of the 8 member countries that are agreed to be of chief industrial importance; the other 8 are elected every three years by the government delegates to the conference.

The governing body elects a chairman from among the government representatives, and two vice-chairmen, one from the workers' group and one from the employers'. It holds meetings three or four times a year. In addition to its responsibilities for the formulation of policies and programs, the governing body controls the work of the International Labor Office, and supervises the activities of a large number of advisory committees and commissions. Among its other responsibilities is the election of the director-general of the office. It also has the job of preparing the agenda of the general conference and making proposals to the conference for the annual budget.

The International Labor Office is composed of the permanent staff, consisting of approximately 600 officials recruited from 50 different nationalities. It is this office that prepares the reports and documents and working papers on which the various organs of the organization base their decisions.

There is also a whole series of advisory committees to assist the organization in its work in particular fields. Finally, there is a committee of experts on the application of conventions and recommendations. It is the job of this body to examine the reports of governments on the way in which they are giving effect to I. L. O. standards.

Procedures

The machinery used to produce an approved I. L. O. convention or recommendation is placed in motion by the submission of a labor "problem" by a member government to the governing body, with the suggestion that it be placed on the next agenda. Following a series of reports by the International Labor Office and technical committees, the text of the recommended convention is submitted to the general conference for a final record vote. In this final vote, if two-thirds of the delegates answer the roll call "yes," the text is approved and another International Labor convention or recommendation is adopted.

Despite the fact that member governments are not required to ratify a convention, the I. L. O. constitution requires the submission of all conventions adopted by such members to their national legislatures within 18 months to be considered for ratification. This policy assures that the convention comes to the attention of the public.

Governments are also required to report to the I. L. O. whether or not a convention has been ratified, what steps they have taken to bring the convention before their legislatures, and "the difficulties which prevent or delay the ratification of such Convention." The committee on the application of conventions and recommendations then prepares a combined report, including a summary of the information that governments have sent in, for submission to the governing body and to the general conference. These documents are referred to a special conference committee composed of government, worker, and employer delegates, which goes over the information that governments have supplied and examines the comments of the committee.

The proceedings of this conference committee are held in public, which tends to insure that the force of public opinion is used to pressure governments into ratifying conventions or implementing those already ratified.

The director-general's report of 1949 points out that: "There is no quick or easy solution to the problem of ratifications, but the exploration of its multiple aspects would be much advanced if the delegates would contribute freely of their views and experience on how implementation of international labor legislation can be furthered, particularly in their own countries."

This statement indicates a failure to recognize and honor the basic purpose and function of the I. L. O. in adopting conventions. While the affirmative vote of the I. L. O. is a prerequisite to submission of a convention to the various countries, this action does not result in international legislation. Under the I. L. O. constitution, it is improper to question the right of any country to refrain from ratification of a convention. Neither the I. L. O. constitution nor any other international agreement establishes such an obligation.

The conventions of the I. L. O. are referred by government delegates of the U. S. A. to the Department of State and the Department of Labor for further study and for a determination as to whether Senatorial consideration is desirable. Only 6 of the 103 conventions framed and adopted by the I. L. O. have been ratified by the United States.

Treaty-Making Powers

While conventions were formerly regarded as international agreements dealing with the less important subject matters of international negotiation, the conventions offered by the I. L. O. have the scope of treaties and are of a distinctively legislative character. It is of extreme importance, therefore, that everyone understand the fact that a treaty, once ratified and in force in the United States, automatically becomes a part of municipal law, a fact that is not generally true in other countries. The country as a whole must realize more than it has in the past that the treaty method of making law lacks the safeguards that exist under its constitutional method of legislating.

Article II, Section 2, of the Constitution of the United States provides: "The President . . . shall have Power, by and with the Advice and Consent of the Senate, to make Treaties, provided two-thirds of the Senators present concur; . . ."

This provision was written at a time when the usual treaty was a contract between sovereignties, a contract that dealt with international affairs—treaties of peace, settlements of war problems, transportation and communication between nations, and similar problems.

The framers of the Constitution were determined that no state should make treaties with foreign powers. It was realized by the founding fathers that in order for stable commercial relations to be established with other countries the Constitution must insure that the federal government could and would carry out its treaties, and thus Article II, Section 2, was enacted in its present form.

James Wilson, one of the members of the Constitutional Convention urged that, if treaties were to have the operation of laws, they should have the sanction of laws. He therefore moved to add the words "and the House of Representatives" after the word Senate. But the view that treaties must be negotiated in secret prevailed, and the supremacy clause was enacted in its present form. The result is that the framing of treaties is a wholly executive function.

While it is true that the two-thirds vote required for ratification by the Senate is sometimes difficult to secure, nevertheless the steps followed in the framing of a treaty neither are within the control of the Senate nor are they publicly recorded in many instances. The fact that a treaty may be submitted either by the United Nations or by a specialized agency presents a procedure essentially different from the negotiation of a treaty between the United States and another country. In this latter instance, the treaty is normally drafted by our own Department of State and presented to another country for consideration.

Of far greater significance than the unorthodox manner in which I. L. O. conventions are prepared is the fact that legislation, the usual mechanism used in deciding domestic issues, is shaped by our own elected officials, and is subject to scrutiny from start to finish. The original bill, the amendments the committee hearings, and the subsequent actions by the House and Senate are all part of the legislative process. Obviously, this is still the proper method to use in considering changes in our domestic law.

It is also worthy of note that in all but a few other countries treaties take effect as municipal or domestic law only when implemented by legislation. Since the United States Constitution makes a treaty the supreme law of the land without regard to implementation, this safeguard does not exist for us. Although states signatory to previous I. L. O. conventions have usually taken the steps necessary to put them in force, the increased use of the treaty for enacting domestic legislation may increase their reluctance to do so and render their advantage in this regard more important.

Summary and Conclusions

It is apparent from the foregoing that the distortion of the treaty power by the I. L. O. could place not only American medicine but national freedom and the Constitution in jeopardy.

Perhaps the greatest danger is that the drafting of treaties by remote commissions not answerable to the American public, the pressure to secure ratification, and the creation of quasi-judicial bodies to hear charges of violations place the administration of important domestic problems in a government of men not of laws. The bureaucratic official is the vital factor in this attempt to secure universality of law throughout countries in which the entire political, religious, and social background is without similarity.

As indicated earlier, representatives from each country to the conferences of the I. L. O. are theoretically chosen as representatives of the government, labor, and management. These conferees meet only for a fraction of each year and have neither the permanent contact nor the experience with general world problems necessary to frame sweeping legislation of the type contained in their conventions. In addition, it can hardly be contended that the delegates for the United States government are true spokesmen for the general public. At the most recent meeting of the I. L. O. in Geneva, and in fact for the past several years, the government delegates appointed by the administration through the Department of Labor have been Sen. James E. Murray of Montana and Assistant Secretary of Labor Philip Kaiser.

The tripartite delegation from the United States, of which these government delegates are a part, operates like a stacked deck. The record of divided votes in the last three international conferences tells this story: On 27 major votes in three years the United States government delegates teamed with the labor delegates 24 times to outvote the delegate representing management.

Although the United States has ratified only six conventions, some of the safeguards that have been suggested to prevent abuse are as follows:

1. The United States Constitution could be amended to provide in substance that:
    (*a*) treaties that conflict with the United States Constitution are invalid.
    (*b*) any treaty that directly and substantially interferes with the domestic legislative jurisdiction is invalid, except when the subject matter presents a truly international problem that requires international action to handle it.
2. The United States could demand that specialized agencies affiliated with the United Nations be prohibited from intervening in the domestic affairs of member governments.
3. The representatives of the United States at I. L. O. meetings could refuse to vote to submit for ratification proposed treaties or conventions that directly and substantially encroach on domestic legislation.

A Senate Joint Resolution was introduced in the Senate on Feb. 7, 1952, by Sen. John W. Bricker of Ohio that would accomplish the first item listed above. This resolution, which was co-sponsored by 58 other Senators, would prevent treaties or executive agreements from nullifying the laws of the United States or the constitutions and laws of the several states and would prevent executive agreements from being used as a substitute for treaties. Although this resolution died with the adjournment of the 82d Congress, it is likely that the same or a similar resolution will be introduced early in the 83d Congress.

MEDICAL NEWS

CALIFORNIA

Radio and Television Speakers' Panel.—The Radio and Television Committee of the San Francisco Medical Society is organizing a panel of physician-members who will be available for radio and television appearances as well as personal addresses before lay audiences. Any member desiring to interpret scientific data for the lay public may join the panel by writing to the society's Radio and Television Committee, consisting of Drs. Anthony B. Diepenbrock, chairman; John J. Miller; and Felix L. Pearl.

COLORADO

Narcotic Violation.—Dr. Leslie Harrison Wade, Denver, pleaded guilty in the United States District Court at Denver to a charge of violation of the federal narcotic law. On May 14 his sentence of one year and one day was suspended, and he was placed on probation for one year.

State Medical Meeting in Estes Park.—The Colorado State Medical Society will hold its annual session Sept. 9-12 at the Stanley Hotel in Estes Park under the presidency of Dr. Harry C. Bryan of Colorado Springs.

On Thursday at 2 p. m. Dr. Louis H. Bauer of Hempstead, N. Y., President of the American Medical Association, will discuss "Recent Developments in A. M. A. Activities." Other guest speakers and their first presentations will include:

Vincent J. O'Conor, Chicago, Neck Obstruction in the Male.
Alexis F. Hartmann, St. Louis, Management of Pediatric Metabolic Emergencies.
Richard B. Cattell, Boston, Surgery of the Gallbladder and Bile Ducts.
Herbert W. Schmidt, Rochester, Minn., Diagnostic Problems Encountered in Chest Diseases.
O. Spurgeon English, Philadelphia, Personality Growth and Development.
Robert M. Zollinger, Columbus, Ohio, Indications for Surgery in Jaundiced Patients.

On Tuesday at 6 p. m. there will be a dinner at which sports trophies won during the afternoon tournaments will be awarded. The dinner is not limited to contestants. The annual banquet, to be held at 7 p. m. Friday, will be followed by a dance at 9:30.

DELAWARE

Dr. Springer Honored.—Dr. Harold L. Springer of Wilmington was honored for his 50th year in medical practice at a testimonial dinner at the Du Pont Country Club. A gold desk clock was presented, and talks in tribute to him were given by Drs. George Wilson and Thomas Klein of Philadelphia. Dr. Springer is a consultant in the surgery division and a former chief surgeon at the Delaware Hospital in Wilmington.

State Medical Meeting.—The Medical Society of Delaware will hold its annual meeting at the Rehoboth Beach Country Club Sept. 8-10 under the presidency of Dr. Ervin L. Stambaugh, Lewes. Guest speakers will include:

Harrison F. Flippin, Philadelphia, The A—B—C of Antimicrobial Therapy.
Paul C. Colonna, Philadelphia, Malunited and Nonunited Femoral Shaft Fractures.
John H. Gibbon Jr., Philadelphia, Surgical Treatment of Carcinoma of the Esophagus.
Theodore R. Fetter, Philadelphia, Management of Urinary Tract Infections.
Reuben Friedman Jr., Philadelphia, Dermatology in Relation to Chiropody.
Anthony Sindoni Jr., Philadelphia, Metabolic Medicine in Relation to Chiropody.
John Lansbury, Philadelphia, The Place of Newer Drugs in Treatment of Arthritis.
Richard W. TeLinde, Baltimore, Endometriosis.
George A. Hahn, Philadelphia, Diagnosis and Management of Uterine Malignancy—A 25 Year Experience.

FLORIDA

Society News.—The newly organized Greater Miami Society of Psychiatry and Neurology has named Dr. James L. Anderson, Miami, temporary chairman and Dr. Paul S. Jarrett, Miami, secretary. Drs. Herman Selinsky and Edward H. Williams, both of Miami, have been appointed as a committee to draw up the constitution and by-laws for the society.

A. M. A. President to Speak in Florida.—The Florida Medical Committee for Better Government will hold its third annual meeting at the George Washington Hotel, Jacksonville, Sept. 6-7. Dr. Louis H. Bauer of Hempstead, N. Y., President of the American Medical Association, will address physicians and their wives at a luncheon meeting at 12:30 p. m. on Sunday. Early reservations are advised.

ILLINOIS

Louis Bothman Fellowship.—The University of Illinois College of Medicine, Chicago, announces the establishment of the Louis Bothman Fellowship in ocular pathology in memory of the late Dr. Bothman, clinical professor of ophthalmology, who was instrumental in developing a laboratory in ocular pathology at the university's Research and Educational Hospitals. The university has received a grant of $5,000 from the Louis Bothman Memorial Fund, Inc., of Phi Delta Epsilon fraternity in support of the fellowship.

Tuberculosis X-Ray Program.—The executive secretary of the Tuberculosis Institute of Chicago and Cook County announces that signs suggestive of tuberculosis were found in about 3 of every 200 persons undergoing the 1952 free chest x-ray examinations in the Chicago area. "The cases have been found," he said, "in time to offer the victim a good chance of recovery and to stop the spread of the infection to others."

A recent study by the institute indicates that the cost of tuberculosis to the community averages more than $15,000 for each case not found until the disease is advanced. The cost includes treatment and aid to the patient's family, if necessary. The x-ray program is being carried out by the institute, the Municipal Tuberculosis Sanitarium, and the Suburban Cook County Tuberculosis Sanitarium District.

Chicago

Pediatric Society Golf Tournament.—The first annual Chicago Pediatric Society golf tournament will be held Sept. 10 at the Northwestern University Course, Lake Street, west of Eden Highway, Wilmette. The fee for golf and dinner is $8, for dinner only, $5.

Annual Conference at Maternity Hospital.—The annual Televised Postgraduate Conference in Obstetrics and Gynecology will be held at Lewis Memorial Maternity Hospital, Sept. 15-19, under the auspices of the department of obstetrics and gynecology, Stritch School of Medicine of Loyola University. Television screens will be placed throughout the hospital to assure clear viewing and listening in connection with the obstetrical procedures, demonstrations, and gynecologic surgery that form the program. There will be no registration fee.

MINNESOTA

Northern Minnesota Meeting.—The annual meeting of the Northern Minnesota Medical Association is being held at Crookston Aug. 22-23. The Friday session features a talk by Dr. Harry E. Ungerleider, director of medical research, Equitable Life Assurance Society, New York, on "The Insurance Aspects of Heart Disease." On Saturday a clinico-roentgenologic-pathological conference will be conducted by Dr. Henry G. Moehring of Duluth and his associates.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

Southern Minnesota Medical Association.—The annual meeting of the Southern Minnesota Medical Association will be held at Austin Sept. 8. Among the topics to be discussed are migraine, use of compound F (17-hydroxycorticosterone) in treatment of post-traumatic bursitis of the knee and ankle, cutaneous malignancies, urgent surgical conditions in the newborn period, diseases of the thyroid, Friedländer's pneumonia, danger of use of cortisone in acute rheumatic fever and concurrent renal damage, coronary insufficiency, physicians' disagreements in court, and neurosurgical treatment of intractable pain owing to metastatic carcinoma.

NEW YORK

Degree in Physical Therapy Offered.—The University of Buffalo announces the establishment of a program in physical therapy, offering a degree. The program, which will begin with the fall semester in September, will be given under the aegis of the School of Medicine and the Chronic Disease Research Institute. A four year program will lead to a bachelor of science degree with a major in physical therapy. A 12 month course leading to a certificate of proficiency in physical therapy is offered only to students of advanced standing. The program will meet standards established by the Council on Medical Education and Hospitals of the American Medical Association, the American Physical Therapy Association, the American Registry of Physical Therapists, and the New York State Department of Education.

The following persons, who are members of the faculties of other divisions of the university, are among those appointed to the staff of the new program: Dr. Peter A. Casagrande, assistant in orthopedic surgery; Eulalia W. Fabian, chief, speech and hearing therapy; Dr. Robert A. Kaiser, instructor in anatomy; Dr. Heinz Lichtenstein, assistant in psychiatry; Dr. L. Maxwell Lockie, head, department of therapeutics; Julia E. Lockwood, Ph.D., associate professor of physiology; Dr. Henry V. Morelewicz, medical director, program in physical therapy; Lyle W. Phillips, Ph.D., professor of physics; Clarice Schwartz, chief, occupational therapy; John H. Warfel, Ph.D., instructor in anatomy; and Dr. Walter T. Zimdahl, director, Chronic Disease Research Institute.

New York City

Heat Prostrations.—According to the department of health, 83 deaths from heat prostration were reported during June and July. No heat prostration deaths were reported to the department in 1951, and in 1950 five such deaths were reported. Of the 83 persons listed as killed by heat, 43 were male; 72 were aged 50 and over; 3 were under 1 year of age; 1 was 1 year of age; and 7 were in their 40's.

The record heat wave of July raised the general death rate from the expected 8.7 to 10.1 per 1,000 of population. Increase of deaths from cardiovascular-renal diseases and the slight increase in infant mortality were also attributed to the extreme heat. The average cardiovascular-renal disease rate per 100,000 of population from July in the years 1949-1951 was 440; this year it was 538. The expected infant mortality rate was 22.6, but the actual infant mortality rate in July was 24.4.

Personals.—Dr. John J. Thorpe, who has been a member of the medical staff of Standard Oil Company (New Jersey) since 1949, has been named assistant medical director of Esso Standard Oil Company. Dr. Thorpe was formerly resident physician and research assistant in heart disease at New York Post Graduate Hospital, now University Hospital.——Dr. John Daley, formerly of the Esso medical staff, has been advanced to physician at large with Standard Oil Company (New Jersey).——Dr. James Allan Campbell, dean of Albany Medical College, has been appointed a member of the medical board of the New York State Employees' Retirement System, which acts on all accidental death and disability claims filed in behalf of retirement system members. Dr. Campbell formerly was assistant professor of internal medicine at the University of Illinois College of Medicine.——Dr. Max A. Schneider was recently honored by the Buffalo Fire Fighters, Local 282, AFL, who presented him with medical equipment in recognition of his volunteer services to injured persons. Dr. Schneider, who is assistant resident in medicine at Buffalo General Hospital, was recently awarded the Nelson G. Russell Fellowship in Medicine.

OHIO

Cerebral Palsy Center.—A cerebral palsy training center has been established by the Ohio B. P. O. E. at 325 Broad Ave., N. W., Canton. For the present, preschool children up to the age of 8 years are accepted. Dr. Jack W. Millis of Toledo has been appointed medical director.

Chief Surgeon Resigns.—Dr. B. Noland Carter, head of the University of Cincinnati College of Medicine department of surgery, has resigned after 30 years of service in the department. Dr. Carter, who was affiliated with Johns Hopkins University, Baltimore, before coming to the University of Cincinnati, was appointed to the Christian R. Holmes professorship of surgery at the latter in 1946. He is director of surgical service at Cincinnati General and Children's hospitals and consulting surgeon at Dunham Hospital. During World War II Dr. Carter served in the Surgeon General's department and was awarded the Legion of Merit medal. Dr. Carter was recently honored by a testimonial dinner at the Queen City Club and by publication of an editorial entitled "Well Done, Doctor!" in the *Cincinnati Enquirer*, which ended as follows: "Whatever the future holds for him, if it is in line with his past accomplishments we may be very sure that it will be for the greater good of humanity, because Noland Carter is that kind of a doctor and that kind of a man."

PENNSYLVANIA

Clinic Plan Revised.—The Pennsylvania Department of Health has announced the initiation of a new procedure in the administration of its syphilis control facilities. A number of syphilis clinics have been discontinued in conformity with the reduction of infectious syphilis cases during the past five years, attributed to treatment of the disease with penicillin. Dr. Edgar S. Everhart, chief, venereal disease division, announces that a system of appointment clinics, adopted "in order that sparsely inhabited communities may not entirely be deprived of free treatment facilities," has proved successful during a five month trial.

Telephone Talks to Eight Teaching Centers.—With the fall program of the Graduate Education Institute sponsored by the Medical Society of the State of Pennsylvania talks by medical authorities will be piped by telephone from Philadelphia to the eight teaching centers throughout the state. From 1 to 2 p. m. on Oct. 22, about 1,000 general practitioners in all parts of Pennsylvania will gather at the teaching centers to listen to the latest developments in diagnosis and treatment of heart disease through telephone connections and amplified at the various meeting places. The teaching centers for next season's graduate education courses include Erie, Pittsburgh, Johnstown, Harrisburg, Williamsport, Allentown, Wilkes-Barre, and Philadelphia. As in the past five years, the courses are conducted one day a week in each of the centers for five weeks in the fall and five weeks in the spring.

Philadelphia

Appoint Director of Philadelphia Hospital.—Dr. August H. Groeschel, assistant medical director of New York Hospital, has been named director of the Philadelphia General Hospital. Dr. Groeschel previously served as assistant medical director of the Health Insurance Plan of Greater New York. He will assume his duties in Philadelphia about Oct. 1.

TEXAS

New Unit for Polio Patients.—Since July 7 Hermann Hospital, Houston, has made 38 beds in its clinic building available for polio patients. This unit was opened because of the epidemic that has exhausted the regularly assigned polio beds in the city. Nurses especially trained in polio care were recruited for Hermann with the aid of the Red Cross and the National Foundation for Infantile Paralysis, and the ward was air-conditioned by the Harris County Polio Association. During its first two weeks in operation, the new unit at Hermann had received 23 polio patients.

WEST VIRGINIA

Gift to Medical Society.—Dr. Walter E. Vest, Huntington, presented to the West Virginia State Medical Association a bound volume of the transactions of the Medical Society of the State of West Virginia for the years 1867-1874, which he had found at a second-hand book shop in the South, and which probably is the only such volume in existence today.

Society News.—The West Virginia Society of Anesthesiologists has elected Dr. Eldon B. Tucker, Morgantown, president; Dr. Lester D. Norris, Fairmont, vice-president; and Dr. John F. Morris, Huntington, secretary-treasurer (reelected).——The West Virginia Obstetrical and Gynecological Society reelected Dr. Clarence H. Boso, Huntington, president; Dr. Gates J. Wayburn, Huntington, vice-president; and Dr. Angelo J. Villani, Welch, secretary-treasurer.——In the Association of Pathologists of West Virginia, Dr. Milford L. Hobbs, Morgantown, was named president, and Dr. Richard C. Neale, Bluefield, secretary.

WISCONSIN

University News.—Dr. Abraham B. Schwartz, associate clinical professor of pediatrics, has been named acting director of the pediatrics department at Marquette University School of Medicine, Milwaukee, to succeed the late Dr. Francis R. Janney.

Personals.—Dr. Thomas L. Tolan of Milwaukee was reelected president of the American Hearing Society at its annual meeting in Chicago.——Dr. James P. Conway was recently appointed by the Shorewood Board of Health as village health commissioner to succeed Dr. Jerome M. Jekel, who resigned after 18 years in the office.

Marquette-Jackson Clinic Postgraduate Program.—The annual postgraduate clinic presented by the faculty of the Marquette University School of Medicine, Milwaukee, and the staff of the Jackson Clinic, Madison, will be held Sept. 27, the day of the Wisconsin-Marquette football game. Physicians desiring to attend the meeting and luncheon may write to Dr. Arnold S. Jackson, Chairman, 16 S. Henry St., Madison 3, for a reservation card.

Dr. John S. Hirschboeck, dean of Marquette University School of Medicine, will give a preluncheon address. The medical program will include discussions on eye emergencies, traumatic lesions, common obstetric problems, RH factors in the newborn, indications and contraindication for sympathectomy, and allergy in pediatrics.

GENERAL

Speech Symposium.—The National Society for Crippled Children and Adults, the American Psychological Association's divisions of educational psychology and school psychologists, and the American Speech and Hearing Association will be cosponsors of an all day speech institute at the Statler Hotel, Washington, D. C., Aug. 31, preceding the annual convention of the American Psychological Association in Washington, Sept. 1-5.

Cancer Seminar.—The annual cancer seminar sponsored by the Penrose Cancer Hospital, Colorado Springs, Colo., and the American College of Pathologists will be held at the Broadmoor Hotel in Colorado Springs, Sept. 6. Cases will be considered from the standpoint of radiologic diagnosis followed by discussion of the different histopathological diagnoses submitted.

Dr. Leo G. Rigler, chief, department of radiology and physical medicine, University of Minnesota, Minneapolis, and Dr. Lauren V. Ackerman, professor of surgical pathology and of pathology, Washington University School of Medicine, St. Louis, will be the guest speakers.

Infectious Encephalitis.—According to the U. S. Public Health Service, 90 cases of infectious encephalitis have been reported from 14 counties of California, with 88 from 12 valley counties located in known endemic areas. Since June, of a total of 166 reported, 40 cases have been confirmed by laboratory examination; 39 were found to be western equine encephalomyelitis infections, and 1 was a St. Louis type of disease. There have been 19 deaths. The area of the epidemic extends from Bakersfield to Sacramento. The disease is also present among horses in this area. A small outbreak involving both humans and horses has also been found in Colorado.

Dr. Lorenzo L. Parks, Florida State Board of Health, has reported 10 cases of eastern equine encephalitis among horses and mules in four northeast counties of the state. No human cases were reported from this source.

New Polio Booklets.—The Division of Professional Education of the National Foundation for Infantile Paralysis has issued two new booklets, "Management of Poliomyelitis Patients with Respiratory Difficulty" and "Isolation Techniques and Nursing Care in Poliomyelitis." The former, a digest of a larger manual of the same title, which will be available for limited distribution in the fall, contains material on factors controlling pulmonary ventilation; adverse effects of poliomyelitis on respiration; recognition and treatment of breathing difficulties caused by paralysis of the respiratory muscles; recognition and treatment of breathing difficulties caused by loss of ability to swallow; weaning the patient from the tank respirator; and recognition and treatment of breathing difficulty caused by medullary center involvement.

The 20 page booklet on "Isolation Techniques and Nursing Care in Poliomyelitis" was developed to meet the demand of nurses and other professional groups for information on isolation and nursing procedures. These booklets may be obtained from the foundation's headquarters at 120 Broadway, New York 5.

Chicago Society Offers Postgraduate Courses.—The Chicago Medical Society is sponsoring two postgraduate courses this fall: (1) "Cardiovascular and Renal Diseases" (Sept. 29-Oct. 3), with 55 presentations by a faculty of 34; (2) "Diseases of the Gastrointestinal Tract, Liver and Pancreas" (Oct. 6-10), with 56 presentations by a faculty of 32.

During the luncheons there will be a question and answer period. On Wednesday, a clinical pathological conference will be held from 4:15 to 5:15 p. m. On Thursday, Oct. 2, there will be a dinner at 6:30 p. m. at the Drake Hotel, followed by a scientific meeting at 8 p. m., at which Dr. Paul D. White, Boston, will discuss "Our Ignorance About Heart Disease in Days Gone By."

On Wednesday evening, Oct. 8, there will be a dinner followed by a scientific meeting at which Dr. Waltman Walters, Rochester, Minn., will discuss "Present Day Status of the Surgical Treatment of Gastric and Duodenal Ulcer," and Dr. Burrill B. Crohn, New York, "Early Diagnosis of Gastric Malignancy."

Tuition fee is $75 for each course. For information write the Committee on Postgraduate Medical Education, Chicago Medical Society, 86 E. Randolph St., Chicago 1.

Pediatric Residency Fellowships.—The American Academy of Pediatrics announces that eight fellowships for pediatric residents will be available annually for a period of one year, beginning Jan. 1, 1953. Each recipient will receive $1,000. The awards are made possible by Mr. D. Mead Johnson and Mead Johnson & Company. Criteria for the awards will be (1) a letter from the chief of service, indicating that the proposed person has completed, or will have completed, by Jan. 1, 1953, one year of pediatric training credited by the American Board of Pediatrics; that the resident has performed his work ably; and that there is real need for financial assistance; and (2) a letter from the resident requesting the fellowship, stating that he intends to enter pediatric private practice at the conclusion of his second year of training. Consideration will be given to geographic spread of appointments, so that all sections of the country will be represented, and preference will be given to well-qualified but smaller training centers, which perhaps have fewer resources for assisting residents in training than do some of the larger centers. Seven awards will be made in the United States and one in Canada. Research interests and accomplishments and desire to enter academic pediatrics are not criteria for the awards. Applications must be filed before Oct. 1.

Ophthalmology and Otolaryngology Conference.—A postgraduate conference in ophthalmology and otolaryngology will be held Sept. 15-17 at the Hotel Robert E. Lee, Winston-Salem, N. C., sponsored by the North Carolina Eye, Ear, Nose and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology. Guest speakers and their first presentations are as follows:

John J. Conley, New York, Diagnosis and Treatment of Cancer of the Head and Neck.
Theodore E. Walsh, St. Louis, Treatment of Acute and Chronic Otitis Media.
Frank Turchik, Bridgeport, Conn., Diagnosis of Nasal Obstruction.
Verling K. Hart, Charlotte, N. C., Interesting Benign Tumors of the Tracheobronchial Tree.
Frank W. Newell, Chicago, Fever Therapy in Ocular Disease.
Harold W. Brown, New York, Diagnosis and Treatment, Motor Anomalies, Vertically Acting Muscles.
P. Robb McDonald, Philadelphia, Safety Procedures in Cataract Extraction.
Irving H. Leopold, Philadelphia, Principles and Practical Application of Autonomic Drug Therapy in Ophthalmology.

The social program includes a barbecue at the Old Town Club on Monday at 6 p. m. and a banquet in the ballroom of the Robert E. Lee on Tuesday, preceded by a cocktail party at 6. A fee of $20 will be charged.

Obstetric Seminar in Florida.—An obstetric seminar under the auspices of the maternal welfare committee of the Florida Medical Association and the bureaus of maternal and child health of South Carolina, Georgia, and Florida will be held at the Sheraton Beach Hotel, Daytona Beach, Fla., Sept. 8-10. Speakers will include:

Warren W. Quillian, Coral Gables, Care of the Premature and the Newborn.
R. Gordon Douglas, New York, Bleeding in Pregnancy, Both Early and Late.
Carl P. Huber, Indianapolis, Prolonged Labor with End-Results; Maternal and Fetal Mortality.
William J. Dieckmann, Chicago, Toxemias of Pregnancy.
F. Bayard Carter, Durham, N. C., Management of Obstetric Difficulties.
Edith L. Potter, Chicago, Pulmonary Disturbance in the Newborn.
Fred L. Adair, Maitland, Fla.; Nutrition in Pregnancy.
Clement A. Smith, New York, Care of the Premature and the Newborn.
John Adriani, New Orleans, Anesthesia and Analgesia in Obstetrics.

Tuesday will be devoted to pediatric problems in relation to obstetric management and the newborn. Maternal and fetal mortality surveys in South Carolina, Georgia, and Florida will be discussed by Drs. J. Decherd Guess, Greenville, S. C.; Guy V. Rice, Atlanta, Ga.; Eugene F. McCall, Jacksonville; and John T. Smedley, Miami, Fla.

A. M. A. First Public Relations Institute.—The American Medical Association's first Medical Public Relations Institute will be held Sept. 4 and 5 at the Edgewater Beach Hotel, Chicago. The institute will be a workshop-type conference where public relations personnel, as well as physicians, can get practical information on the day-to-day operations of a medical public relations program. Primarily for lay public relations and employees of state and county medical societies, the institute supplements the annual Medical Public Relations Conference, which will be held Dec. 1, preceding the A. M. A. Clinical Session in Denver. The two day program will be built around five panel discussions: practical ways to increase physician participation in society activities; modernization of the code of ethics to bring about better relations between mass media and the medical profession; development of the best working relationship between the medical profession and the press, radio, and television; tips to the medical profession in working with community groups; and strengthening medical public service activities.

Friday will be devoted to medicine's place in the community. This session will cover the county medical society's place in community affairs, including tips on how to work with voluntary health agencies, other professions, labor, farm groups, schools and the P. T. A., women's clubs, service clubs, and veterans' groups. In the afternoon the institute will break up into roundtable discussions on field service, radio and television programming, newsletter editing, and direct mail techniques.

A special feature of the institute will be a "Project Parade," which will consist of displays of public relations projects of state and county medical societies. These societies have been invited to display pamphlets, printed materials, or photographs of local events. Guests appearing on the program include Mr. Edward Lindsay, Decatur, Ill., editor of the Decatur Newspapers, Inc.; Mr. Jules Herbuveaux, Chicago, manager of television station WNBQ; Mr. Fred R. Jolly, Peoria, Ill., manager of the Community Relations Department, Caterpillar Tractor Co.; Mr. Ben Park, Chicago, president of a radio documentary company; and Mr. Charles R. Jordan, president of a Chicago advertising agency.

Prevalence of Poliomyelitis.—According to the National Office of Vital Statistics, the following number of reported cases of poliomyelitis occurred in the United States, its territories and possessions in the weeks ended as indicated:

Area	Aug. 2, 1952	July 26, 1952	Aug. 4, 1951
New England States			
Maine	2	3	1
New Hampshire	4	1	9
Vermont	..	2	2
Massachusetts	19	8	19
	3	..	..
	16	15	12
Middle Atlantic States			
New York	89	76	65
New Jersey	38	10	17
Pennsylvania	47	24	35
East North Central States			
Ohio	128	179	37
Indiana	41	34	10
Illinois	140	68	81
Michigan	159	79	71
Wisconsin	67	33	34
West North Central States			
Minnesota	113	38	28
Iowa	244	116	14
Missouri	14	33	12
North Dakota	7	1	5
South Dakota	8	15	5
Nebraska	96	72	23
Kansas	83	24	36
South Atlantic States			
	4	4	1
	2	3	1
	8	..	4
Virginia	46	15	11
West Virginia	58	32	9
North Carolina	15	15	20
South Carolina	6	..	3
Georgia	16	18	49
Florida	20	20	16
East South Central States			
	94	51	15
	27	15	30
	18	12	43
	24	54	38
West South Central States			
Arkansas	13	16	23
	51	39	34
	59	65	52
Texas	276	259	92
Mountain States			
Montana	10	8	2
Idaho	11	16	..
Wyoming	9	2	9
Colorado	26	31	71
New Mexico	30	22	2
Arizona	13	7	3
Utah	..	6	23
Nevada	6	..	1
Pacific States			
Washington	50	36	8
Oregon	9	14	14
California	86	75	110
Territories and Possessions			
Alaska	1	..	..
Hawaii	5	10	1
Puerto Rico	..	1	4
Total	2,311	1,677	1,205

FOREIGN

Congress of World Medicine.—The World Congress of Medicine will be held Oct. 16-18 at Montecatini, Italy, under the presidency of Prof. Pietro Verga of Naples. Topics listed for discussion include "Living Conditions and Health Conditions of Populations," "Repercussions of the War on Physical and Mental Health," and "Duties of Physicians in Relation to These Problems."

Cardiovascular Surgeons.—The first meeting of the European Society of Cardiovascular Surgery (European Section of the International Society of Angiology) will be held at the University House in Strasbourg, France, Oct. 5 and 6. Discussions will be confined to the chronic obliterations of arteries, aneurysm and embolism excepted. Physicians desiring to discuss this subject should submit their names no later than Sept. 1 to Prof. R. Fontaine, Clinique Chirurgicale A, Strasbourg.

Embryologists to Meet in The Netherlands.—A three day symposium on "The Biochemical and Structural Basis of Morphogenesis" will open the sessions of the International Institute of Embryology in Utrecht, The Netherlands, Aug. 27. The meetings will be held in the zoological laboratory of the University, Janskerkhof 3, which will also be the scene of the meetings of the general assembly of the institute Aug. 30 and 31; the latter will be open to nonmembers.

Medical College in India.—At the request of authorities of the Indian government, the World Health Organization has designated a principal for the medical college in Trivandrum, State of Travancore, S. Indian Union, naming Dr. Axel Hojer, who retired in June as director-general of health in Sweden after serving in that post for 17 years. He previously taught at Lund University. In addition to serving as principal of the medical college, which opened a year and a half ago and which WHO has termed one of the best equipped in India, he will lecture on social and preventive medicine and on public health.

Meeting of French Otorhinolaryngologists.—The 50th Congress of the French Society of Oto-Rhino-Laryngology will be held in the Grand Amphitheatre de la Maison de la Chimie, 28 rue Saint Dominique in Paris, Oct. 13-16, under the presidency of Prof. Portmann of Bordeaux.

Occupational diseases in the field of otorhinolaryngology and cancer of piriform sinus (therapeutic indications and results) will be discussed. Surgical sessions will be held in the mornings in various hospitals, and there will be an exhibit of instruments and otorhinolaryngologic specialties. Information may be obtained from Dr. H. Flurin, Secretary General, 19, avenue Mac Mahon, 17^{e}, Paris.

World Federation for Mental Health.—This organization will meet in Brussels Aug. 24-30, under the presidency of Dr. Alfonso Millan of México, D. F. Plenary sessions will be concerned with a report on the WFMH seminar on "Mental Health and Infant Development" recently held at Chichester, England, "Mental Health Problems of Old Age," "Social Case Work," and "Research and Developments in the International Mental Health Field." Groups will meet daily to discuss "Techniques of Mental Hygiene in Schools for Normal and for Abnormal Children"; "Education of the Public in Principles of Mental Health"; "Practical Measures for Dealing with the Mental Health Problems of Refugees and Displaced Persons"; "Techniques of Mental Hygiene in the Industrial Field"; "Formulation of the Unsolved Problems of Mental Hygiene"; and "Public Health and Mental Health."

DEATHS IN OTHER COUNTRIES

Dr. Jules Tinel of Paris, neurologist, died March 4 at the age of 72. Dr. Tinel in 1911 became chief of Déjerine's Clinic. In 1912 he was appointed chief of the pathological laboratory of the clinic for nervous diseases and from 1915 to 1918 was chief of the neurologic center at Mans. Among Dr. Tinel's more important works are "Injuries of the Nerves," "Endocrinologic Clinic," and "The Nervous Vegetative System."——**Dr. Carlos Comba**, Italian pediatrician, died in Florence, Italy, Nov. 4, 1951, at the age of 80. In 1894 he joined the pediatric department of Professor Mya in Florence, where he remained until 1903, when he was appointed professor of pediatrics at Bologna. In 1911, he returned to Florence as professor, remaining in that position until his retirement in 1943. He was one of the founders of La Societa Italiana de Pediatria and its president from 1920 to 1924. He was editor of the *Revista de clinica pediatrica* for many years. Dr. Comba's clinical contributions covered many fields, notably diphtheria, tuberculosis, meningitis, and measles.

MEETINGS

AMERICAN MEDICAL ASSOCIATION PUBLIC RELATIONS INSTITUTE, Edgewater Beach Hotel, Chicago, Sept. 4-5. Mr. Leo E. Brown, 535 N. Dearborn St., Chicago 10, Director.

AMERICAN ACADEMY FOR CEREBRAL PALSY, Durham, N. C., Oct. 2-4. Dr. Meyer A. Perlstein, 4743 North Drake Ave., Chicago 25, Secretary.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, Chicago, Oct. 12-17. Dr. W. L. Benedict, 100 First Avenue Bldg., Rochester, Minn., Executive Secretary.

AMERICAN ASSOCIATION OF BLOOD BANKS, Hotel Schroeder, Milwaukee, Oct. 9-11. Miss Marjorie Saunders, 3500 Gaston Avenue, Dallas 1, Texas, Secretary.

AMERICAN ASSOCIATION OF MEDICAL RECORD LIBRARIANS, Shoreham Hotel, Washington, D. C., Oct. 13-17. Miss Doris Gleason, 510 N. Dearborn St., Chicago 10, Executive Secretary.

AMERICAN ASSOCIATION OF OBSTETRICIANS, GYNECOLOGISTS AND ABDOMINAL SURGEONS, The Homestead, Hot Springs, Va., Sept. 4-6. Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Texas, Secretary.

AMERICAN CLINICAL AND CLIMATOLOGICAL ASSOCIATION, Elmira, N. Y., Oct. 16-18. Dr. Marshall N. Fulton, 124 Waterman St., Providence 6, R. I., Secretary.

AMERICAN COLLEGE OF SURGEONS,Waldorf-Astoria, New York, Sept. 22-26. Dr. Michael L. Mason, 40 East Erie St., Chicago 11, Secretary.

AMERICAN CONGRESS OF PHYSICAL MEDICINE, Roosevelt Hotel, New York, Aug. 25-29. Dr. Frances Baker, One Tilton Ave., San Mateo, Calif., Secretary.

AMERICAN HOSPITAL ASSOCIATION, Philadelphia, Sept. 15-18. Mr. George Bugbee, 18 East Division St., Chicago 10, Executive Director.

AMERICAN MEDICAL WRITERS' ASSOCIATION, Jefferson Hotel, St. Louis, Oct. 1. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

AMERICAN PHYSIOLOGICAL SOCETY, New Orleans, Sept. 4-6. Dr. E. F. Adolph; University of Rochester School of Medicine and Dentistry, Rochester, N. Y., Secretary.

AMERICAN ROENTGEN RAY SOCIETY, Shamrock Hotel, Houston, Texas, Sept. 23-26. Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Secretary.

AMERICAN SOCIETY OF CLINICAL PATHOLOGISTS, Chicago, Oct. 13-17. Dr. Clyde G. Culbertson, 1040 W. Michigan St., Indianapolis, Secretary.

AMERICAN SOCIETY OF MAXILLOFACIAL SURGEONS, Hotel del Coronado, San Diego, Calif., Sept. 10-13. Dr. Casper M. Epsteen, 25 East Washington St., Chicago 2, Secretary.

ANNUAL GULF COAST REGIONAL CONFERENCE ON INDUSTRIAL HEALTH, Rice Hotel, Houston, Tex., Sept. 24-27. Dr. W. H. Hamrick, P. O. Box 2371, Houston, Tex., Chairman.

BIOLOGICAL PHOTOGRAPHIC ASSOCIATION, Hotel New Yorker, New York, Sept. 10-12. Mr. Lloyd E. Varden, Pavelle Color, Inc., 533 W. 57th St., New York 19, Secretary.

COLLEGE OF AMERICAN PATHOLOGISTS, Drake Hotel, Chicago, Oct. 13-17. Dr. M. G. Westmoreland, 203 N. Wabash Ave., Chicago 1, Executive Secretary.

COLORADO STATE MEDICAL SOCIETY, Stanley Hotel, Estes Park, Sept. 9-12. Mr. Harvey T. Sethman, 835 Republic Bldg., Denver 2, Executive Secretary.

CONSTANTINIAN SOCIETY, Lake Placid Club, Lake Placid, N. Y., Oct. 1-4. Dr. C. F. Shook, P. O. Box 1035-36, Toledo 1, Ohio, Secretary.

DELAWARE, MEDICAL SOCIETY OF, Rehoboth, Sept. 8-10. Dr. Andrew M. Gehret, 822 North American Bldg., Wilmington, Secretary.

DISTRICT OF COLUMBIA, MEDICAL SOCIETY OF THE, Hotel Statler, Washington, Sept. 29-Oct. 1. Mr. Theodore Wiprud, 1718 M St. N.W., Washington, Secretary.

GULF COAST CLINICAL SOCIETY, San Carlos Hotel, Pensacola, Fla., Oct. 16-17. Dr. Dale E. York, 21½ E. Wright St., Pensacola, Fla., Secretary.

KANSAS CITY SOUTHWEST CLINICAL SOCIETY, Kansas City, Mo., Oct. 6-9. Dr. Galen M. Tice, 630 Shukert Bldg., Kansas City, Kansas, Secretary.

KENTUCKY STATE MEDICAL ASSOCIATION, Brown Hotel, Louisville, Oct. 7-9. Dr. Bruce Underwood, 620 S. Third St., Louisville 2, Secretary.

MICHIGAN STATE MEDICAL SOCIETY, Book-Cadillac Hotel, Detroit, Sept. 24-26. Dr. L. Fernald Foster, 606 Townsend St., Lansing 15, Secretary.

MISSISSIPPI VALLEY MEDICAL SOCIETY, Jefferson Hotel, St. Louis, Oct. 1-3. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

MONTANA MEDICAL ASSOCIATION, Florence Hotel, Missoula, Sept. 18-21. Mr. L. R. Hegland, 104 North Broadway, Billings, Executive Secretary.

NEUROSURGICAL SOCIETY OF AMERICA, The Cloister, Sea Island, Ga., Sept. 18-20. Dr. C. D. Hawkes, 22 North Manassas St., Memphis 5, Tenn., Secretary.

NEW HAMPSHIRE MEDICAL SOCIETY, Mt. Washington Hotel, Bretton Woods, Sept. 7-9. Dr. Deering G. Smith, 44 Chester St., Nashua, Secretary.

NORTH PACIFIC PEDIATRIC SOCIETY, Challenger Inn, Sun Valley, Idaho, Sept. 19-20. Dr. S. Gorham Babson, 1107 S.W. 13th Ave., Portland 5, Ore., Secretary.

NORTH PACIFIC SOCIETY OF INTERNAL MEDICINE, Sun Valley, Idaho, Sept. 19-20. Dr. Robert L. King, 1115 Terry Ave., Seattle 1, Wash., Secretary.

NORTH TEXAS-SOUTHERN OKLAHOMA FALL CLINICAL CONFERENCE, Wichita Falls, Texas, Sept. 17. Dr. E. Aubrey Cox, Hamilton Bldg., Wichita Falls, Texas, Chairman.

OREGON STATE MEDICAL SOCIETY, Portland, Oct. 8-11. Dr. Robert F. Miller, 833 S.W. 11th Ave., Portland 5, Secretary.

PACIFIC DERMATOLOGIC ASSOCIATION, Palace Hotel, San Francisco, Aug. 29-30. Dr. Ervin H. Epstein, 447 29th St., Oakland, Calif., Secretary.

Pennsylvania, Medical Society of the State of, Bellevue-Stratford Hotel, Philadelphia, Sept. 28-Oct. 2. Dr. Walter F. Donaldson, 500 Penn Ave., Pittsburgh 22, Secretary.

Tri-State Medical Society of Texas, Louisiana and Arkansas, Texarkana, Texas, Oct. 2-3. Dr. Albert M. Hand, 619 Main St., Texarkana, Texas, *Secretary.*

U. S. Chapter, International College of Surgeons, Conrad Hilton Hotel, *Chicago, Sept. 2-5. Dr. Arnold S. Jackson,* 1516 Lake Shore Drive, Chicago, Executive Secretary.

Utah State Medical Association, Salt Lake City, Sept. 4-6. Dr. T. C. Weggeland, 42 S. Fifth East St., Salt Lake City, Secretary.

Vermont State Medical Society, Mt. Washington Hotel, Bretton Woods, N. H., Sept. 7-9. Dr. James P. Hammond, 128 Merchants Row, Rutland, Secretary.

Virginia, Medical Society of, Jefferson Hotel, Richmond, Sept. 28-Oct. 1. Mr. Robert L. Howard, 1105 West Franklin St., Richmond, Executive *Secretary.*

Washington State Medical Association, Olympic Hotel, Seattle, Sept. 13-17. Dr. Bruce Zimmerman, 338 White-Henry-Stuart Bldg., Seattle, *Secretary.*

Wisconsin, State Medical Society of, Hotel Schroeder, Milwaukee, Oct. 6-8. Mr. Charles H. Crownhart, 704 E. Gorham St., Madison, Secretary.

INTERNATIONAL

Aero Medical Association Interim Meeting, Paris, France, Sept. 26-28. Dr. Armand Robert, 2 Rue Marbeuf, Paris 8e, France, Secretary.

American Congress of Industrial Medicine, Rio de Janeiro, Brazil, Sept. 20-28. Prof. Dr. Jose Pedro Reggi, Calle Arenales No. 981 Buenos Aires, Argentina, Director General.

Australasian Medical Congress, Melbourne Victoria, Australia, Aug. 22-30. Dr. C. H. Dickson, Medical Society Hall, 426 Albert St., East Melbourne, Victoria, Australia, General Secretary.

Congress of International Anesthesia Research Society, Virginia Beach, Virginia, U. S. A., Sept. 22-25.

Congress of International Institute of Embryology, University of Utrecht, Zoological Laboratory, Utrecht, Netherlands, Aug. 27-31. Dr. M. W. Woerdeman, Amsterdam, Holland, President.

European Congress of Cardiology. University of London, Bloomsbury, W.C.1, England, Sept. 10-12. Dr. K. Shirley Smith, 35 Wimpole St., *London,* W.1, England, Secretary.

European Society of Cardiovascular Surgery, Strasbourg, France, Oct. 3-4. Sir James Learmonth, Department of Surgery, University New Buildings, Edinburgh 8, Scotland.

Interamerican Cardiological Congress, Buenos Aires, Argentina, Aug. 31-Sept. 7. Dr. Blas Moia, Larrea 1132, Buenos Ajres, Argentina, Secretary-General.

Inter-American Congress of Public Health, Havana, Cuba. Sept. 26-Oct. 1. Pan-American Sanitary Bureau, 1501 New Hampshire Ave. N.W., Washington 6, D. C.

Inter-American Congress of Radiology, Mexico City, Mexico, Nov. 2-8. Dr. Guido Torres Martinez, Marsella No. 11, Mexico, D.F., Secretary-General.

International Congress of Anthropology and Ethnology, Vienna, Austria, Sept. 1-8. For information write: Secretaries, Royal Anthropological Institute, 52 Upper Bedford Place, London, W.C.1, England.

International Congress on Diseases of the Chest, Rio de Janeiro, Brazil, Aug. 24-30. Mr. Murray Kornfeld, 112 East Chestnut St., Chicago 11, Illinois, Executive Secretary.

International Congress of Haematology, Mar del Plata, Argentina, Sept. 21-26. Dr. Carlos Reussi, *Anchorena 1710,* Buenos Aires, Argentina, Secretary.

International Congress of the History of Medicine, Nice-Cannes, France, and Monaco, Sept. 8-14. Dr. F. A. Sondervorst, 124 Avenue des Allies, Louvain, Belgium, Secretary-General.

International Congress on Hydatid Disease, Santiago, Chile, Nov. 21-24. Organizing Committee, P. O. Box 9183, Santiago, Chile.

International Congress of Internal Medicine, Friends House, London, N.W.1, *Eng. Sept. 15-18.* Sir Harold Boldero, 12 Pall Mall East, London, S.W.1, England, Secretary.

International Congress of Medical Press, Venice, Italy, Oct. 4-5. Dr. Bruno de Biasio, S. Marco 4646, Venice, Italy, *Secretary.*

International Congress on Medical Records, London, England, Sept. 7-12. Miss Gwen Perkins, 120 C Street S.E., Washington, D. C., Chairman.

International Congress of Military Medicine and Pharmacy, Montevideo, Uruguay, March 1-7, 1953. Dirreccion General del *Servico de Sanitad Militar, 8 de Octubre y Mariano* Moreno, Montevideo, Uruguay, Secretariat of the Congress.

International Congress on Neuropathology, Rome, Italy, Sept. 8-13. Dr. Armando Ferraro, 722 W. 168th St., New York, N. Y., U. S. A., Secretary General.

International Congress of Otorhinolaryngology, *Amsterdam,* Netherlands, *June 8-15, 1953.* Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam, Netherlands, Secretary.

International Congress of Radiology, Copenhagen, Denmark, July 19-25, 1953. Professor Flemming Norgaard, 10 Oster Yoldgade, Copenhagen K., Denmark, Secretary General.

International Congress of Therapeutics, Madrid, *Spain, Oct. 16-18.* Professor Armijo, Instituto de Farmacologia, Cuidad Universitaria, Madrid, Spain, Secretary.

International Congress of Urology, The Waldorf-Astoria, New York, N. Y., U. S. A., Sept. 15-18. Dr. John A. Taylor, 2 East 54th St., New York, N. Y., *U. S. A., Secretary-General.*

International Hospital Congress, London, *England, May 25-30, 1953.* Capt. J. E. Stone, 10 Old Jewry, London, EC2, England, Hon. Secretary.

International Study Conference on Child Welfare, Bombay, India, Dec. 5-12. For information write: All India Save the Children Committee, 5 Carmichael Road, Bombay, India.

International Union Against Tuberculosis, Rio de Janeiro, Brazil, Aug. 24-27. Prof. Etienne Bernard, 66 Blvd. St. Michel, Paris 6e, France, *Secretary-General.*

Medical Women's International Association, Vichy, France, Sept. 12-16. Dr. Margaret M. Wurts, 27 East Wellesley Road, Montclair, N. J., U. S. A., National Secretary.

Neuroradiological Symposium, Stockholm, Sweden, Sept. 17-20. Docent Ake Lindbom, Symposium Neuroradiologicum, Serafimerlasarettet, Stockholm K., Sweden, Secretary.

Pan American Congress of the Medical Press, Buenos Aires, Argentine, July 12-16, 1953. Secretaria del Congress, 763 *Uriburu, Buenos Aires,* Argentine.

World Congress of Medicine, Montecatini, Italy, Oct. 16-18. Secretariat: Corso Trieste, 65, Rome, Italy.

World Federation of Mental Health, Brussels, Belgium, Aug. 25-Sept. 1. Dr. J. R. Rees, 19 Manchester St., London, W.1, England, Secretary.

World Medical Association, Athens, Greece, Oct. 12-16. Dr. Louis H. Bauer, 2 East 103d St., New York 29, N. Y., Secretary General.

EXAMINATIONS AND LICENSURE

NATIONAL BOARD OF MEDICAL EXAMINERS

National Board of Medical Examiners: *Part I,* Sept. 8-10. All centers where there are five or more candidates. Exec. Sec., Dr. John P. Hubbard, 225 S. 15th St., Philadelphia 2.

EXAMINING BOARDS IN SPECIALTIES

American Board of Anesthesiology: *Oral.* Swampscott, Mass., Sept. 28-Oct. 1. Sec., Dr. C. B. Hickcox, 80 Seymour St., Hartford 15.

American Board of Dermatology and Syphilology: *Written.* Various Centers, September 1953. Final *date for filing applications* was May 1. All candidates are now required to take the written examination. *Oral.* Philadelphia, October 1953. Ex. Sec., Miss Janet Newkirk, 66 East 66th St., New York 21.

American Board of Internal Medicine: *Written.* Various centers and overseas, *Oct. 20. Closing date for the acceptance of applications for the written examination was May 1. Oral.* San Francisco, Sept. 3-6; Philadelphia, Nov. 13-14 and Washington, D. C., Nov. 17-18. Exec. Sec.-Treas., Dr. William A. Werrell, 1 West Main Street, Madison 3.

American Board of Neurological Surgery: *Written.* New Haven, Sept. 19-21. *Oral.* Chicago, May or June 1953. Final date for filing application for the oral examination is Jan. 15, 1953. Sec., Dr. W. J. German, 789 Howard Ave., New Haven 4, Conn.

American Board of Obstetrics and Gynecology: *Written.* Various Centers, Feb. 6, 1953. Final date for filing applications is Nov. 1. Sec., Dr. R. L. Faulkner, 2105 Adelbert Road, Cleveland 6.

American Board of Ophthalmology: *Written.* Various Centers, January 1953. Final date for filing applications was July 1. *Practical.* Chicago, Oct. 6-10 and New York City, June 6-10, 1953. Sec., Dr. Edwin B. Dunphy, 56 Ivie Road, Cape Cottage, Maine.

American Board of Orthopaedic Surgery: The deadline for the receipt of applications for the 1953 Part II examinations is Aug. 15. Sec., Dr. Harold A. Sofield, 122 S. *Michigan* Ave., Chicago.

American Board of Otolaryngology: *Written.* Chicago, Oct. 6-10. *Oral.* Chicago, Oct. 6-10 and New Orleans, *April 21-25,* 1953. Sec., Dr. Dean M. Lierle, University Hospital, Iowa City.

American Board of Pathology: *Written and Oral.* Pathologic Anatomy and Clinical Pathology examination. Chicago, Oct. 9-11. Sec., Dr. William B. Wartman, 303 E. Chicago Ave., Chicago 11.

American Board of Pediatrics: *Oral.* Chicago, *Oct. 24-26; Boston, Dec.* 5-7. Exec. Sec., Dr. John McK. Mitchell, 6 Cushman Road, Rosemont, Pa.

American Board of Plastic Surgery: *Oral and Written.* New York, Sept. 26-28. Final date for filing of case reports is Aug. 15. Sec., Dr. Bradford Cannon, 4647 Pershing Ave., St. Louis 8.

American Board of Preventive Medicine and Public Health: Cleveland, Oct. 18-20. Sec., Dr. E. L. Stebbins, 615 N. Wolfe St., Baltimore.

American Board of Proctology: *Oral and Written.* Philadelphia, Sept. 27-28. Sec., Dr. Louis A. Buie, 102-110 *Second Ave., S.W., Rochester,* Minn.

American Board of Psychiatry and Neurology: New *York City, Dec.* 15-16. *Final date for filing applications* is Sept. 15. Sec.-Treas., Dr. David A. Boyd, Jr., 102-110 *Second Ave. S.W., Rochester, Minn.*

American Board of Radiology: *Oral.* Dec. 1-7. Final date for filing application was June 1. *Oral.* Tampa, *April 8-15. Sec., Dr. B. R. Kirklin,* 102-110 Second Ave., S.W., Rochester, Minn.

American Board of Surgery: *Written.* Various Centers. *October 1952.* Final date for filing application was July 1. *Written.* Various Centers, March 1953. Final date for filing *application is Dec. 1.* Sec., *Dr. J.* Stewart Rodman, 225 S. 15th St., Philadelphia.

American Board of Urology: *Written.* Various Centers, *December. Oral.* Chicago, Feb. 7-11. Sec., Dr. Harry Culver, 30 Westwood Road, Minneapolis 16, Minn.

Board of Thoracic Surgery: *Written.* Various Centers, Sept. 5. Sec., Dr. William M. Tuttle, 1151 Taylor Ave., Detroit 2.

DEATHS

Van Hoosen, Bertha ⊕ Rochester, Mich.; born in Rochester, March 26, 1863; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1888; began practice in 1892 in Chicago where she was professor and head of the department of obstetrics at Loyola University School of Medicine from 1918 to 1937, when she became professor emeritus, professor of clinical gynecology at the College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, from 1902 to 1912, and professor of gynecology and embryology at Northwestern University Woman's Medical School from 1894 to 1902; from 1915 to 1918 first president of the Medical Women's National Association, now known as the American Medical Women's Association; member of the Illinois State Medical Society, Chicago Pathological Society, Illinois Academy of Science, Medical Women's International Association, and Daughters of the American Revolution; honorary member of the International College of Surgeons and the International Association of Medical Women; fellow of the American College of Surgeons; served as consultant to Women and Children's Hospital and Lewis Memorial Maternity Hospital in Chicago; formerly affiliated with Misericordia, Provident, Woman's, Mary Thompson, Cook County, and Frances E. Willard hospitals all in Chicago, and Oak Park (Ill.) Hospital; received the honorary degree of master of arts from her alma mater in 1912 and an LL.D. degree from Loyola University in 1926; formerly editor of the *Medical Woman's Journal;* author of "Petticoat Surgeon" published in 1947; died June 7, aged 89.

Bartley, Samuel Potter ⊕ Brooklyn; born in Brooklyn June 6, 1890; Long Island College Hospital, Brooklyn, 1914; clinical associate professor of surgery at University of New York Medical Center; member of the American Association for the Surgery of Trauma and the Industrial Medical Association; fellow of the American College of Surgeons; founder and chairman of the Brooklyn Cancer Committee of the American Cancer Society; for his service during World War I, in 1925 was made a Chevalier of the Legion of Honor by France; affiliated with Kingston Avenue Hospital and House of St. Giles the Cripple in Brooklyn and Huntington (N. Y.) Hospital; died in Long Island College Hospital June 10, aged 62, of leukemia.

Alexander, William Franklin ⊕ Terrell, Texas; Southwestern University Medical College, Dallas, 1905; owner of a hospital bearing his name; died June 6, aged 73.

Alexander, William Graham ⊕ Peoria, Ill.; Northwestern University Medical School, Chicago, 1904; served during World War I; formerly practiced in Evanston, where he was a member of the board of health and school board and affiliated with Evanston and St. Francis hospitals; died June 14, aged 81, of cerebral hemorrhage.

Anderson, Oliver Wendell ⊕ Luverne, Minn.; University of Minnesota Medical School, Minneapolis, 1936; died in Minneapolis April 28, aged 40, of coronary occlusion.

Andrew, James ⊕ Cordova, Ala.; University of Georgia School of Medicine, Augusta, 1922; died March 26, aged 53.

Arthur, Herbert Spencer ⊕ McKeesport, Pa.; Northwestern University Medical School, Chicago, 1902; at one time mayor of McKeesport; formerly on the city school board; vice-president and director of the Daily News Publishing Company of McKeesport; president and director of the Massillon (Ohio) Steel Casting Company; director of the First National Bank of McKeesport; affiliated with McKeesport Hospital; died in Fort Louden June 12, aged 73, of coronary occlusion.

Bach, Arthur ⊕ Lexington, Ky.; University of Louisville Medical Department, 1917; affiliated with St. Joseph Hospital and Good Samaritan Hospital, where he died June 8, aged 58, of pulmonary embolism, following a gastrectomy for duodenal ulcer.

Baketel, Roy Vincent ⊕ Salem, N. H.; Dartmouth Medical School, Hanover, N. H., 1899; member of the Massachusetts Medical Society; formerly practiced in Methuen, where he was school physician, affiliated with Nevins Home for the Aged, and a director of the Methuen National Bank; for many years on the staff of the Lawrence (Mass.) General Hospital; died May 26, aged 75, of a heart attack.

Barnes, Herbert Trumbull ⊕ Delafield, Wis.; Rush Medical College, Chicago, 1903; past president of the Waukesha County Medical Society; served during World War I; for many years health officer of Delafield; resident physician and medical officer at St. John's Military Academy; affiliated with Waukesha (Wis.) Memorial Hospital; died May 15, aged 76, of cancer.

Berwald, William Paul Eugene ⊕ Patton, Calif.; Syracuse University College of Medicine, 1935; affiliated with Redlands Community Hospital in Redlands and Patton State Hospital; died in St. Bernardine's Hospital in San Bernardino June 11, aged 41.

Bowser, William Francis ⊕ Davenport, Iowa; State University of Iowa College of Medicine, Iowa City, 1905; died June 13, aged 79, of carcinoma of the stomach with metastasis and arteriosclerotic heart disease.

Bradner, Frank William ⊕ Brooklyn; Bellevue Hospital Medical College, New York, 1889; active in the National Guard and served as a major in the 13th regiment; for many years affiliated with Kings County Hospital, where he died May 4, aged 83, of cardiovascular disease.

Braislin, Doris Orlenda Tuttle, Rutland, Vt.; Woman's Medical College of Pennsylvania, Philadelphia, 1922; certified by the National Board of Medical Examiners; member of the American Psychiatric Association; died in New York April 11, aged 57, of carcinoma.

Breister, Benjamin Walter ⊕ Chicago; Rush Medical College, Chicago, 1928; on the staff of the Norwegian American Hospital; died in Waukegan, Ill., July 5, aged 59, in an automobile accident.

Breivis, John James ⊕ Binghamton, N. Y.; University of Rochester School of Medicine and Dentistry, Rochester, 1933; for many years county coroner; affiliated with Wilson Memorial Hospital in Johnson City and Binghamton City Hospital, where he died May 8, aged 47, of dissecting aneurysm of the aorta.

Brennan, Albert Edward ⊕ Buffalo; Niagara University Medical Department, Buffalo, 1898; served as health officer of the village of Sloan; on the staff of Emergency Hospital; surgeon for the Delaware, Lackawanna and Western Railroad and Lehigh Valley Railroad for many years; died May 2, aged 77, of dilatation of the heart.

Brooks, Charles Sumner, Mount Union, Pa.; Howard University College of Medicine, Washington, D. C., 1910; died May 17, aged 76.

Brookshire, William Henry, Phoenix, Ariz.; Columbian Medical College, Kansas City, Mo., 1901; died June 11, aged 78, of arteriosclerotic heart disease.

Brown, Stewart Dixon ⊕ Royston, Ga.; Atlanta College of Physicians and Surgeons, 1907; for many years chairman of the board of education; medical superintendent of Cobb Memorial Hospital; served during World War I; died May 20, aged 71, of pulmonary infarction and arteriosclerosis.

Brown, William Lee Jr. ⊕ Chicago; University of Chicago, the School of Medicine, 1942; served in the regular navy during World War II; on the staff of the Illinois Central Hospital, where he died July 10, aged 34, of cor pulmonale.

Brucker, Edward Arthur ⊕ Chicago; University of Illinois College of Medicine, Chicago, 1916; on the staff of St. Anne's Hospital; died May 27, aged 62, of coronary occlusion.

⊕ Indicates Member of the American Medical Association.

Brumbaugh, Gaius Marcus ⊕ Washington, D. C.; Howard University College of Medicine, Washington, 1885; Georgetown University School of Medicine, Washington, 1888; for many years president and later president emeritus of the board of trustees of Juniata College in Huntington, Pa.; died May 14, aged 90, of hypertensive cardiovascular disease.

Bryson, Edmond Jones, Liberty, S. C.; Bennett Medical College, Chicago, 1913; for many years mayor of Liberty; served during World War I; formerly health officer of Greenwood, Oconee, and Pickens counties; died May 29, aged 66, of cerebral hemorrhage.

Cannon, John Henry, Shreveport, La.; University of the South Medical Department, Sewanee, Tenn., 1900; past president of the city board of health; formerly city health officer; died May 5, aged 85, of arteriosclerotic heart disease.

Childers, Joseph Edward, Tipton, Okla.; Vanderbilt University School of Medicine, Nashville, Tenn., 1915; county health officer; formerly a physician in the Indian Service; examiner for the selective service board during World War II; physician for the Missouri, Kansas and Texas Railway; died June 1, aged 65, of coronary disease.

Ciccone, George Vico, Philadelphia; Medico-Chirurgical College of Philadelphia, 1901; also a graduate in pharmacy; member of the selective service board during World Wars I and II; died May 9, aged 76, of carcinoma of the prostate.

Collier, Thomas Jefferson ⊕ Atlanta, Ga.; Atlanta College of Physicians and Surgeons, 1900; chairman, Section on Anesthesiology, American Medical Association, 1941-1942; past president of the Associated Anesthetists of the United States and Canada; member of the American Society of Anesthesiologists; specialist certified by the American Board of Anesthesiology; served during World War I; affiliated with Piedmont Hospital, where he died March 10, aged 77, of cerebral hemorrhage.

Colson, Dell Cassidy, Glenwood, Ga.; University of Georgia School of Medicine, Augusta, 1914; died May 16, aged 70, of carcinoma of the bladder.

Cook, Samuel LeCount, Washington, D. C.; University of Illinois College of Medicine, Chicago, 1913; clinical assistant professor of gynecology at Howard University College of Medicine; died in Rochester, Minn., June 11, aged 64.

Davies, Morgan Cryder ⊕ Columbus, Ohio; Ohio State University College of Medicine, Columbus, 1927; specialist certified by the American Board of Ophthalmology; member of the American Academy of Ophthalmology and Otolaryngology; affiliated with White Cross Hospital and Children's Hospital; died May 26, aged 56, of myocardial infarction.

Dennis, Thomas Edmund ⊕ Monroeville, Ala.; University of the South Medical Department, Sewanee, Tenn., 1908; died March 2, aged 78, of acute coronary occlusion.

Donn, Frederick Young, Washington, D. C.; George Washington University School of Medicine, Washington, D. C., 1915; died June 7, aged 63, of arteriosclerotic heart disease.

DuBose, David J., Purvis, Miss.; Mississippi Medical College, Meridian, 1910; died in Methodist Hospital, Hattiesburg, May 21, aged 72, of arteriosclerosis.

Fitz, Erwin Otto L., Milwaukee; Marquette University School of Medicine, Milwaukee, 1914; died in Veterans Administration Hospital, Marion, Ind., March 17, aged 62, of pulmonary tuberculosis.

Fleck, Harry Willard ⊕ Stratford, Conn.; Jefferson Medical College of Philadelphia, 1896; formerly practiced in Bridgeport, where he was affiliated with St. Vincent's and Bridgeport hospitals; died May 8, aged 79, of chronic myocarditis and arteriosclerosis.

Flournoy, Harrison Clinton ⊕ Warwick, Ga.; Vanderbilt University School of Medicine, Nashville, Tenn., 1911; died May 4, aged 66, of coronary infarction.

Friesleben, William ⊕ Sauk Rapids, Minn.; Medical College of Ohio, Cincinnati, 1906; past president of the Stearns-Benton Counties Medical Society; past president of the Benton County Fair Association; served on the school board; member of the American Academy of General Practice; died May 20, aged 73, of angina pectoris.

Fulda, Harry Christian, Brooklyn; Columbia University College of Physicians and Surgeons, New York, 1908; died May 21, aged 66, of coronary occlusion.

Gallupe, Harold Quimby ⊕ Waltham, Mass.; Harvard Medical School, Boston, 1918; secretary of the Massachusetts Medical Society; formerly secretary of the state board of registration in medicine; served on the staff of the Waltham Hospital, where he died May 29, aged 63, of cancer.

Gauen, George Otto ⊕ St. Louis; Washington University School of Medicine, St. Louis, 1904; specialist certified by the American Board of Obstetrics and Gynecology; fellow of the American College of Surgeons; served as chief of obstetrics at Lutheran Hospital, where he died June 6, aged 71, of uremia and heart disease.

Gifford, Allen William ⊕ Springfield, Mo.; Central College of Physicians and Surgeons, Indianapolis, 1901; died in Burge Hospital April 3, aged 78, of arteriosclerotic heart disease.

Gossom, Kelley D. ⊕ Clinton, Okla.; Hospital College of Medicine, Louisville, 1903; died in Western Oklahoma State Hospital May 15, aged 74, of acute myocardial insufficiency.

Gregory, William Grant, Tampa, Fla.; Rush Medical College, Chicago, 1891; died in Bay Pines recently, aged 88, of adenocarcinoma of the stomach with metastases to liver, lungs, and regional lymph nodes.

Griffis, Frank C. ⊕ Major, U. S. Army, retired, Fruitland Park, Fla.; Ohio Medical University, Columbus, 1896; entered the medical corps of the U. S. Army in 1917 and retired Nov. 30, 1930; died recently, aged 85, of arteriosclerotic heart disease.

Group, George Abraham, Syracuse, N. Y.; Syracuse University College of Medicine, 1919; associated with the city health department; served as assistant professor of medicine (syphilology) at his alma mater; died in St. Joseph's Hospital May 18, aged 59, of acute myocardial infarction.

Guild, William Alva ⊕ Coral Gables, Fla.; Chicago Homeopathic Medical College, 1903; Kansas City College of Medicine and Surgery, Kansas City, Mo., 1922; member of the Illinois State Medical Society; died in Miami Springs recently, aged 72, of hypertension and cancer of the large bowel.

Gullo, Charles ⊕ Buffalo; Loyola University School of Medicine, Chicago, 1928; past president of the Livingston County Medical Society; certified by the National Board of Medical Examiners; formerly affiliated with Dansville (N. Y.) Memorial Hospital; died in Oneida, N. Y., April 12, aged 52.

Gustafson, Axel Walfred ⊕ Gustine, Calif.; College of Physicians and Surgeons, Los Angeles, 1922; served during World War I; health officer and school physician; died in West Side Hospital in Newman May 14, aged 71, of cerebral hemorrhage.

Halpern, Harry Samuel, Philadelphia; Jefferson Medical College of Philadelphia, 1911; for many years medical inspector of the board of education; died June 2, aged 63.

Ham, Cornelius, Baltimore; Atlantic Medical College, Baltimore, 1910; died recently, aged 83, of cerebral hemorrhage.

Hatcher, George Edward ⊕ Cerulean, Ky.; University of Tennessee Medical Department, Nashville, 1906; member of the American Academy of General Practice; past president of the Trigg County Medical Society; served as a member of the county board of health; died May 25, aged 69, of a heart attack.

Hazard, John David ⊕ Albany, N. Y.; New York Medical College, Flower and Fifth Avenue Hospitals, in New York, 1942; certified by the National Board of Medical Examiners; served during World War II; resident at Veterans Administration Hospital, where he died June 22, aged 36.

Holbrook, Charles Albert ⊕ Haverhill, Mass.; Harvard Medical School, Boston, 1908; affiliated with Hale Hospital, where he died April 9, aged 73, of arteriosclerotic heart disease.

Holt, Rufus Andrew ⊕ Los Angeles; College of Physicians and Surgeons, Medical Department of the University of Southern California, Los Angeles, 1915; affiliated with California Lutheran Hospital; died May 16, aged 61.

Hoppe, Herman C., Columbus, Ohio; Ohio Medical University, Columbus, 1901; also a graduate in pharmacy; died May 30, aged 82, of pulmonary edema.

Isbell, Jesse E. D. ⊕ Toccoa, Ga.; Atlanta College of Physicians and Surgeons, 1901; served overseas during World War I; died April 30, aged 75.

Jenson, James ⊕ Saybrook, Ill.; Chicago College of Medicine and Surgery, 1911; died in Bloomington recently, aged 74, of coronary heart disease.

Johnson, Arnold Edwin ⊕ Minneapolis; Rush Medical College, Chicago, 1902; died in St. Barnabas Hospital recently, aged 76, of cerebral hemorrhage and hypertension.

Jonnes, Lloyd ⊕ Circleville, Ohio.; University of Cincinnati College of Medicine, 1918; served during World War I; a captain in the Ohio National Guard for many years; West Virginia state medical inspector for Civilian Conservation Corps from 1935 to 1937; coroner of Pickaway County since 1945; district medical officer, Norfolk and Western and Chesapeake and Ohio Railroads; president, Ohio History Day Association; owner, Park Place Press; died May 21, aged 62.

Kelley, Clarence Joseph, Washington, D. C.; Georgetown University School of Medicine, Washington, 1936; died in Providence Hospital July 3, aged 41, of bleeding gastric ulcer.

Kelty, Harvey Egbert, Chico, Calif.; Willamette University Medical Department, Salem, 1906; also a graduate in dentistry; veteran of the Spanish-American War; for many years practiced in Lakeview, Ore., where he was county judge and county health officer; died June 5, aged 72, of carcinoma of the colon.

Keough, Matthew Joseph, Cohoes, N. Y.; Albany (N. Y.) Medical College, 1905; formerly commissioner of health of Cohoes, and first medical director for Cohoes public and parochial schools; served during World War I; for many years affiliated with Cohoes Hospital, where he died April 30, aged 76, of cerebral hemorrhage.

Kerrick, Charles L., Chrisman, Ill.; Kentucky School of Medicine, Louisville, 1893; Medical College of Indiana, Indianapolis, 1894; president of the State Bank of Chrisman; died in Veterans Administration Hospital, Danville, May 20, aged 83, of bronchopneumonia.

Kidd, Claude Everitt, Paducah, Ky.; University of Louisville Medical Department, 1907; at one time county health officer; served on the staffs of the Illinois Central and Riverside hospitals; died May 8, aged 80, of cerebral thrombosis.

Lange, Pauline E. ⊕ Chicago; Hering Medical College, Chicago, 1900; at one time on the faculty of her alma mater; affiliated with Illinois Masonic Hospital, where she died June 17, aged 76, of coronary insufficiency, pulmonary embolism, and carcinoma of the cervix.

Lecrone, Harris Richard ⊕ York, Pa.; Jefferson Medical College of Philadelphia, 1885; surgeon emeritus at York Hospital; died May 11, aged 91.

Leonard, Ralph Davis, Boston; Harvard Medical School, Boston, 1910; specialist certified by the American Board of Radiology; member and past vice-president of the American Roentgen Ray Society; past president of the New England Roentgen Ray Society; president of the Melrose Savings Bank; affiliated with Brooks Hospital in Brookline, Malden (Mass.) Hospital, and Melrose Hospital, where he died May 27, aged 65, of a heart attack.

Lesser, Henry Rothschild, Toledo, Ohio; Medico-Chirurgical College of Philadelphia, 1906; died in Toledo Hospital March 29, aged 68, of cerebral hemorrhage.

Lipsett, Henry, Rochester, N. Y.; University of Toronto Faculty of Medicine, 1920; died in Strong Memorial Hospital May 19, aged 57, of nephrosclerosis and arteriosclerosis.

Littlefield, Frank Browning ⊕ Providence, R. I.; Harvard Medical School, Boston, 1926; certified by the National Board of Medical Examiners; fellow of the American College of Surgeons; member of the New England Surgical Society; served during World War II; affiliated with Rhode Island and Charles V. Chapin hospitals; died in Jane Brown Memorial Hospital June 3, aged 52.

Lowe, Lewis Morgan, Seattle; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1902; died May 19, aged 76, of acute coronary occlusion.

Lutterloh, Pearlie Watson ⊕ Jonesboro, Ark.; Washington University School of Medicine, St. Louis, 1910; associate clinical professor of surgery, University of Arkansas School of Medicine, Little Rock; past president of the Arkansas Medical Society, Frisco Railway Surgeons Association, and the Mid-South Post Graduate Medical Assembly; associated with St. Bernard's Hospital; died May 15, aged 67, of coronary artery disease.

McCrumb, Ray Richmond ⊕ Lansing, Mich.; Detroit College of Medicine and Surgery, 1914; affiliated with Edward W. Sparrow and St. Lawrence hospitals; died May 5, aged 65, of cerebral thrombosis.

McGarry, Charles Patrick, Chicago; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1910; affiliated with St. Francis Hospital in Evanston, where he died June 2, aged 70, of coronary occlusion.

McGready, Joseph Hugh ⊕ Independence, Iowa; College of Physicians and Surgeons of Chicago, 1896; city health officer; died May 11, aged 80, of uremia and chronic myocarditis.

MacKenzie, Charles Fisk, Jackson, Miss.; Vanderbilt University School of Medicine, Nashville, 1904; medical missionary in China for many years; formerly medical director for the Standard Life Insurance Company; died May 3, aged 84, of cerebrovascular accident and congestive heart failure.

McPherrin, Henry Irl ⊕ Des Moines, Iowa; Drake University College of Medicine, Des Moines, 1912; served during World War I; died April 9, aged 65.

Maas, Elizabeth C., Rockford, Ill.; the Hahnemann Medical College and Hospital, Chicago, 1894; died in North Rockford Hospital May 17, aged 92, of cerebral hemorrhage.

Mallette, Lester Birch ⊕ San Francisco; Northwestern University Medical School, Chicago, 1944; specialist certified by the American Board of Radiology; served during World War II; formerly affiliated with Peoples Hospital in Akron, Ohio; died May 15, aged 32, of coronary thrombosis.

Marable, John Hartwell, Cowan, Tenn.; Vanderbilt University School of Medicine, Nashville, 1898; died in Winchester (Tenn.) Hospital April 6, aged 77.

Marchmont-Robinson, George ⊕ Chicago; Loyola University School of Medicine, Chicago, 1917; died June 14, aged 67.

Marquart, James Roger ⊕ Springfield, Ohio; Ohio State University College of Medicine, Columbus, 1926; member of the American Academy of General Practice; served during World Wars I and II; medical director of the Ohio Masonic Home; affiliated with Rickley Memorial Hospital; physician at Wittenberg College; died May 23, aged 52, of coronary occlusion.

Marshall, Hal Ellsworth ⊕ Wichita, Kan.; University of Kansas School of Medicine, Kansas City, Mo., 1925; specialist certified by the American Board of Otolaryngology; past president of the Sedgwick County Medical Society; member of the American Academy of Ophthalmology and Otolaryngology; on the staff of St. Francis Hospital, where he died June 15, aged 55, of coronary sclerosis.

Martin, Carman Ira ⊕ Akron, Ohio; Medical College of Virginia, Richmond, 1927; since 1948 county coroner; affiliated with St. Thomas Hospital and City Hospital, where he died May 16, aged 50, of acute coronary occlusion.

Miller, Charles Hyette ⊕ Dalhart, Texas; Memphis (Tenn.) Hospital Medical College, 1898; died in Northwest Texas Hospital, Amarillo, April 26, aged 78, of multiple fractures and internal injuries received in an automobile accident.

Mol, Henry L. ⊕ Grundy Center, Iowa; Northwestern University Medical School, Chicago, 1926; member of the American Academy of General Practice; died in Allen Memorial Hospital, Waterloo, June 3, aged 55, of coronary thrombosis.

Moldenhauer, Gustav H. ⊕ Chicago; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1900; an Associate Fellow of the American Medical Association; surgeon for the Illinois Central Railroad; died in Pinal Sanitarium May 17, aged 77, of carcinoma of the breast.

Mongel, Ernest Bernhardt, Philadelphia; University of Pennsylvania Department of Medicine, Philadelphia, 1899; affiliated with Wills Eye Hospital and the Episcopal Hospital; died May 12, aged 76.

Moody, Joseph Warren, Poplarville, Miss.; Medical Department of Tulane University of Louisiana, New Orleans, 1906; formerly affiliated with Sunflower Hospital (State Penitentiary) at Parchman; died in Methodist Hospital, Hattiesburg, May 2, aged 77, of cerebral hemorrhage.

Moore, Luman Gordon Jr., Kinsman, Ohio; Western Reserve University Medical Department, Cleveland, 1906; served during World War I; died May 7, aged 71.

Moore, Telford Ira ⊕ North Hollywood, Calif.; University of Illinois College of Medicine, Chicago, 1934; specialist certified by the American Board of Ophthalmology; served during World War II; shot and killed May 6, aged 45.

Moorman, Marion Ridley ⊕ Huntsville, Ala.; University of the South Medical Department, Sewanee, Tenn., 1900; died April 13, aged 73, of bronchitis and chronic myocarditis.

Myers, Frank Lewis, Sheldon, Iowa; College of Physicians and Surgeons of Chicago, 1888; died recently, aged 87, of pneumonia.

Owsley, William McGehee ⊕ Eclectic, Ala.; University of Alabama School of Medicine, 1914; died recently, aged 65, of cerebral hemorrhage.

Paterson, William Frederick ⊕ Conrad, Mont.; McGill University Faculty of Medicine, Montreal, Canada, 1900; on the staff of St. Mary's Hospital; died May 2, aged 77, of coronary thrombosis.

Patterson, Henry Hooker ⊕ Braymer, Mo.; Ensworth Medical College, St. Joseph, 1904; past president of the Caldwell-Livingston Counties Medical Society; died May 9, aged 81, of cerebral hemorrhage.

Porter, Earl Stevens ⊕ La Jolla, Calif.; Rush Medical College, Chicago, 1909; member of the Montana State Medical Association and past president of the state board of medical examiners of Montana; died in Eugene, Ore., recently, aged 69, of coronary occlusion.

Price, John Beadle, Norristown, Pa.; Medico-Chirurgical College of Philadelphia, 1914; specialist certified by the American Board of Otolaryngology; served during World War I; district medical director for the state health department; on the staffs of the Graduate Hospital of the University of Pennsylvania and Montgomery Hospital; died May 11, aged 68.

Price, Melvin Delbert ⊕ Nappanee, Ind.; Medical College of Indiana, Indianapolis, 1900; an Associate Fellow of the American Medical Association; physician for the Baltimore and Ohio Railroad; for many years part owner of the Price & Price Hospital; affiliated with Elkhart General Hospital in Elkhart, Ind., where he died May 31, aged 81, of heart failure.

Reed, Fred Romer ⊕ Three Rivers, Mich.; University of Michigan Homeopathic Medical School, Ann Arbor, 1914; fellow of the American College of Surgeons; affiliated with Three Rivers (Mich.) Hospital, Sturgis (Mich.) Memorial Hospital, and the auxiliary staff, Grace Hospital in Detroit; died June 2, aged 62, of myocardial infarction.

Reed, Guy Pace ⊕ Davis City, Iowa; St. Louis College of Physicians and Surgeons, 1899; died May 24, aged 77, of a perforated gallbladder.

Rhodes, William J. ⊕ Louisville, Ga.; University of Georgia Medical Department, Augusta, 1884; died recently, aged 94, of carcinomatosis.

Richardson, James Covington, Miami, Fla.; Vanderbilt University School of Medicine, Nashville, Tenn., 1920; died May 25, aged 57, of congestive rheumatic heart disease.

Riche, H. Guy ⊕ Baton Rouge, La.; Medical Department of Tulane University of Louisiana, New Orleans, 1899; past vice-president of the Louisiana State Medical Society; vice-president and medical director of the Guaranty Income Life Insurance Company; past chief of staff of Our Lady of the Lake Sanitarium, where he died April 24, aged 76, of adenocarcinoma of the colon.

Ridgway, Alfred M. ⊕ Annandale, Minn.; University of Minnesota College of Medicine and Surgery, Minneapolis, 1890; also a pharmacist; served as mayor of Annandale; died May 19, aged 90, of coronary insufficiency and bronchopneumonia.

Robbins, Charles Sumner ⊕ Washington, D. C.; Rush Medical College, Chicago, 1901; died June 12, aged 75.

Roberts, Peter Franklin, Raleigh, N. C.; Leonard Medical School, Raleigh, 1907; served on the faculty of his alma mater; died recently, aged 71, of cerebral hemorrhage.

Robertson, Andrew Alexander ⊕ Council Bluffs, Iowa; Medical Department of Omaha University, 1901; died recently, aged 74, of cerebral hemorrhage and arteriosclerosis.

Rodenmayer, Henry, Bloomington, Calif.; Barnes Medical College, St. Louis, 1905; died in San Bernardino May 6, aged 71, of hypertensive heart disease.

Root, Peter Nathaniel ⊕ Bakersfield, Calif.; University of Oregon Medical School, Portland, 1924; died recently, aged 64, of arteriosclerosis.

Rosen, Samuel Richard ⊕ New York; born in Brockton, Mass., Jan. 9, 1912; Albany (N. Y.) Medical College, 1937; specialist certified by the American Board of Psychiatry and Neurology; certified by the National Board of Medical Examiners; member of the American Psychiatric Association; served in Italy during World War II, and received the Bronze Star; affiliated with Goldwater Memorial and Mount Sinai hospitals; council member of the University Settlement; died in the Hospital for Joint Diseases May 24, aged 40, of coronary occlusion.

Rugh, John B. ⊕ Pitcairn, Pa.; Western Pennsylvania Medical College, Pittsburgh, 1897; died May 4, aged 81, of coronary occlusion.

Rumph, Thomas Gladstone ⊕ Fort Worth, Texas; University of Louisville (Ky.) Medical Department, 1913; died May 31, aged 65, of intracranial hemorrhage and arterial hypertension.

Ryder, Bayard Edward ⊕ Los Angeles; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1900; died May 17, aged 77, of cerebral hemorrhage.

Sandoz, Louis Albert ⊕ South Bend, Ind.; University of Louisville School of Medicine, Louisville, Ky., 1924; member of the American Academy of Dermatology and Syphilology; affiliated with Memorial and St. Joseph hospitals; died in St. Joseph Hospital, Lexington, Ky., May 13, aged 56, of adenocarcinoma of the left colon and peritonitis.

Thomas, Earl Jesse, Findlay, Ohio; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1905; fellow of the American College of Physicians; served for many years on the board of directors of the First National Bank; died in Blanchard Valley Hospital May 23, aged 69, of cerebral vascular accident.

Thompson, Floyd C. ⊕ Ontario, N. Y.; Cleveland Homeopathic Medical College, 1905; for many years health officer; formerly a member and president of the local school board; died in the Genesee Hospital, Rochester, May 7, aged 75, of myocardial infarction, diabetes mellitus, and thrombophlebitis of the right leg.

Wright, LaVerne ⊕ San Francisco; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1901; member of the American Society of Anesthesiologists; died recently, aged 73, of a cerebral accident.

GOVERNMENT SERVICES

AIR FORCE

Aviation Medical Examiners.—In July, in the new class of aviation medical examiners at the Air Force School of Aviation Medicine, Randolph Field, Texas, 93 student officers enrolled. Five officers of foreign air forces are in the class. The two from Pakistan are the first Pakistani students at the school, which has graduated flight surgeons of 33 other foreign countries. The class contains nine rated pilots, and all but three have had World War II experience overseas. The Navy has contributed 24 of these medical officers to the Air Force. Most of them were trained under the seagoing arm's V-12 program. This class is the first to include two veterans of the war in Korea.

ARMY

Personal.—Col. Dean M. Walker, chief of the department of surgery, Tripler Army Hospital, Moanalua, Hawaii, is retiring from the Army after 25 years' service and will enter private practice, limited to general surgery, in Honolulu.

Recall 500 Reserve Nurses.—The recall to active military service of 500 reserve Army nurses and 125 reserve women's medical specialists was approved July 31 by the Assistant Secretary of Defense. Included in the recall of reservists from the Women's Medical Specialist Corps are 70 dietitians, 31 physical therapists, and 24 occupational therapists. The first involuntary recall of women reservists in the medical branches of the Army since early in the Korean campaign is needed to replace women about to be released from active duty. The National Advisory Committee of the Selective Service System was directed to assure that no person essential to the national health, safety, and interest will be obliged to leave her civilian job. Those who have been recalled since June 25, 1950, will not be affected by this recall. The call-up will continue for three months beginning in February, 1953. Of those to be ordered to active duty 50% are scheduled for recall the first month and 25% in each following month. Reservists selected for recall will be given at least 120 days' notice in which to close out personal business.

PUBLIC HEALTH SERVICE

Personal.—Dr. James R. Shaw has been appointed chief, division of hospitals, Public Health Service, succeeding Dr. G. Halsey Hunt, who was named associate chief, bureau of medical services. Dr. Shaw will have charge of the 22 hospitals, 19 outpatient clinics, and over 100 outpatient offices operated for the beneficiaries of the Public Health Service. The division of hospitals is part of the bureau of medical services, of which Dr. Jack Masur is chief.

Marriages and Divorces.—The marriage rate in 1951 dipped to its lowest point since 1938, and the divorce rate to the lowest since 1941, according to provisional vital statistics released by the Public Health Service. The 1951 marriage rate—10.4 per thousand population—showed a 37% drop from the 1946 all-time peak. Since the postwar demobilization period, marriage rates have generally fallen off as the wave of marriages reduced the number of single persons of marrying age. During the past few years the number of young persons reaching marrying age has been relatively small because of low birth rates during the business depression of the early 1930's.

Marriages in 1951 totaled 1,594,900, or 72,300 fewer than in 1950. The marriage rate fell by 6%. An estimated 371,000 divorces were granted in 1951, compared with 385,100 divorces in 1950. The 1951 total includes reports from 32 states and the District of Columbia, and an estimate based on 1950 divorces in states not reporting last year. For every 1,000 persons, 2.4 divorces were granted in 1951, compared with 2.5 in 1950. Since the 1946 all-time peak, the divorce rate has dropped 44%.

Meetings of Advisory Councils.—Seven of the National Advisory Councils to the Public Health Service met during June. The councils consist of outstanding nongovernmental experts in medical sciences, public health, and public affairs. They have as ex officio members representatives of the Public Health Service, the Veterans Administration, and the Department of Defense. The councils were established by law to advise the Surgeon General on health problems in their respective fields. One of their important functions is to make recommendations for research grants to nonfederal institutions. The councils which met in June were the Mental Health Council, Arthritis and Metabolic Diseases Council, National Advisory Health Council, Cancer Council, Dental Research Council, Neurological Diseases and Blindness Council, and Heart Council.

VETERANS ADMINISTRATION

Hospital News.—Dr. Gregory Zilboorg, New York, addressed the members of the hospital staff at the VA Hospital, Northport, L. I., June 2, on "The Major Difficulties in Psychotherapy," and Dr. Ben Karpman, clinical professor of psychiatry at Howard University, Washington, D. C., June 19, on "Psychiatric Findings in Criminals."

Residencies in Psychiatry.—Residencies in psychiatry are available at the Veterans Administration Hospital, Hines, Ill., beginning Oct. 1, 1952, and Jan. 1, 1953, for a period of three years. The residency program is under the direction of the Department of Psychiatry, University of Illinois. The hospital is within commuting distance of all other psychiatric facilities in the Chicago area. The training is fully accredited by the American Board of Psychiatry and Neurology.

Personals.—Dr. Jesse F. Casey, manager of the Veterans Administration Hospital at Topeka, Kan., has been transferred to the VA Central Office in Washington, D. C., as Chief of the Hospital Psychiatry Section. Dr. Casey, who joined VA in 1930, succeeds Dr. A. H. Fachner, who has been appointed manager of the new VA Hospital in Salt Lake City, which is scheduled to be dedicated next fall.——Mr. Bert C. Moore has been appointed manager of the Veterans Administration Center in Dayton, Ohio, comprising a 1,120-bed general hospital and a 2,098-bed domiciliary unit. Mr. Moore has spent most of his 30 years' service with VA and its predecessor agencies in a managerial capacity.

Repairs for Veterans' Wheel Chairs.—More than 20,000 disabled veterans who use wheel chairs because of service-connected disabilities will be able to get emergency repairs for them without prior Veterans Administration approval. VA regional offices across the country will soon begin to issue a new "Prosthetic Service Card—Wheel Chairs" to each veteran entitling him to the new service. These veterans will get prompt service from commercial concerns or VA when they need emergency or major repairs. On presentation of the service card at any commercial repair shop, the veteran may receive emergency repairs up to $15 for each wheel chair. On-the-spot eligibility for major repairs or emergency replacement of wheel chairs at any of the 30 VA orthopedic shops located throughout the country also can be established by this card. Heretofore, veterans had to obtain prior approval from VA before emergency repairs to wheel chairs could be authorized.

FOREIGN LETTERS

CHILE

Alcoholism.—One of the main topics discussed at a clinical meeting held in Vina del Mar during January, 1952, was the subject of alcoholism. Dr. I. Roizblat pointed out the importance of the problem in Chile. The total number of persons arrested for drunkenness in Chile during 1940 was 120,000. Nine years later the number increased to 217,000. The maximal number of persons were within the ages of 25 and 34 (32%), followed by the group 35-44 years old (27%). Of those arrested, 147,688 had a primary education only, 9,177 a secondary education, and 156 were university graduates; 58,000 were illiterates. Men made up 98% of the cases, and there were twice as many unmarried as married men. After carefully analyzing the advantages and disadvantages of the conditional reflex treatment Dr. Roizblat prefers dipsotherapy. The author plans an anti-alcohol campaign.

Dr. F. Beca studied the question of psychopathology and some of the psychic disorders produced by alcoholism. He put special emphasis on encephalopathy and alcoholic dementia. The number of cases presented was 130. Sixty-four patients belonged to the encephalopathic group. The author reminded the audience that in 1947 he and Prof. Egana studied 18 patients (7 cases of delirium tremens and 8 of alcoholic encephalopathy) and noted a decrease of the blood level of thiamine, the level of which was also decreased in the spinal liquid and in the urine. The level was corrected by the administration of thiamine, but no influence on the symptoms was seen. Of the 490 cases of alcoholic psychosis that left the Manicomio Nacional, only 8% corresponded to delirium tremens. Dr. Beca thinks that this phenomenon is due to the fact that wine is drunk much more than brandy and that the majority of the consumers are men.

Some aspects of the personality of the alcoholic were studied by A. Arcaya and A. Schneider. The authors made a complete medical check-up and a neurological and psychiatric examination, giving great importance to the Rorschach test. Of 533 cases the diagnosis of ethylism could be made in 72 cases, and only 7 presented symptoms of psychosis. In the majority of cases the patients started to work at a very early age, 12-14 years old, and started to drink between the ages of 14 and 20. The authors explain that during puberty the "paternal image" is of great importance; the youth either tries to identify himself with it or else to reject it. The best friend takes the place of the paternal image, and his attitude towards alcoholism is of fundamental importance. The patients had adequate incomes, and, therefore, the authors rejected the idea that poverty or hypoalimentation was a direct cause of alcoholism; however, alcoholism was the cause of later lack of employment. The majority of the patients had neurotic manifestations since childhood. These manifestations were accentuated with the use of alcohol. The inadaptability of the patient to the environment was another reason. The family was often characterized by the presence of an oversolicitous mother filled with neurosis, by the early death of the father, or by the parents' divorce. The authors believe that the addiction to alcohol is, in the vast majority of cases, the result of repeated frustrations in early childhood that prolonged the oral phase to the advanced stages of life.

The neurological aspects of alcoholism were studied by Prof. Lea-Plaza and colleagues, who presented 215 cases of confirmed and inveterate alcoholics. The patients were divided into two groups. The first group, in which alcohol was the main determining factor of the clinical picture, were patients who suffered from neuromyelosis, myelosis, encephalomyelosis, neuritis, and polyneuritis. The total number of patients in this group was 145. In the second group, in which alcohol was only one factor, were patients suffering from epilepsy and vascular accidents. The total number of persons in this group was 168. The authors point out that it seems to them that chronic alcoholism acts in a diffuse manner on the central and peripheral nervous systems.

Professor Mardones studied the experimental problems of alcoholism. He pointed out that if we accept the theory that in problems of human alcoholism there are two aspects, one the biochemical one and the other the psychological one, we must then conclude that experimental alcoholism performed in animals in autoselection will only give us light regarding the biochemical aspect of the problem. The author observed individual variations in the conduct of animals to alcohol, and he showed that there exist conditions transmissible by heredity that influence alcoholism. Regarding the food factor, the author noted that mice that received a diet free of thiamine increased their consumption of alcohol and that the posterior administration of thiamine did not decrease their consumption.

Dr. L. Acevedo and colleagues presented the results of the medical treatment of alcoholism. They found that better results were obtained in those patients that willingly submitted to therapy and that the cultural standard of the patient influenced to a great extent the success of the treatment. Another point of interest was the fact that the greater the economic sacrifice the patient had to make to undergo therapy the better the results obtained. Of the 161 persons treated, 70% were chronic alcoholics and 30% had, besides alcoholism, a psychotic personality. The success achieved was around 50% among professional persons and only 35% among nonprofessional employees.

Neutralization Test of Poliomyelitis Virus in Santiago.—After analyzing and summarizing the history and epidemiology of poliomyelitis, Dr. Cattaneo noted that each successive poliomyelitis wave in Chile is increased in the number of cases involved. If the disease in its endemic form is new in Chile, then the majority of the population will be susceptible to the disease. For this reason the author, in 1949, investigated this problem for the first time in Chile. An analysis of the population of Santiago of different ages was made, using the Lansing test of neutralization. If the proportion of positive tests is comparable to those in the United States or European countries, then the time of introduction of poliomyelitis in Chile is similar to that in the above-mentioned countries, thereby showing that a high percentage of subclinical cases is responsible for the phenomenon. The study was facilitated to a great extent by the fact that in 1949-1950 there was a poliomyelitis epidemic. The patients were divided in three groups: (1) those under 5 years old, (2) those from 5 to 15 years old, and (3) those over 16 years old. No selection was made in the cases that were studied, thereby ensuring a cross section of the population. The results obtained were as follows: positive tests under 5 years of age, 48%; between 5 and 15, 98%; and over 16 years, 94%.

Thus it is apparent that the antibody increases with age, this being due to a subclinic phenomena. Turner in the United States reported similar figures. In 1950 there was an epidemic, from which 50 more patients under 5 years old were included. There were 58% who had positive tests, which, when compared with the 48% of the normal cases presumably positive, show no statistical significance whatsoever. The author does not accept as an explanation for this fact the possible late increase of antibodies, because all known data up to date contradicts that fact. The author explains this phenomenon by saying that the latest Chilean epidemic was not due to the Lansing strain and that the percentage of positivity to the strain was due to a contact with the strain previous to the sickness.

Fourth International Congress of Hydatid Disease.—It was recently announced that the Fourth International Congress of Hydatid Disease will be held in Santiago between Nov. 21 and 24, 1952. This congress will be held simultaneously with the Fourth National Congress of Surgery. The Chilean government through the Ministry of Public Health will sponsor the congress. The medical, surgical, immunologic, and epidemiological

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The items in these letters are contributed by regular correspondents in the various foreign countries.

aspects of the disease will be treated in the congress. All communications should be addressed to P. O. B. 9183, Santiago de Chile.

Postgraduate Courses.—A second postgraduate course was solemnly inaugurated on June 30. The main object of the course is to give training in up-to-date methods to physicians from out of Santiago preferably. The capacity of the course is approximately 40, and it will last around 1½ months. During this time the mornings will be spent in the main hospitals of the capital doing practical work in internal medicine (electrocardiography and radiology) and clinical cases, whereas the afternoon will be dedicated to problems of internal medicine. Problems of nutrition and dietetics will also be treated. It was announced that the first postgraduate course in surgery will open on July 21. The following specialities will be included: abdominal surgery, esophageal and thoracic surgery, plastic surgery, orthopedics and traumatology, and proctological surgery. Each subject will be treated with theoretical lectures, practical and clinical work, and operations. Every pupil will attend all the theoretical lectures, but in the practical field he will choose only one subject.

DENMARK

New Morbidity Census.—Just before World War II plans were prepared by Dr. J. Frandsen, head of the Danish Public Health Service, for a morbidity census in Denmark. These plans are now being realized owing to the support of the Rockefeller Foundation, certain Danish organizations, and the government. A representative committee was appointed early in 1950 to put these plans into execution, and early in 1951 they were tested in Copenhagen and some country districts to make sure that they worked well. The census itself was started in June, 1951, and it is hoped that in the course of the following three years some 100,000 persons will have been questioned in regard to their health and the social conditions under which they live. The census is carried out by social workers, nurses, and various other persons specially trained for the purpose and enjoined to observe professional secrecy with regard to the information they collect. The questions set for this census are asked by laymen and answered by laymen, with the result that the information gained is of somewhat limited scientific value and requires close medical scrutiny. To this end the cooperation of the physicians in attendance on the persons questioned is needed. The Danish Medical Association has been approached on this subject, and it is arranged that the part to be played by the medical profession is to be as light as possible. It is hoped that this census of a typical cross section of the population of Denmark will yield valuable information with regard to morbidity and social conditions.

Causes of Death in Diabetics.—Two studies by Dr. Frantz Heintzelmann, of the Medical Statistical Bureau of the Danish Public Health Service, deal with the 2,620 deaths of diabetics during the three year period 1947-1949. Before 1947, the notifications of the deaths of diabetics did not always distinguish between deaths among diabetics and deaths due to diabetes. Since January of this year an attempt has been made to render death certificates more informative on this score. Dr. Heintzelmann finds that since 1936 the annual number of deaths among diabetics has remained fairly constant at about 20 per 100,000 of the population, the sex distribution during the three years under review showing 38.5 men to 61.5 women. The observation that deaths among diabetics has been fairly constant during the past 15 years is to a certain extent confirmed by Per Hanssen, who found that in Bergen, Norway, there had been no change in the total diabetes mortality between 1929 and 1940.

Most of Heintzelmann's 2,620 deaths were due to degenerative changes in the heart. It was chiefly between the ages of 45 and 64 that this cause of death predominated, but it was also important among men between the ages of 15 and 44. Renal disease and tuberculosis were also frequent causes of death, and in childhood death was almost always due to a diabetic coma. Cancer was remarkably rare as a cause of death among diabetics. An editorial in *Ugeskrift for Laeger* for May 1 suggests that the rarity of cancer in general, and cancer of the stomach in particular, in diabetics may be more apparent than real and that the malnutrition induced by cancer renders the milder forms of diabetes so free from manifestations of this disease that it is often overlooked by a physician when writing a death certificate. The same editorial comments also on the high death rate from hypertension and arteriosclerosis among diabetics. It is intimated that if these diseases of the arterial system can be prevented by dietetic measures not only diabetics but the community as a whole will be benefited

Progress in the Treatment of Poisoning by Hypnotics.—Since World War II the progress made in the treatment of poisoning by hypnotics deserves to be called revolutionary. The milestones marking this progress begin in 1946, when Kirkegaard showed the value of combating shock. In 1947 Lous and others demonstrated the importance of keeping the respiratory passages open and providing an uninterrupted supply of oxygen. The year 1949 is notable for the centralization in Copenhagen of these patients in hospital departments specially equipped and staffed for the purpose, a reform associated with the names of Clemmesen and Bie. In 1950 Eric Nilsson did much to discredit the treatment of these patients by stimulants. Dr. Carl Clemmesen and Dr. Marie Lindhardt of the medical statistical department of the Danish Public Health Service have now succeeded in presenting statistical evidence indicative of the improvements effected by these reforms. In the six year period 1945-1950 the number of victims of poisoning by hypnotics, chiefly the barbiturates, treated in hospital has more than doubled, with 1,509 in 1945 as compared with 3,051 in 1950. At the same time the death rate among these hospitalized patients has fallen from 24.5% in 1945 to only 6% in 1950. A classification of the patients according to whether they were treated in Copenhagen, with its new system of centralization, or elsewhere in Denmark shows that the death rate was only 3.4% in Copenhagen as compared with 8.2% in the rest of Denmark. Though the figures are not yet fully worked out for 1951, it looks as if the death rate for Copenhagen will be still further reduced to about 1.5%. It is probable that the example set by Copenhagen in the treatment of these cases will be followed elsewhere in Denmark.

The many articles dealing with barbiturate and allied poisonings recently published in the Danish medical press emphasize the scope and importance of this problem. The author of one editorial on the subject writes: "Why has there been such a widespread need for hypnotics and sedatives in the Danish population during these years? Has this need always existed? And is it only now gratified as a result of social progress and increased opportunities for taking medicines? Or is the atmosphere at the present time in Denmark such that constantly growing sections of the community must have drugs which can relieve insomnia, tension and restlessness?" Whatever the answer, it is at least a consolation to know that Denmark is doing pioneer work in this field with special reference to reducing the number of fatalities.

Death of Dr. H. J. Schou.—Dr. Hans Jacob Schou, who was born on Aug. 20, 1886, died on May 2, 1952, after a long illness. He came into prominence in 1921 on the death of Adolf Sell, the founder of the Filadelfia Colony for mental cases. Sell's work had been characterized by deep religious convictions and a genius for getting other persons to help him in his creativ efforts. But he was no scientist, and his colony was sadly i need of administrative reforms. Schou was appointed as his suc cessor in the hope that Sell's religious influence would not onl be perpetuated but also supplemented by administrative talen Since 1921 Schou has converted Filadelfia into a model of i kind for the whole of Denmark. The administrative talent hop of him developed most successfully, and his scientific bent l to the issuance of several important contributions to psychiat by his assistants. In 1928 Schou founded the first sanatoriu of its kind in Denmark for nervous or borderline mental cas Dianalund Nervesanatorium. Meanwhile, at the Filadelfia C ony he founded a special department for patients with epilep guiding its evolution so that it ultimately came to be a m or less independent unit under a medical officer of its own. was the "nervesanatorium" type of institution such as Dia lund that appealed most strongly to Schou, and he was tirel in his efforts to create a similar institution in Jutland. His p health did not prevent his enormous output of literary wo both scientific and popular. Even after his retirement in 1951, because of ill health, his literary activities continued. Among his later works, published in 1945, were his studies of the physiology of the manic depressive psychoses. His popular works often reflected his religious convictions, which showed no special preference for any one particular version of the Christian creed.

SWEDEN

Prevention of Tuberculosis in Schools.—A motion, addressed in 1952 to the Swedish Parliament, draws attention to an anomalous state of affairs with regard to the prevention of infection of schoolchildren by their teachers. The inmates in "day-homes" are carefully shielded by the law in this respect. Anyone employed in such institutions, even when the employment is of a temporary character, must be able to present a certificate that his or her lungs have been radiologically examined. Such workers are also kept under continuous supervision with the possibility of tuberculosis always in mind. A much less satisfactory state of affairs exists with regard to older children attending school. School medical officers are expected to take the necessary measures for preventing infectious diseases among teachers and other school personnel, and schoolchildren are kept under constant medical supervision throughout their scholastic careers; but legislation does not provide for the systematic medical control of the school staff by radiological examinations.

Between 1944 and 1946 as many as 65 cases of tuberculosis occurred among schoolteachers. In 1943 a manual training teacher infected eight boys between the ages of 12 and 14, and in 1945 a substitute teacher in less than three months infected another teacher and 16 scholars. After giving further evidence of school infections, the authors of this motion recommend that throughout their careers teachers should be obliged to undergo periodic radiological examinations at least every other year and preferably every year, with even more frequent examinations when special conditions call for them.

Barbiturate Poisoning.—At the Southern Hospital (Södersjukhuset) in Stockholm, Dr. C. M. Ideström and Dr. G. von Reis have undertaken an analysis of the 350 cases of barbiturate poisoning admitted during the period 1946-1950. There was a remarkable rise in the number of such cases for so short a period, from 42 admitted in 1946 to 120 in 1950. There was little change during the same period in the ratio of severe to light cases, about 50% of the patients being unconscious on admission. The reasons for the attempted poisonings were different for men and women, the most common reason for women being marital conflicts and love affairs, while depression was the most common reason for men. Men were outnumbered by women in the ratio of 134 to 216. While the age distribution for men was fairly even between 20 and 60 years, most of the women were of child-bearing age, between 20 and 40 years. The death rate was 5.1% (18 deaths). As many as 14 of these 18 deaths were due to poisoning with barbiturates with a prolonged action. A useful aid to diagnosis was the spectrophotometric method for demonstrating barbiturates in the serum reported on in 1948 by Goldbaum and subsequently modified by Helldorff, Ideström, and von Reis in 1949.

Is Pharmacy to Be Nationalized?—A committee appointed to investigate the possibilities of state control of the sale of drugs in Sweden has issued a report with recommendations, which have been responded to by lively protests from several quarters. These recommendations have included far reaching standardizations of drugs, extensive inspection and control of the pharmaceutical industry, economies of administration with quite a considerable reduction of the number of employees, and an annual saving of millions of Swedish crowns. It is calculated by the committee that, had a state organization of pharmacy been in operation in 1949, an economy of some 10 million crowns could have been effected in this year. In criticism of the present system of private enterprise it is argued by the committee that it has little or no inducement to prevent the excessive consumption or misuse of drugs. It is also intimated that the cost of certain drugs is at present excessive. A scheme is proposed for the centralization of the dispensing of drugs figured in physicians' prescriptions.

In memoranda issued early in 1952, the Swedish Medical Association, the Swedish Ministry of Health, and the Central Committee of the Swedish Pharmaceutical Association have drawn attention to the many faults found in this committee report. The proposed standardization of drugs is regarded as too radical from the medical point of view to be satisfactory. Considerable progress has already been made and is still proceeding with the standardization of drugs under the present system. As for the alleged excessive consumption and misuse of drugs, it is intimated that this is largely a problem of public health and education of the public. As for the complaint that the cost of drugs is too high, it should be noted that many of the newest and most costly drugs are just those drugs that by their effectiveness are greatly reducing the duration and thereby the cost of treatment. A comparison of the cost of drugs in Sweden and various other countries is much in favor of the former. The Swedish Medical Association has some suggestions to make with regard to the revision of certain features of the present system. Among other things it suggests that pharmacists should no longer be under an obligation to sell homeopathic drugs whose distribution to the public should be free. It is also recommended that the teaching of pharmacology should be deferred to a later stage in the medical curriculum than is the case at present. Such teaching should then include information on the economy of drugs. It is also proposed that the "Swedish Pharmaconomia" should be issued free of charge to the medical profession at short intervals.

TURKEY

Chloroma of the Eye.—Dr. Nuri Fehmi Ayberk, director of Istanbul-Haidar Pasha General Hospital and chief of the department of ophthalmology, presented a case of chloroma before the Turkish Ophthalmologic Society. Thirty-five days prior to admission, the patient, a boy, aged 8, had been ill with a fever for eight days. He had a severe headache, and gradually exophthalmos developed. After a week the temperature became normal, but exophthalmos, blurred vision, and general weakness remained. The family's and patient's histories were negative. He was pale and anemic, 140 cm. in height and 28 kg. in weight; both eyes had the same degree of exophthalmos, the eye movements were limited and irreducible, and there was no diplopia. Fundus examination revealed a colorless pupil, narrow arteries, no hemorrhage, vision 8/10, normal eye pressure, and a free field of vision. The patient had lymph nodules under the chin, under both ears, and in the groins, and none under the armpits; the spleen and liver were slightly enlarged. The red blood cell count was 1,300,000, the white blood cell count 172,000, and the hemoglobin value was 20%. The differential study of the white blood cells showed 75% myeloblasts, 13 adult eosinophils, 8 transitional mononuclears, 3.75 large mononuclears, 2.25 normoblasts, 0.25 myelocytes, 0.75 myelocyte eosinophils, 0.25 macroblasts, 0.25 erythroblasts, 0.25 lymphocytes, 0.25 histocytes, 0.25 plasmocytes, and 0.50 basophil normoblasts. Wassermann, Kahn, Weinberg's, and Casoni's tests were negative, and the Takata-Ara test was 5 to 8 positive. The rate of sedimentation after half an hour was 12 mm., after 1 hour 40 mm., after 2 hours 125 mm., and after 24 hours 175 mm. X-ray examination revealed no lesions in bones or sella turcica, head, and face bones. The right lung showed hilitis. The patient was given penicillin, streptomycin, urethan, nitrogen, and blood transfusion; the temperature decreased, and there was a slight improvement. The patient died after six weeks. Necroscopy revealed that chloroma had caused metastasis in the brain, orbits of the eyes, lungs, testicles, and lymph nodules. In the United States leukemia has been treated successfully with aminopterin (4-aminopteroylglutamic acid), but this new drug has not entered Turkey yet. Only about 150 cases of chloroma have been reported in world medical literature. The first three cases of chloroma in Turkey were reported by Dr. Shükrü Hazim Tiner, Prof. Muzaffer Güçhan, and Prof. Naci Bengisu.

CORRESPONDENCE

CARCINOMA IN SITU

To the Editor:—Since the introduction of cytodiagnosis, carcinoma in situ has become recognized as a neoplasm of surprising frequency. Since the lesion is often found in cervical tissues that appear normal, one of the significant problems has been to secure biopsy confirmation of these lesions following initial detection with cytology. The absence of a target lesion for biopsy has indicated the need for a "ring biopsy" if histological study is to be accurate. Considerable mystery surrounds this so-called embryo cancer, since numerous reports have appeared in the literature indicating apparent spontaneous regression, especially during or following pregnancy.

In an effort to secure a comprehensive expression of authoritative medical opinion concerning its character, the Dade County Cancer Institute recently prepared a questionnaire on carcinoma in situ that was mailed to leading authorities in the fields of gynecology, pathology, and cytology. Replies were received from members of the American Gynecological Society, the South Atlantic Association of Obstetricians and Gynecologists, the American Association of Obstetricians, Gynecologists and Abdominal Surgeons, and from many cytologists and pathologists whose names have appeared prominently in the scientific literature dealing with carcinoma in situ. A total of 160 opinions are represented by the report.

A summation of the results of the questionnaire was presented at a recent two day conference on carcinoma in situ conducted by the Cancer Cytology Center, Dade County Cancer Institute. Participating in this program were various authorities in this field, including Dr. F. Bayard Carter of Duke University; Dr. Edgar R. Pund, Medical College of Georgia; Dr. John E. Dunn of the Field Investigation Section, Cancer Control Division, National Cancer Institute; Dr. Joseph K. Cline of the University of Alabama, Department of Cancer Research; Dr. Lyndon E. Lee, Director of Cancer Control in Puerto Rico; and Lieut. John T. Rogers, U. S. Naval Hospital, St. Albans, N. Y., in addition to the writers of this letter.

The following conclusions, based on the questionnaire on carcinoma in situ, came out of the conference.

What Is Agreed.—There is general agreement that: 1. Cancer in situ is stage 0 cervical cancer (international classification). 2. Cancer in situ is a preinvasive stage of genuine cervical carcinoma. 3. Routine cytological study is the detection method of choice, to be confirmed later by biopsy. 4. The biopsy method of choice in common use at present is either the mutiple punch or the "ring biopsy" technique. Most replies agreed that single biopsies or random biopsies were inadequate for confirmation, since there was no "target" lesion. 5. Repeated positive results of cytological tests with repeated negative results of biopsy do not rule out carcinoma. More adequate histological studies are indicated under these conditions. 6. Total hysterectomy is adequate therapy and is the treatment of choice in most clinics. A significant group, including many of those representing university teaching centers, favored treatment depending on age, emphasizing more conservative therapy (ring biopsy and electroconization or amputation of the cervix) in the reproductive age group. 7. Claims of spontaneous regression after biopsy may be fallacious. It was the general opinion that biopsy, to be adequate in this "preclinical" stage of neoplasm, may frequently extirpate the entire lesion or in other cases be followed by sloughing of the residual anaplastic tissues. Out of 141 observers, 138 stated they could not eliminate the possibility that biopsy might have removed the entire lesion. Therefore, adequate biopsy invalidates study of the behavior of carcinoma in situ. 8. There is no evidence of metastasis to lymph nodes from carcinoma in situ (when invasion has been definitely ruled out by adequate histological study).

What Is Controversial.—Controversy exists regarding: 1. Spontaneous regression. Most investigators have never observed spontaneous regression in their personal experiences, but 31 believe that it may occur. Fifteen observers reported regression in one or two cases, and six had observed it in three or more cases. 2. Tissue diagnosis of carcinoma in situ during pregnancy is equivocal; 50 would accept it, 64 would not, 34 were uncertain, and 16 preferred to confirm after delivery. 3. In reply to the question "Should in situ cancer be studied by frequently repeated (serial) cytology, surgery withheld?" 70 answered yes, in order to determine behavior and prove or disprove regression; 76 were not in favor; 8 were agreeable to such a course of action during pregnancy; 2 were uncertain; and 4 believed that such cases should be followed with further cytological studies and biopsy.

The nature of cervical carcinoma in situ is receiving critical scrutiny by the medical profession at the present time. This report provides a cross section of authoritative opinions concerning what factors are agreed on and those that remain controversial. Much further clinical research remains to be conducted before the true character of this provocative growth entity will be revealed.

In our opinion, the "surface biopsy" cell scraping provides the simplest, most accurate, and least expensive method for detection of cancer in situ or preclinical invasive cancer. In practice, all positive cytological diagnoses should be confirmed by adequate biopsy before treatment. Apparently, it will be necessary to perform periodic cytological studies for long periods on selected patients with carcinoma in situ to determine the true frequency of spontaneous regression and of progression to invasive cancer.

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TICKS FOR STUDY OF TICK PARALYSIS

To the Editor:—I would be very grateful if you would inform your readers that I am anxious to study ticks removed from tick paralysis patients. However, I must have live ticks.

Tick paralysis may not be the result of an infectious agent. The rarity of paralysis following the engorgement of the tick suggests that the ticks responsible must be exceptional and capable of introducing a very potent neurotoxin. I should gladly pay all expenses concerned and suggest that living ticks, even though damaged by removal, be sent by air mail. The ticks should be placed in an airtight, clean, 1 oz. ointment tin, in which a disc of filter paper moistened with one drop of water is placed. The tin containing the ticks should be enclosed in the ordinary mailing tube used for sending fragile materials and sent to me at the address below.

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EMERGENCY MEDICAL CALL SERVICES

To the Editor:—During the meeting of the American Medical Association in June, 1951, the House of Delegates adopted a resolution which requested all national specialty boards to facilitate general participation of specialists in emergency medical call services. These boards were asked to assure diplomates and potential diplomates that participation in such a community activity would not jeopardize specialty ratings. This resolution was adopted in response to a report of the Council on Medical Education and Hospitals of the American Medical Association and was approved by the Advisory Board for Medical Specialties on Feb. 10, 1952. The American Board of Proctology approves of this plan and wishes all of its diplomates and potential diplomates to know that their "specialty ratings" will not be jeopardized by participation in this worthy community activity.

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SKIN SCRAPINGS IN LEPROSY

To the Editor:—The clinical note in The Journal, Jan. 5, page 44, by Dr. R. K. Maddock, entitled: "Skin Scrapings in Leprosy; Positive Results by the Wayson Technique in Eighty-Four Supposedly Arrested Cases," is, I fear, likely to cause some confusion in the literature. Leprosy workers who see the title will wish to learn of this supposedly improved technique of obtaining material for bacteriological smears. Actually it is the long-established and widely-used "scraped-incision" method, with nothing new except the insistence that the cut be made through the full thickness of the skin; and the advantage of that, if any, is offset by the facts that, apparently, the incision is scraped only once and only one lesion or lesion-site is examined.

Dr. Maddock referred to the technique he described as the "snip" method. That method, however, long used in certain quarters, involves removal of a shallow piece of the skin by curved-on-flat scissors. The raw surface of the bit removed is then rubbed on the slide or scraped with a scalpel to obtain pulp. This technique has long since been abandoned.

The scraped-incision method was introduced by me in the official leprosy examining committee in Manila when I became chairman in 1917-1918, to replace the previously used puncture method by which the main element obtained was blood, exuded or expressed. In examining smears of that material much time was wasted, and, undoubtedly, not a few cases were erroneously reported as negative. The new method has been described repeatedly, first in an article entitled "A Plea for the Early Recognition of Leprosy, with Notes on Diagnosis and Methods," by H. W. Wade and C. B. Lara (*J. Philippine Islands M. A.* 4:132-140, 1924) and most recently by me—because of practices observed during a world tour of leprosy institutions—in a detailed and illustrated article, "The Bacteriological Examination in Leprosy" (*Leprosy Review* 6:54-60, 1935). The latter article was quoted by R. G. Cochrane in his "Practical Textbook of Leprosy" (New York, Oxford University Press, 1947, pp. 68-69), photographs being used to illustrate certain points, including the depth of the incision.

The more important features of the technique, as stated in the first description are (*a*) making an actual cut, "deep into the skin," with a sharp-pointed, keen-edged instrument, a small (short-bladed) scalpel being preferred, although a razor blade or other instrument might be used, and (*b*) getting tissue juice and pulp by scraping the wall of the incision, measures being taken by compression and otherwise to avoid or minimize dilution with blood. This method cannot properly be dismissed as one obtaining material "only from the superficial layers" of the skin, although that would apply to the real snip method. Emphasis was given to the (usual) necessity of making multiple smears. In practice four to six or more are usually made from different sites, including the nasal mucosa on both sides.

That the cut should be "through the full thickness of the skin" is, in my opinion, neither necessary nor desirable. Nor is a single scrape of the blade always sufficient, because, in many instances, in both the lesions of patients with the macular cases coming for diagnosis and in thoroughly resolved lesion-areas of candidates for parole, the tissue is relatively firm and tough, and repeated scraping is required to obtain any material amount of pulp.

Few leprosy institutions could afford to use a new slide for every examination, and there is no need of it whatever if elementary care is exercised in cleansing used slides. Dr. Maddock does not say whether the razor blade, once used, is discarded, but presumably it is. At Carville, where I have watched that implement being used and have tried it myself, a new one is used for each smear. In few places could that be done, because of the expense, especially if multiple smears were made at each examination as they should be. Personally, I do not like that instrument, for it is not so easily controlled as is a suitable short scalpel, and the straight edge is not so well suited as a bellied one for obtaining pulp from the cut surface. It could not, of course, be used for the nasal mucosa.

The experience related by Dr. Maddock is paralleled in a way by that of the late Dr. E. V. Pineda, who for several years was my first assistant in the laboratory here when I was in the government service, and who in 1927 or 1928 left to take charge of the leprosy department of the San Lazaro Hospital in Manila. Two or three years later he found himself in difficulties with his patients and otherwise, it being held that he was not so successful in treatment as his predecessor had been. The latter, who supposedly had been using our standard method in making bacteriological examinations but actually had not, was discharging about 100 patients a year as having negative tests. Dr. Pineda, using the method of examination I had introduced at Culion—properly taken smears from multiple sites—was able to discharge under the regulations only about 25 a year.

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PRECAUTIONS IN AUTOPSY AND EMBALMING PROCEDURES FOLLOWING THE ADMINISTRATION OF RADIOISOTOPES

To the Editor:—As administration of radioisotope therapy is becoming widespread, problems arise concerning the handling of postmortem tissues, which, because of antemortem high dosages of certain radioisotopes, may emit radioactivity for some time after death. This notice is designed to disseminate recommendations regarding precautions to be taken in the handling of radioactive tissues during postmortem examination or in embalming procedures.

An up-to-date list of all patients who have received radioisotopes should be maintained in the record office of hospitals. This information should be supplied by the radioisotope laboratory. Names of all deceased persons should be checked against this list by the record office personnel. If the deceased has ever received isotopes, the person in charge of the radioisotope laboratory should be promptly notified.

If the deceased has received a therapeutic isotope dose within two months, the radioactivity survey officer should monitor the body before autopsy or release to a mortician. If an autopsy is to be performed, the record office should inform the pathologist who is to perform the autopsy that radioisotopes have been given. The pathologist should secure from the radioactivity survey officer a report of the amount of radioactivity, and copies should be attached to the autopsy protocol and the clinical chart.

The precautions to be taken will depend on the level of radioactivity found by the radioactivity survey officer. In cases in which the level is less than one-half milliroentgen per hour (mr/hr.), no special precautions are necessary at any time. In cases in which the level is from ½ to 6 mr/hr., the only precaution necessary in handling the body would be the wearing of rubber gloves, which should be washed with soap and water before they are removed from the hands. When the level is over 6 mr/hr. further precautions are necessary in order that the maximum permissible exposure of 0.3 r per week shall not be exceeded. The additional precautions in such cases should be (1) the wearing of a lead apron and a dosimeter; (2) thorough cleansing with soap and water or detergent of tables and other surfaces on which blood or other body fluids have been spilled; and (3) the avoidance of eating and smoking while wearing the rubber gloves.

In all cases in which the level of radioactivity is over ½ mr, every effort should be made to confine body fluids that are removed during the course of the postmortem procedures to special vessels, to pour them directly into the drain, and to flush copiously with water. In cases in which it is desired that organs or body fluids be retained for further study, they should be kept in special containers and suitably labeled. As the radioactivity decreases rather rapidly, these materials could be retained until their level of radioactivity has fallen below the danger level as determined by radioactivity survey; otherwise, the precautions as noted herein should be carried out.

The mortician should be given instructions similar to those for the pathologist, the nature of which will depend on the degree of radioactivity remaining at the time of delivery of the body to the mortician. The precautions herein outlined were approved by the Division of Biology and Medicine of the United States Atomic Energy Commission.

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BUREAU OF LEGAL MEDICINE AND LEGISLATION

MEDICOLEGAL ABSTRACTS

Workmen's Compensation Acts: Permanence of Cinnamon Sensitivity.—The claimant sought a compensation award for permanent disability resulting from his sensitivity to cinnamon. From a denial of such award the claimant appealed to the district court of appeals, first district, division 1, California.

The claimant had worked as a baker since 1922, during which time he handled cinnamon without harmful results. In November, 1948, he was employed by Masonic Homes as a pastry baker. The following February a rash developed on his hands; it became so acute that he quit work on March 10, 1949. The commission determined that he had contracted a disabling dermatitis, which was caused by sensitivity to cinnamon and gave him temporary total compensation. The condition disappeared in April, and he went to work as a baker for another employer. After three days the condition reappeared, and a few days later he was forced to quit. After a short period of rest the condition disappeared. Twice later he went to work for other employers, and after extremely short periods of employment he again broke out with dermatitis due to his sensitivity to and contact with cinnamon. Each time he received total compensation, and each time his dermatitis cleared on cessation of contact with cinnamon and reappeared on subsequent exposure. He then filed his petition for a permanent disability rating. At the hearing before the commissioner the claimant submitted the reports of five physicians, one of whom was his personal physician. All agreed substantially that at the present time he is sensitive to cinnamon and that exposure to it produces dermatitis. None of the physicians other than his own reported on the permanency of this sensitivity. The only medical evidence in the record is the following report of his personal physician: "Because this skin test is still positive to cinnamon, the patient would still be unable to work as a baker without having a recurrence of his skin eruption as long as he contacts cinnamon. This skin sensitivity to cinnamon will probably remain for an indefinite period of time, but it is impossible to know how long such a sensitivity will remain." On the basis of this evidence the claimant contended on appeal that the commissioner erred in finding that the claimant suffered no permanent disability.

After pointing out that whether the injury was permanent at a particular time was purely a question of fact and that the burden of proof was on the claimant to show that his sensitivity to cinnamon was permanent, the court of appeals said that it was a fact that on four different occasions since 1949, when exposed to cinnamon, dermatitis developed, and all the physicians agreed that the claimant is now sensitive to cinnamon, and exposure to it at present will result in dermatitis. These facts alone, continued the court, do not establish that such condition will continue for the balance of his life or meet the test of permanent disability. A disability is generally regarded as permanent when further change, for better or worse, is not to be reasonably anticipated under usual medical standards. It may be that no further treatment is possible or that the only treatment suggested is so problematical of success that the employee's refusal to undergo it is warranted. In such an event, it is permanent within the meaning of the act. In practical legal results, the healing period is over, and a permanent aftermath of disability exists. Ordinarily, the term permanent, when applied to a personal injury, means "lasting during the future life of the injured party." The evidence on which the claimant relies, said the court, is the statement of his personal physician that the "sensitivity to cinnamon will probably remain for an indefinite period of time but it is impossible to know how long such a sensitivity will remain." This is not a prognosis that it will remain all his life or will be permanent. The most that can be said of the statement is that it is susceptible of two reasonable inferences, one that it might continue for his lifetime, the other that it might not so continue. The commission adopted the second inference. The physician's statement in no wise compels the adoption of the first inference. Certainly the statement of the physician that it is impossible to know how long the sensitivity will continue is not sufficient to compel the conclusion that it is a permanent condition. Accordingly, the court of appeals affirmed the denial of an award by the compensation commissioner. *Sweeney v. Industrial Accident Commission, 236 P. (2d) 651 (California 1951).*

Medical Practice Acts: Revocation of License for Income Tax Evasion.—The defendant Raab had engaged in the duly licensed practice of medicine in Ohio for several years when he was convicted of income tax evasion under the federal statutes and served a sentence of 18 months in a federal penitentiary. Subsequent to the serving of that sentence, the state medical board of Ohio revoked Raab's license to practice medicine on the ground that he had been guilty of a felony, that being a ground for such revocation under the medical practice act. On appeal to the court of common pleas, the action of the state medical board was reversed, so the board appealed to the Supreme Court of Ohio.

The controlling question, said the Supreme Court, is whether conviction of violation of a federal statute that carries the penalty of imprisonment in a federal penitentiary constitutes the practitioner "guilty of felony" within the meaning of the medical practice act and is thus a valid ground for revocation of his license to practice medicine. The term "felony" is not defined in the medical practice act but is defined in the Ohio general code, which provides: "Offenses which may be punished by death, or by imprisonment in the penitentiary, are felonies. . . ." It was argued on behalf of Raab that "felony" as used in the general code included only a violation of the criminal statutes of Ohio and that the criminal charge must be based on the Ohio statutes. In support of this position it was claimed that the legislature evidenced intent to confine the term "felony" to violations of Ohio statutes by failing to specifically state in either the medical practice act or the general code that violation of any statute, other than a statute of Ohio, could constitute a felony. On the other hand, the state medical board contended that the term "felony" as used in the general code included a violation of any federal statute that is punishable by imprisonment in a federal penitentiary.

In the case now under consideration, said the court, the crime consisted of a violation of a law of the United States. Citizens and residents of Ohio, as they go about their daily work or pursue their professions, are subject to the laws of the United States as well as the laws of Ohio. They are protected by both and must pay the penalty for violation of either. The operation of the federal courts in Ohio is necessary for the good of Ohio citizens. The enforcement of federal statutes in Ohio is as necessary as the enforcement of Ohio statutes. No one would deny that every Ohio citizen is amenable to the federal income tax laws. We, therefore, consider it reasonable to conclude, said the Supreme Court, that the General Assembly of Ohio had in mind this dual obligation of Ohio citizens when it enacted its general code. We are of the opinion that the term "felony," as used in that section, includes a violation of a federal as well as an Ohio criminal statute, the penalty for which violation is imprisonment in either a federal or the state penitentiary. Conviction of a violation of a federal statute punishable by imprisonment in a federal penitentiary satisfies the requirement of the general code. Accordingly, the judgment of the trial court was reversed and the order of the state medical board revoking Raab's license to practice medicine was affirmed. *In re Raab, State Medical Board of Ohio v. Raab, 101 N. E. (2d) 294 (Ohio 1951).*

MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A.M.A. Arch. Dermat. and Syphilol., Chicago

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Diethylstilbestrol Therapy in Young Men with Acne: Correlation with Urinary 17-Ketosteroids. C. B. White and C. F. Lehmann.—p. 601.

Skin Resistance in Absence of Sweat Glands.—A 43-year-old woman and her 10-year-old daughter who had the rare anhidrotic type of hereditary ectodermal dysplasia were studied with regard to their sweating. Electrical skin resistance studies revealed areas of high resistance over the entire body except the axillas, palms, and soles. On the face and upper chest the resistance was high but relatively lower than over the remainder of the body surface. Chemical tests for sweat and biopsies confirmed the absence of sweating in the areas demarcated by the electrical skin resistance studies. Galvanic skin responses were absent from nonsweating palmar areas, and were present, but very weak, in sweating palmar areas. These findings support the concept that electrical skin resistance is inversely related to sweat gland activity, rather than to vasomotor activity or other changes accompanying sympathetic nervous system activity. The electrical resistance of the skin in the areas where sweat glands are congenitally absent is as high as that observed in denervated or sympathectomized areas of the body in normal persons. In areas where sweat glands are present, the electrical resistance is dramatically lower, and is inversely proportional to environmental temperature and consequent sweat gland activity.

Pre-Lupus-Erythematosus (L. E.) Cell.—In six of seven patients with acute disseminated lupus erythematosus, the L. E. phenomenon, i. e., the presence of L. E. cells and/or rosette formation, was demonstrated on admission examination. In the seventh patient this finding was not revealed on first examination despite a characteristic clinical picture of acute disseminated lupus erythematosus, which responded dramatically to treatment with cortisone. A pronounced clinical relapse after discontinuation of this drug brought the patient back to the hospital six weeks later, and this time typical L. E. cells and rosette formation were observed. In addition the following changes in the polymorphonuclear cells were seen, which were interpreted as stages in the development of the L. E. cell: (1) pyknosis of one or more nuclear lobes, (2) pyknosis of all nuclear lobes and rounding of one or more lobes with preservation of intact interlobar filaments, (3) disappearance of interlobar filaments leaving cells containing one to five round homogenous masses of nuclear material but with normal cytoplasm and granules (the pre-L. E. cell), (4) partial extrusion of round nuclear masses from the cells, (5) presence of free nuclear material outside cells, and (6) phagocytosis of nuclear material by other polymorphonuclears to form L. E. cells. The pre-L. E. cell is the distinctive stage in this process in which the destroyed fragments of nuclear origin are still within their own cell boundaries. Recognition of the pre-L. E. cell may help in the early diagnosis of disseminated lupus erythematosus.

A.M.A. Arch. Neurology and Psychiatry, Chicago

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Id.: II. Histologic and Embryologic Consideration. R. J. Brzustowicz and J. W. Kernohan.—p. 592.
Id.: III. Their Relationship to Development of Gliomas. R. J. Brzustowicz and J. W. Kernohan.—p. 602.
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Incidence of Cavum Septi Pellucidi and Cavum Vergae in 1,032 Human Brains. J. T. Schwidde.—p. 625.
Occlusive Cerebrovascular Disease Simulating Brain Tumor: Angiographic Differentiation. G. L. Smiley.—p. 633.
*Subarachnoid Hemorrhage, Intracerebral Hemorrhage, and Intracranial Aneurysms. A. Dekaban and D. McEachern.—p. 641.
Rate of Advance of Hoffmann-Tinel Sign in Regenerating Nerves: Further Observations. S. Sunderland and K. C. Bradley.—p. 650.
Optic Atrophy Caused by Arteriovenous Angioma: Report of Case. R. I. Band.—p. 655.
Clinical and Electroencephalographic Effects of Prefrontal Lobotomy and Topectomy in Chronic Psychoses. E. G. Krueger and H. L. Wayne. —p. 661.
Reactions of Dogs Without Neocortex During Cycles of Addiction to Morphine and Methadone. A. Wikler.—p. 672.

Intracranial Hemorrhage and Aneurysms.—Dekaban and McEachern selected 143 cases from the records of the Montreal Neurological Institute for analysis of the relation of spontaneous subarachnoid hemorrhage, intracerebral hemorrhage, and intracranial aneurysm. These cases were observed from 1938 to 1948. The criteria for selection were as follows: 1. The condition had to be proved by lumbar puncture, arteriogram, operation or autopsy. 2. The hemorrhage must have occurred within four days prior to admission. 3. The hemorrhage must not have been due to trauma. There were 30 cases of ruptured aneurysm, 8 cases of nonruptured intracranial aneurysm, 13 cases of abnormal arteriovenous communication, 1 case of subarachnoid hemorrhage with proved rupture of anatomically normal artery, 56 cases of subarachnoid hemorrhage of undetermined cause, and 35 cases of primary intracerebral hemorrhage. The authors stress that blood in the subarachnoid space, in addition to raising the intracranial pressure, acts as an irritant, causing general systemic and local meningeal reactions. The latter, in some cases, are severe, with as many as 4,000 white blood cells per cubic millimeter of cerebrospinal fluid. Aseptic meningitis, or even constitutional fever, may be suspected wrongly in the mild cases unless lumbar puncture is carried out. The meninges show cellular infiltration and phagocytosis in the acute stage, followed later by patchy fibrosis. This distribution may explain the infrequent occurrence of hydrocephalus in surviving patients. Ruptured aneurysm was found to be responsible for the subarachnoid hemorrhage in nearly all cases in which autopsy was performed. Twenty-six of 30 ruptured aneurysms were of the congenital, berry type; the remaining 4 were fusiform and of arteriosclerotic origin. Because of the frequent occurrence of multiple and bi-

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Titles marked with an asterisk (*) are abstracted.

lateral aneurysms, the necessity of bilateral angiography is stressed. The majority of patients with spontaneous subarachnoid hemorrhage recover without surgical treatment; in some patients the risk of repeated bleeding may be abolished by intracranial clipping and excision of an aneurysm. The possibility of blood dyscrasia and an abnormal bleeding tendency should be kept in mind in all cases of subarachnoid hemorrhage, and appropriate treatment should be carried out with rutin, ascorbic acid, and vitamin K.

A.M.A. Arch. Otolaryngology, Chicago

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American J. Digestive Diseases, Fort Wayne, Ind.

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Am. J. Syphilis, Gonorrhea and Ven. Dis., St. Louis

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•Intramuscular Chloramphenicol in Treatment of Gonorrhea and Granuloma Inguinale. R. C. V. Robinson and T. L. Wells.—p. 264.
•Chloromycetin (Chloramphenicol) and Penicillin in Gonorrheal Urethritis: Two Effective Single Dose Plans. P. G. Butler, A. F. Brewer, P. K. Condit and J. Johnston.—p. 269.
Egg Lecithin in Cardiolipin Antigens. M. R. Flynn, V. Tompkins and T. Beecher.—p. 272.
Further Observations on Biologic False Positive Serologic Tests in Young Children: Possibility of Erroneous Diagnosis of Syphilis. H. C. S. Aron, G. E. Parkhurst, J. Rodriquez and W. W. Harris.—p. 278.
The Ito Skin Test for Chancroid in Adult and Juvenile African. R. R. Willcox.—p. 284.

Injection Schedules for Early Syphilis Treatment.—Of 146 patients with darkfield-positive secondary syphilis 60 were given 2,400,000 units of procaine penicillin G in peanut oil with 2% aluminum monostearate in a single administration of 600,000 units at each of four sites, while 86 patients were given 2,400,000 units of the same penicillin preparation in two administrations of 1,200,000 units, 96 hours apart. After 12 to 15 months there were 88.2% successes, 3.6% reinfections, and 8.2% failures in the group receiving one injection, and 89.5% successes, 2.5% reinfections, and 8% failures in the group receiving two injections. Surface spirochetes and syphilitic lesions showed a response similar to that obtained with aqueous penicillin or trivalent arsenicals. No serious reactions were noted. Comparisons of penicillin blood levels during the one and two injection schedules revealed a much higher average peak concentration as well as a slightly greater area under the curve for the one injection schedule. No penicillin activity was observed in samples of cerebrospinal fluid drawn from patients during a 24 hour period following a single injection. Eleven patients from the one injection group and 11 patients in the two injection group became pregnant after treatment and were not retreated. Eight babies were born alive in each group. All of the 16 infants who were born alive were clinically, serologically, and roentgenologically negative for syphilis 1 to 14 months after birth. Three fetal deaths occurred in each of the two groups. The clinical evidence suggests that the one and two injection schedules were equally effective in treatment of early syphilis. On one of two other schedules aqueous crystalline penicillin G was injected intramuscularly every three hours in doses totalling 2,400,000 units over seven and a half days, and on the other 600,000 units of penicillin in peanut oil and beeswax were injected daily for eight days in total doses of 4,800,000 units. Comparison of all four methods revealed that the one injection schedule is as effective as the others. Its main advantage is that treatment, exclusive of follow-up, can be accomplished in one visit to the clinic.

Chloramphenicol in Treatment of Venereal Diseases.—Chloamphenicol (chloromycetin®) was given intramuscularly in a single dose of 1 gm. to 51 patients with gonorrheal urethritis. Of the 51 patients, 36 returned for at least one post-treatment examination, and 31 of these were cured. If the two patients who admitted exposure after treatment are considered reinfections rather than failures, the cure rate is 92%. The authors feel that chloramphenicol is probably as effective as penicillin. Seven Negro patients with granuloma inguinale were treated with chloramphenicol intramuscularly in doses of 1 to 2 gm. daily for 9 to 15 days, with total doses of 10 to 18 gm. In all seven patients the lesions healed rapidly. In three patients there was noted an extraordinarily rapid return of pigment in the lesions. Final evaluation of treatment of granuloma inguinale with chloramphenicol must await a larger series of patients and longer follow-up periods.

Chloramphenicol and Penicillin in Urethritis.—Of 313 male patients with acute gonorrheal urethritis, 192 were given a single intramuscular injection of 900,000 units of procaine penicillin G in oil with 2% aluminum monostearate, and 121 patients received a single oral dose of 3 gm. of chloramphenicol (chloromycetin®). Penicillin proved to be curative in 165 patients who could be followed up to the 23d post-treatment day, and chloramphenicol proved curative in 103 patients who were followed for the same post-treatment period. There were no untoward reactions to the penicillin treatment. The single dose of 3 gm. of chloramphenicol was well tolerated; mild side-effects were bitter taste persisting for two to six hours, noted by 50% of the patients, and a mild transitory diarrhea, noted by 5%. Both penicillin and chloramphenicol in single doses in the amounts used by the authors are effective enough in treatment of acute male gonorrheal urethritis so that measures to insure the return of patients who lapse during the post-treatment follow-up period can be eliminated with no appreciable loss of efficiency in the gonorrhea control program. Epidemiological control efforts should stress the bringing of sexual contacts of newly diagnosed cases to medical supervision to eliminate potential new foci of infection as rapidly as possible. The single dosages used may be sufficient to abort syphilitic infection acquired at the same time as the gonorrhea. However, until such time as further data are available on this point, routine serologic tests for syphilis should be done on all patients treated for gonorrhea, both at the time of diagnosis and three to six months after treatment.

Am. Practitioner & Digest of Treatment, Philadelphia

3:349-432 (May) 1952

Progressive Bronchospastic Disease: Primary Obstructive Emphysema. J. Bradford.—p. 349.
Weil's Disease: Report of Four Cases Emphasizing Two Adjuncts to Early Diagnosis. F. E. Cowden, F. D. Ownby and R. L. Isham. —p. 353.
Practical Bacteriologic Aspects of Urinary Tract Infection. E. Jawetz. —p. 361.
Sigmoidorectal Intussusception with Incarceration. E. Granet and S. S. Pearl.—p. 365.
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What to Tell Patient with Early Senile Cataract. R. I. Pritikin and M. L. Duchon.—p. 384.
Some Psychosomatic Aspects of Essential Dysmenorrhea. M. Ross. —p. 385.
Tumors of Small Bowel. J. Shaiken.—p. 388.
Tumors of Breast in Adult Female. R. S. Pollack.—p. 393.
Prothrombin: Physiology and Deficient States. A. J. Quick.—p. 397.

Anal Irritation Following Use of Antibiotics.—An epidemic of influenza presented an opportunity to observe certain proctologic sequelae in 21 patients who had been treated with aureomycin or similar antibiotics. These effects became manifest from four weeks to eight weeks after administration of the drug, so that the relationship frequently was unrecognized. A variety of ointments and other therapeutic measures had been used with little or no relief. The major symptom was itching and burning about the anus, which at first was intermittent but later became continuous. Defecation aggravated the distress. The perianal skin was a dusky red, with scattered rosy follicles and many erosions. Scales, crusts and excoriations from scratching were present in some patients. The process often involved the anal canal. The crypts were sometimes reddened, and in several patients the papillae were cherry-red. Some patients had a low-grade proctitis. The cause of this anorectal disorder is not definitely known, but there has been speculation on possible changes in the intestinal flora due to selective suppression of the normal bacteria by the antibiotic, with consequent changes in the pH of the feces or chemical constituents of cholinogenic or steroid origin. Interference with vitamin B complex absorption has been suggested as a possible cause. The following treatment was found helpful: The perianal skin was shaved, then sprayed with a 2% solution of tetracaine (pontocaine®). Tincture of benzoin was applied and allowed to dry. Zinc oxide (Lassar's) paste was then spread thinly over the area. This procedure was repeated twice weekly until the lesions cleared. While local anesthetic ointments may give temporary relief, they often tend to aggravate the irritation or cause violent local reactions.

Annals Otol., Rhin. and Laryngology, St. Louis

61:1-314 (March) 1952. Partial Index

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Nasopharyngeal Radium Treatment: Follow-Up Study of 263 Patients. W. E. Loch and N. D. Fisher.—p. 198.
Fractures of Facial Bones—Standardization of Diagnosis and Treatment. C. S. McLemore.—p. 242.
•Analysis of 240 Cases of Cancer of Larynx, with Respect to Terminal Phases and Death. P. D. Latella.—p. 266.

Acoustic Trauma in Children.—The histories of three children referred by a school nurse to a hearing test center during the past year indicate that more attention should be given to the effect of loud noises on the hearing acuity of children. In all three children, permanent hearing defects were produced by inadvertent exposures to cap pistols or fire crackers. The deafness was of the nerve type, and was similar to that encountered among military personnel exposed to gunfire. The detection of these cases of hearing loss was made possible by carefully testing each subject in a quiet room with frequencies of 500, 2,000, 4,000 and 8,000 vibrations per second, at 15 decibels and utilizing principles of audiometry. Physical and otological examination of all three patients revealed that the membrana tympani and middle ears were normal, and there was no evidence of adenoid hypertrophy in the nasopharynx. While these cases are too few to draw any definite conclusions, it seems obvious that the possibility of acoustic trauma in children deserves more attention. The hearing defects resulting from acoustic trauma in these patients are probably not progressive.

Terminal Phases of Cancer of Larynx.—In order to determine the cause of death and terminal condition of patients with cancer of the larynx, Latella reviewed the records of 240 patients, of whom 117 had died and the remainder were in the terminal phases. He found that the cause of death and terminal phases of these patients are the same as of the patients who have cancer elsewhere in the body. Because the growth primarily involves the upper airway, one might conclude that laryngeal occlusion would be a usual cause of death, but this does not occur when tracheotomy is available. Hemorrhage as the cause of death occurred in only 16% of the patients, which is not as high as might be expected with a cancerous growth that can produce so much destruction and ulceration of such vascular areas as the larynx, the root of the tongue, and the neck. The laryngectomized patients were spared the foul-smelling, painful, ulcerating necrotic larynx so often seen in patients who were treated ineffectively with radiation. However, once neck metastases become progressive in a laryngectomized patient, the subsequent course is little different than in the nonoperated case. If the secondary neck metastasis has received ineffective radiation, the terminal phase is severer than if irradiation is not given. Bronchopneumonia is secondary to blocking of the tracheobronchial tree by accumulated secretions, vomitus, or blood. These patients have no terminal rise in temperature as with primary infections; they apparently drown in their own secretions. Only two patients out of 116 died of cardiac failure. Metastases to distant organs and involvement of heart, spleen, liver, and kidneys were infrequent. Malignant cachexia appeared as nutrition failed and pain, sleeplessness, and anxiety appeared, but it was not produced by toxins or poisons emitted by the growing tumor. Conscientious scientific nursing is of great importance. Knowledge of the final stages of laryngeal cancer will enable the physician to carry out a plan of treatment that will make the last days of these patients more bearable.

Archives of Physical Medicine, Chicago

33:267-308 (May) 1952

Simplified Electrotherapy: New Stimulator for Home Treatment of Denervated Muscle. S. Mead.—p. 267.
Effects of Cerebral Diathermy on Normal Adults. E. K. Stoner and H. Shapiro.—p. 272.
Precision of Elbow Flexion Ergography Under Varying Degrees of Muscular Fatigue. H. H. Clarke.—p. 279.
•Use of Cortisone in Management of Shoulder-Hand Syndrome: Case Report. N. Sussman.—p. 289.
Ethyl Chloride Spray for Painful Muscle Spasm. J. Travell.—p. 291.

Cortisone in Shoulder Hand Syndrome.—The 71-year-old woman whose case is reported had a healed posterior myocardial infarct and diabetes mellitus. She complained of fatigability, exhaustion on walking, insomnia, and nocturia, recurring episodes of substernal distress, and tightness in the chest. An attack of left-sided hemiplegia largely subsided, but some weeks later she experienced an ache in her left shoulder, with limitation of motion because of increased pain. This became rapidly worse, and in about 7 to 10 days the hand and wrist were swollen, and the skin had a dusky cyanotic appearance. Movement of fingers, hand, and wrist became very painful. There was no evidence of rheumatoid arthritis. Treatment consisted of such physiotherapeutic procedures as paraffin baths, infrared ray therapy, and passive exercise. When these proved ineffective, she was given several nerve blocks plus local infiltration of trigger points, but relief was transient. Then she was given 100 mg. of cortisone daily by mouth. Within 48 hours improvement began. The pain subsided sufficiently so that administration of all narcotics was discontinued, and comfort was achieved with five grains (0.3 gm.) of acetylsalicylic acid given every three to four hours. After one week the swelling of her fingers, hand, and wrist subsided. The range of motion of the shoulder was increased without pain. The appetite was increased, and the mental depression and fatigue disappeared. When her blood sugar became elevated and glycosuria appeared, increased doses of insulin reestablished control. After 10 days of cortisone therapy, using 100 mg. daily, the dose was gradually reduced and then discontinued. The author recommends cortisone in the management of the shoulder hand syndrome.

Arkansas Medical Society Journal, Fort Smith

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Modern Diagnostic Routine for Cancer of Lung.—J. K. Donaldson. —p. 265.
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Cation Exchange Resin in Treatment of Congestive Heart Failure: I. Electrolyte Exchanges During Initial Periods of Resin Therapy. J. R. Elkinton, R. D. Squires and W. C. Klingensmith Jr.—p. 747.
Tissue Cations and Water in Arterial Hypertension. L. Tobian Jr. and J. T. Binion.—p. 754.
Heart Failure and Pulmonary Edema Produced by Certain Neurologic Stimuli. R. Paine, J. R. Smith, H. R. Butcher and F. A. Howard. —p. 759.
Death in *Potassium Deficiency: Report* of Case Including Morphologic Findings. J. D. Keye Jr.—p. 766.

Intra-Arterial Injection of Histamine in Peripheral Arterial Disease.—This paper reports the results obtained with intra-arterial injections of histamine in a small group of men with intermittent claudication, whose average age was 64 years. They had been under observation for several months, during which time Buerger's exercises, oral doses of priscoline® (2-benzyl-2-imidazoline hydrochloride), reduction in smoking, moderate doses of alcohol, foot hygiene and reflex heat had been used with little effect. The patients were taken to a constant temperature room and their skin temperature response in the lower extremities to immersion of the arms in hot water was measured. If the temperature in the affected leg did not rise significantly, a posterior tibial nerve block was performed and the temperature was again measured. Several patients were given a paravertebral block or spinal anesthetic as a further check. The histamine injections were given by means of the ordinary intravenous apparatus, plus a hand bulb to raise the pressure in the bottle, and 3 mg. of histamine diphosphate in 500 cc. of isotonic solution of sodium chloride were introduced into the femoral artery in about one-half hour. The usual schedule was two injections per week for the first three weeks, then one injection per week until the tolerance for walking appeared to be stabilized. Injections were then discontinued, and the patient's walking tolerance was checked at monthly intervals. If regression was noted, weekly injections were reinstituted. The walking tolerance was tested by means of a motor-driven continuous belt-type of treadmill. Five of seven patients treated by the intra-arterial injection of histamine diphosphate obtained favorable results, one fair, and one poor results. Improvement was usually evident by the sixth injection. The following changes occurred during the arterial infusion of histamine: increase in heart rate, stroke volume, cardiac output, skin temperature and oxygen saturation in the femoral vein, and decrease in blood pressure. These physiological changes are similar to those seen in association with arteriovenous shunts, and it is suggested that the beneficial effects of intra-arterial histamine may be due to acute arteriovenous shunting and subsequent improvement in the collateral arterial circulation.

Bishydroxycoumarin in Acute Myocardial Infarction.—Many physicians find it difficult to decide whether or not a patient sustaining a mild episode of acute myocardial infarction should be treated with anticoagulant drugs. The committee for the evaluation of anticoagulants in treatment of coronary occlusion with myocardial infarction of the American Heart Association has recommended the employment of such therapy in patients with this disease unless contraindications to anticoagulant therapy exist. However, an analysis of the mortality and incidence of thromboembolism in 1,047 cases of acute myocardial infarction treated conservatively shows no justification for the routine employment of bishydroxycoumarin (dicumarol®) in this disease. The death rate in 489 "good risk" cases in which anticoagulants were not given was only 3.1%, and the incidence of thromboembolism in the same group was only 0.8%. The mortality preventable with bishydroxycoumarin would have been, at most, only 1.0% in these selected cases. Since such small benefit is more than likely to be nullified or even overbalanced by complications induced by bishydroxycoumarin, the authors feel that anticoagulant therapy should be reserved for the more serious cases of acute myocardial infarction, in which the frequency of thromboembolism justifies the risk entailed in its use.

Gastroenterology, Baltimore

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Effects of X-Ray Radiation on Pancreatic Function in Dogs. R. F. Rauch and K. W. Stenstrom.—p. 595.
Lack of Effect of Crystalline Lysozyme on Isolated Intestinal Pouch of Dog. H. C. Moeller, A. P. Klotz and J. B. Kirsner.—p. 604.
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Beneficial Action of Certain Salts on Course of Peptic Ulcer in Shay Rat. C. Funk, P. Tomashefsky R. Soukup and A. Ehrlich.—p. 625.
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GP (J. Am. Acad. Gen. Practice), Kansas City, Mo.

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Illinois Medical Journal, Chicago

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*Case of Fatal Thyroid Crisis Occurring After Radioactive Iodine Therapy. R. B. Nelson, J. B. Cavenagh and A. Bernstein.—p. 265.
Traumatic Diaphragmatic Hernia Complicated by Acute Traumatic Pancreatitis. F. B. Tabaka, S. J. Nigro and E. Nora.—p. 269.
Dermatitis Medicamentosa in Newborn. M. M. Scannell, J. E. Rorie and C. J. White.—p. 271.
Alcoholic Cirrhosis Simulating Idiopathic Polycythemia Vera. E. Greer.—p. 272.

Thyroid Crisis After Radioactive Iodine Therapy.—Surgical and diagnostic procedures, digitalis intoxication, infections, and psychic shock have been considered capable of precipitating thyroid crisis in patients with thyrotoxicosis. More recently it has been observed that treatment of severe thyrotoxicosis with radioactive iodine may be followed by thyroid crisis. The authors present the case of a man who had thyrotoxicosis with decompensated thyrotoxic heart disease. The patient was treated with maintenance doses of digitalis, bed rest, and phenobarbital. He was given subcutaneous injections of meralluride (mercuhydrin®) solution, which resulted in excellent diuresis. Later he was given a therapeutic dose of 5 mc. of I^{131}. Being restless and impatient, he refused to remain in the hospital. Seven days after I^{131} therapy, he appeared nervous, sweaty, and moderately tachypneic, and had pitting ankle edema. The heart rate was 150 and irregular. He was given 10 drops of strong iodine solution (Lugol's solution) three times daily and 1 cc. of meralluride injection subcutaneously. He was instructed to stay in bed and return to the clinic in one week. The following day he was violently ill with vomiting and diarrhea. Approximately 30 hours later he was brought to the hospital in an irrational state, and died two hours later. The high grade of thyrotoxicosis and the velocity of its development with thyrocardiac complications might have led to thyroid crisis under any therapeutic regimen; however, its occurrence following radioactive iodine therapy suggests that this therapy acted as a factor. The authors recommend daily observation during the critical two week period following I^{131} therapy, when a dangerous exacerbation of toxicity might occur. In selected patients with marked toxicity and limited cardiac reserve, preliminary control with propylthiouracil may be indicated before radioactive therapy is given.

Journal of Applied Physiology, Washington, D. C.

4:699-770 (March) 1952

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Journal of Aviation Medicine, St. Paul

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Paradoxical Action of Oxygen. R. Grandpierre and C. Franck.—p. 181.
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Journal of Bacteriology, Baltimore

63:429-562 (April) 1952. **Partial Index**

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Observations on Strain of Neisseria Meningitidis in Presence of Glucose and Maltose: II. Studies with Washed Cells. C. Fitting and H. W. Scherp.—p. 545.

Minnesota Medicine, St. Paul

35:273-380 (April) 1952

*Cervical Smears in Detection of Preclinical Carcinoma. D. C. Dahlin, E. Mussey and M. B. Dockerty.—p. 305.
Are Grievance Committees Serving Their Purpose? R. E. Fitzgerald.—p. 313.
Doctor's Responsibility to His Patient. E. M. Hammes.—p. 316.
Interpretation of Medical Case Costs. J. D. McCarthy.—p. 318.
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Acute Laryngotracheobronchitis. F. G. Folken.—p. 336.
Routine Chest Examinations in Private General Hospital. J. P. Medelman and J. F. Briggs.—p. 340.

Cervical Smears in Detection of Carcinoma of Uterine Cervix.—Cervical smears are an important means of detecting carcinoma of the cervix, which ranks second to carcinoma of the breast as a cause of death from malignant diseases in women. More than 10,000 smears have been obtained almost routinely in the obstetrics and gynecology section of the Mayo clinic since August, 1950. Records were reviewed of 92 of these patients who proved to have carcinoma of the cervix, although at the time of examination lesions suggesting its presence were not seen. Of these patients, 73 had cervical carcinoma in situ, and their average age was 42.1 years; 11 patients had early invasive carcinoma, and their average age was 47.8 years. Of them 81 were married, 4 widowed, 6 divorced, and one single; 12 had never been pregnant, 4 had had pregnancies resulting in abortion, and 76 had had at least one full-term pregnancy. Only 17 sought medical aid because of gynecologic problems, and in 11 only was malignant growth suspected, whereas in 17 the cervix seemed to be normal. Leukoplakia, believed to be a precancerous lesion, was found in eight patients only. The appearance of the cervix justified biopsy in 49, whereas only a smear was taken in 38 at the time of the first examination. When the patient has an obvious carcinoma or positive and equivocal smears biopsy should be done before treatment is undertaken. Many times, however, a single smear was more efficacious than random biopsy. In detection of carcinoma of the uterine fundus smears were not helpful. It appeared that patients whose smears are positive for carcinoma of the cervix do not have characteristic symptoms or physical signs, and carcinoma in situ and invasive carcinoma of the cervix may be asymptomatic and/or clinically undetectable. Annual or biannual smear screening of apparently normal women is a good preventive measure.

New York State Journal of Medicine, New York

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Primary Closure in Chronic (Nontuberculous) Osteomyelitis. E. B. Leahey.—p. 1045.

North Carolina Medical Journal, Winston-Salem

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*Newer Aspects of Lipid Metabolism in Atherosclerosis. F. W. Ellis.—p. 169.
Laboratory Diagnosis of Lymphogranuloma Venereum. G. P. Manire.—p. 176.
On Pathogenesis of Obesity. A. T. Miller Jr.—p. 179.
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Relation of Some Recent Biochemical Investigations to Diseases of Intermediary Metabolism. C. E. Anderson.—p. 189.
Fibrinolytic Enzyme System of Dog Serum. J. H. Lewis and J. H. Ferguson.—p. 196.
Veratrum. W. C. Nungesser.—p. 203.
Secretion and Enterohepatic Circulation of Bile Acids: Replacement of Bile Acids in Biliary Insufficiency. J. L. Irvin.—p. 206.

Lipid Metabolism in Atherosclerosis.—Results of recent studies reported in the literature indicate that the various lipid and lipoprotein components of serum are transported normally in a physicochemical state of equilibrium that maintains lipid material in a finely dispersed stable "emulsion." It is believed that any deviation in normal lipid metabolism great enough to unbalance this mechanism results in the aggregation of abnormal colloidal lipid-bearing particles, which are susceptible to deposition in the arterial intima. At least three indications of such a metabolic defect have been related recently to the etiology of atherosclerosis: (1) elevation of the serum cholesterol-phospholipid ratio, (2) sustained chylomicronemia (the presence in human serum of relatively large and numerous fat particles determined by the dark field microscope), and (3) certain abnormal serum lipoproteins in the form of giant molecules. The etiology of the faulty mechanism that produces these changes is obscure at present. However, some hormonal deficiency or imbalance is suggested by the close association of degenerative vascular disease with certain alterations of endocrine function, and by increasing evidence that lipid metabolism is influenced by the action of various hormones. Thus it is possible that atherosclerosis may be shown ultimately to be a metabolic disease initiated by abnormalities in lipid metabolism that result from endocrine dysfunctions. Under these conditions, an excessive fat intake by a person with a preexisting metabolic deficiency might be important in determining his susceptibility to atherosclerosis. In this connection it has been demonstrated that certain atherosclerotic patients benefit from dietary restriction of fats. This concept of the role of metabolism does not exclude other factors in the pathogenesis of atherosclerosis.

Pennsylvania Medical Journal, Harrisburg

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Radiology, Syracuse, N. Y.

58:481-636 (April) 1952

*Angiocardiography in Congenital Heart Disease of Cyanotic Type: III. Observations on Complete Transposition of Great Vessels. R. N. Cooley and R. D. Sloan.—p. 481.
Dental Roentgenologic Manifestations of Systemic Disease: II. Developmental Disturbances. E. C. Stafne.—p. 507.
Roentgen Studies of Maxillae and Mandible in Sickle-Cell Anemia. I. B. Robinson and B. G. Sarnat.—p. 517.
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Roentgen Manifestations of Lymphosarcoma of Stomach. P. H. Debb and W. L. Stilson.—p. 529.
Emergency Diagnosis of Upper Digestive Tract Bleeding by Roentgen Examination Without Palpation ("Hampton Technic"). H. C. Knowles, B. Felson, N. Shapiro and L. Schiff.—p. 536.
Two Unusual Tumors of Diaphragm. M. M. Keirns.—p. 542.
Teleroentgenographic Pelvimetry: Preliminary Report. D. R. Germann.—p. 548.
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The Country Radiologist: Presidential Address. J. S. Bouslog.—p. 556.
Absorption of 2-mev Constant Potential Roentgen Rays by Lead and Concrete. W. W. Evans, R. C. Granke, K. A. Wright and J. G. Trump.—p. 560.
Monitoring of Diagnostic X-Ray Department. L. E. Jacobson, J. J. Schwartzman and S. Heiser.—p. 568.

Angiocardiography in Complete Transposition of the Great Vessels.—The angiocardiographic findings in 13 patients with proved complete transposition of the great vessels are described. They are classified according to the presence of associated anomalies, such as interatrial or interventricular septal defects, a patent ductus arteriosus, or a combination of these defects. The procedure is evaluated according to its ability to establish a diagnosis of transposition of the great vessels and to reveal the presence and size of the concomitant anomalies. It was found that the angiocardiographic findings were sufficient to make the diagnosis of complete transposition of the great vessels in 10 of the 13 cases. The diagnosis had been suspected, however, from routine clinical and roentgenographic findings. In three cases in which the diagnosis was not suspected, angiocardiography did not establish the diagnosis. In each instance bizarre features made the diagnosis more difficult. The presence and size of a significant associated interatrial defect can be determined with a fair degree of reliability by angiocardiography. If the defect is so large that further enlargement by surgery is not indicated, the authors believe that it will always be demonstrated by a technically satisfactory angiocardiogram. Concomitant interventricular defects rarely were directly demonstrated. Occasionally their presence could be inferred from associated findings. Angiocardiography did not demonstrate a patent ductus arteriosus in four cases in which the ductus proved to be over 3 mm. in diameter. No deaths resulted from angiocardiography either in the 13 cases or in an additional group of 38 patients in whom a transposition of the great vessels was thought to be present. Even though most of the patients were severely incapacitated, they tolerated the procedure well, with only a transient period of discomfort. There was an immediate high concentration of the contrast substance in the aorta and presumably in the coronary arteries. The difficulties in differentiating complete transposition from a complete transposition of the aorta with a levoposition of the pulmonary artery are discussed. Other limitations and shortcomings of the procedure are considered.

Rhode Island Medical Journal, Providence

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South Dakota Journal of Medicine, Sioux Falls

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Tennessee State Medical Assn. Journal, Nashville

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Texas Reports on Biology and Medicine, Galveston

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Polysaccharides of Serum Proteins in Cancer. M. R. Shetlar.—p. 228.
Experiences in Spontaneous Regression of Neoplastic Disease in Man. F. W. Stewart.—p. 239.

Cancerigenicity of Nickel in Rats.—In view of the growing importance of metal cancerigenesis, experimental studies were initiated dealing with the following metals or their compounds: arsenic, chromium, chromite ore, nickel, uranium, beryllium, and asbestos. This paper reports results obtained in 70 rats to which pure metallic nickel powder suspended in lanolin was administered by three different parenteral routes: into the femur, pleural cavity, and nasal cavity. Cancerous reactions were observed in 30% of the animals that died from 7 to 16 months after the start of the experiment. Six of the neoplasms were apparently osteogenic sarcomas, one originated from connective tissue at the site of injection, one from the epithelial lining of an osteomyelitis fistula, and two seemed to have developed from abdominal lymph nodes. The evidence obtained strongly suggests that finely dispersed metallic nickel may elicit cancerous reactions. The minimal latent period was about six months. The importance of these observations lies in the fact that reports from some English nickel ore refineries indicated an excessive frequency of cancer of the lung, nasal cavity and nasal sinuses among workers engaged in operations involving exposure to nickel carbonyl. No similar observations have been recorded from countries where the Mond process of nickel ore refining is employed. While some investigators favored the view that exposure to sulfuric acid fumes containing arsenic generated during the refining process was responsible for these respiratory cancers, this view has been largely abandoned in recent years. Circumstantial evidence incriminates exposure to nickel carbonyl vapors.

Virginia Medical Monthly, Richmond

79:233-292 (May) 1952

Abdominal Tuberculosis Without Evident Tuberculosis of Lungs. E. S. Roberts and H. Nushan.—p. 237.
Hemorrhage Due to Dicumarol and Its Analogues. J. B. Twyman. —p. 246.
*Herpes Zoster of Cranial Nerves. F. H. McGovern and G. S. Fitz-Hugh. —p. 250.
Inequality of Leg Length. E. B. Carpenter and A. A. Kirk.—p. 254.
Lower Nephron Nephrosis and Artificial Kidney—Case Report. F. M. Jacobs.—p. 260.
Neuroses of Everyday Living. D. C. Wilson.—p. 264.
Non-Traumatic Gas Gangrene. H. W. Kimball and A. J. Rawson. —p. 269.
Chloromycetin Palmitate—Clinical Evaluation in Allergic and Non-Allergic Children and Adults. J. W. Thomas, F. R. Kelley Jr., A. M. Edmonds and E. L. Kindig Jr.—p. 272.

Herpes Zoster of Cranial Nerves.—With the exception of herpes zoster ophthalmicus, herpes zoster of other cranial nerves is rarely diagnosed, although it occurs often. The basic lesion of herpes zoster is probably an inflammatory process in the dorsal root or cranial sensory nerve, caused by a neurotrophic and epidermotrophic virus. The cutaneous or mucosal vesicle is thought to result from peripheral migration of the virus from the ganglionic focus to cutaneous and/or mucosal epithelial cells. A virus has been shown to be present in the vesicles. The authors discuss the manifestations of herpes facialis (gasserian ganglion), herpes oticus (geniculate ganglion), herpes auditorius (vestibular and cochlear ganglia), herpes pharyngis (glossopharyngeal ganglia), herpes laryngis (vagal ganglia), and herpes occipitocollaris (cervical ganglia). It is common for herpes of cranial nerves to be manifested by only part of the usual symptoms. For example, in the vagus syndrome there may only be pain and vesicles in the hypopharyngeal region, with no involvement of the ear, motor manifestations, or other possible complications. There are also combinations of involvement of multiple nerves, such as herpes facialis with ocular paralysis or facial paralysis, or geniculate and auditory ganglia involvement, or a vagoglossopharyngeal syndrome. In the event of the development of pain and vesicles in a zoster zone of a sensory nerve, it is important to search carefully for possible motor involvement of the fibers of the same nerve or of a neighboring nerve. An attack of herpes zoster does not confer permanent immunity. The disease is self-limited, lasting three to six weeks. Motor paralysis, if present, may last longer, and in elderly and debilitated patients a troublesome, painful, postherpetic neuritis may persist for months. There is no specific therapeutic agent for herpes zoster. Aureomycin or chloramphenicol in large initial doses (4 gm. daily) should be given in selected cases to combat secondary infections and ophthalmic complications. Local treatment should be directed towards combating secondary infections by the use of medicated powders or ointments. Attempt should be made to control pain with such analgesics as the salicylates, with codein or meperidine (demerol®) in severe cases. Caution is necessary in the use of opiates, particularly in cases with persistent postherpetic neuralgia.

Wisconsin Medical Journal, Madison

51:437-440 (April) 1952

Recognition and Treatment of Vaginal Diseases. H. C. Hesseltine. —p. 360.
Treatment of Granulating Burns. S. K. Wynn.—p. 363.
Treatment of Acute Laryngotracheobronchitis. H. C. High Jr.—p. 367.
Duodenal Obstruction from Gallstone: Review and Report of Case. C. F. Broderick.—p. 371.
Intestinal Obstruction of Proximal Ileum Due to Gallstone: Report of Case. B. I. Pippin.—p. 373.
Central Lumbar Disk Simulating Cauda Equina Tumor Occurring in 12 Year Old Boy with Dyschondroplasia. J. J. Van Driest and H. P. Maxwell.—p. 374.

FOREIGN

Acta Medica Scandinavica, Stockholm

142:399-480 (No. 6) 1952

*Cor Pulmonale Resulting from Deformities of Chest. S. Samuelsson.—p. 399.
Clinical Use of Measurements of Clearance and Maximum Capacity of Liver. J. C. M. Verschure.—p. 409.
Hemagglutination Test with Sensitized Sheep Cells in Rheumatoid Arthritis and Some Other Diseases. N. Svartz and K. Schlossmann.—p. 420.
Histological Technique for Examination of Cell Content of Sputum. G. Wihman and I. Bergström.—p. 433.
Adrenal Function in Coronary Thrombosis. O. Forssman, G. Hansson and C. C. Jensen.—p. 441.
Studies on Agglutination of Sensitized Sheep Cells in Rheumatic Diseases: I. Agglutination Titer After Primary Absorption of Serum by Sheep Cells. S. Winblad.—p. 450.
Id.: II. Nature of Agglutinating Serum Factor. S. Winblad.—p. 458.
Influence of Heparin on Coagulability of Fibrinogen. H. Winazzer.—p. 468.

Cor Pulmonale Resulting from Chest Deformities.—Severe and possibly also moderate chest deformities have a harmful effect on the thoracic viscera. Deformities such as kyphoscoliosis, scoliosis, and kyphosis may affect the lungs, causing atelectasis as a result of lack of space, compensatory dilatation and emphysema, and frequent attacks of bronchitis. The heart is frequently displaced upwards to the side opposite the scoliosis, and this displacement is often accompanied by bending and twisting of the aorta and great vessels, especially in cases of right-sided kyphoscoliosis and scoliosis. The changes accompanying right-sided kyphoscoliosis cause compression and concavity of the left chest, which in turn produce a horizontal position of the heart. Hypertension in the pulmonary circulation caused by the pathological pulmonary changes associated with kyphoscoliosis results in hypertrophy and dilatation of the right side of the heart. The author's series, chosen from persons awarded invalidity pensions on account of spinal deformity by the Danish Invalidity Court between 1935 and 1944, included 41 living patients and 62 who had died. Grade 3 deformity was present in most cases. Signs of cor pulmonale were found in 20 of the 41 patients still living. Electrocardiograms were made in all 41 cases; right cardiac strain appeared in 15 and left cardiac strain in 8. Dyspnea is usually the earliest symptom; it may be present for years without interfering with working capacity, but when it is increased by minor exertion, heart failure is impending. The patients with cor pulmonale showed greater debility than the others, and orthopnea with or without cyanosis was found in 15. Study of the death certificates and inquiry from attending physicians showed that 39 of the deceased patients had had signs of cor pulmonale; of these, 37 died in heart failure. Causes of death next in frequency were pneumonia and, according to the certificates, "heart disease" and "myocardial degeneration." The depressant effect of morphine on the respiration is particularly dangerous to patients with spinal deformities who are in pulmonocardiac failure; one patient in this series died following injection of 1 cc. of morphine. Another death followed administration of a local anesthetic.

Archivum Chirurgicum Neerlandicum, Arnhem

4:1-80 (No. 1) 1952. **Partial Index**

*Use of Küntscher's Intramedullary Pin in 541 Cases. E. J. Moeys.—p. 1.
Method of Suturing in Operations for Congenital Oesophageal Atresia. J. ten Kate.—p. 43.
Wire Extension in Treatment of Horizontal Fracture of Palate (Guérin's Fracture). P. L. Allaart.—p. 49.
Results Obtained in Treatment of Acute Appendicitis. R. R. Swinnen.—p. 53.

Use of Küntscher's Intramedullary Pin.—Kütscher's intramedullary pin was used in 541 fractures treated at the surgical clinic of the University of Utrecht and at a hospital in Nijmegen. Most of the patients observed at the latter hospital had war injuries and were treated without penicillin, whereas most of those treated in Utrecht were admitted after the war, when penicillin and other antibiotics were available. About 50% of the total were treated without antibiotics. Küntscher's method consists of introducing a stainless steel pin, U-shaped in cross section, into the medullary cavity through a small incision well away from the site of the fracture. Failure following the use of Küntscher's medullary pin is almost always due to technical errors or imperfections. Good results can be expected only when these imperfections are avoided. The method is superior to others in that it makes possible stable fixation of the fracture and complete functional restoration. The method is indicated in all cases of transverse diaphyseal fracture of the long bones. Other indications depend on the skill and experience of the operating surgeon. Since the "open" and the "closed" method at present offer an equal risk of infection, the "open" method is to be preferred, because it is technically less difficult and safer. Indications for use of this method will increase with further simplification and introduction of various special types of pins. The method should be used only by those who are experienced in its use and have at their disposal all necessary equipment.

Archivos Españoles de Urologia, Madrid

8:1-96 (Jan.) 1952. **Partial Index**

*Strangulation of Penis. E. Pérez Castro and A. Pérez Coutiño.—p. 1.
Neurogenic Dysfunction of Bladder Treated by Resection of Hypogastric and Presacral Nerves. L. Cifuentes and C. Younger.—p. 31.
Prolonged Streptomycin Treatment in Renal Tuberculosis. F. Romero Aguirre.—p. 44.

Strangulation of Penis.—The most frequent causes of strangulation of the penis are revenge, onanism, and superstition. Strangulation of the penis is followed within the first 24 hours by acute edema, stagnation of urine, and frequently by fulminant gangrene, which can be prevented by prompt removal of the strangulating body. Cases of tolerance for nine months before appearance of gangrene have been reported. The authors report two new cases in a child and an onanistic man 73 years old. The victims were seen 3 and 15 days, respectively, following the strangulation. The case in the old man is unique in the literature. The metallic cylinder introduced weighed 155 gm. It was 8.5 cm. long, with a lumen 2.5 cm. in diameter and walls 0.5 cm. thick. Amputation under general anesthesia was necessary.

Belgisch Tijdschrift voor Geneeskunde, Leuven

8:337-386 (April 15) 1952. **Partial Index**

Results of Pulmonary Resection for Bronchiectasis. L. D. Eerland and R. R. Swinnen.—p. 337.
Catheterization of Heart in Study of Acquired Heart Disease. K. Vuylsteek.—p. 358.
Combat of Cancer in Belgium. J. De Backer.—p. 373.
*Diagnosis and Treatment of Bronchopneumonia Due to Whooping Cough. Beckers.—p. 377.

Bronchopneumonia Due to Pertussis.—Beckers stresses that in diagnosis of bronchopneumonia due to pertussis two forms should be differentiated: (1) the pneumowhooping cough of Julien-Marie, which is hematologically characterized by the presence of many leukocytes and lymphocytes; (2) bronchopneumonia of the mixed infection type, with an increase in neutrophils but a less decisive increase in leukocytes. The treatment of these two forms differs: in the so-called pneumowhooping cough, the author recommends administration of a specific antibiotic such as terramycin, aureomycin or streptomycin, inasmuch as there may be a difference in sensitivity of some strains against these specific antibiotics. In the treatment of bronchopneumonia due to mixed infection it is essential to administer besides one of these antibiotics also penicillin, which has previously been the drug of choice in treatment of pneumococci and streptococci, chief causes of mixed infections.

British Journal of Dermatology, London

64:169-212 (May) 1952. ***Partial Index***

Primary Systematised Amyloidosis with Macroglossia. G. C. Wells.—p. 169.
Histological Changes in Tonofibrils in Vesicular and Bullous Disease of Skin. T. G. Nelemans, F. J. Keuning, T. G. van Rijssel and M Ruiter.—p. 177.
Staining of Thick Sections of Skin. E. A. Leach.—p. 183.
Making and Fitting of Plastic Noses. H. W. Siemens and R. D. G. P Simons.—p. 190.

British Journal of Industrial Medicine, London

9:93-164 (April) 1952

History of Lung Diseases of Coal Miners in Great Britain: Part II. 1875-1920. A. Meiklejohn.—p. 93.
Pneumoconiosis of Coal Miners in North East England with Special Reference to the Durham Coalfield. R. I. McCallum.—p. 99.
Mechanism of Dispersion of Coal Particles in Lungs of Miners. A. Policard.—p. 108.
Sampling Procedure for Measuring Industrial Dust Exposure. P. D. Oldham and S. A. Roach.—p. 112.
Dust Sampling and Lung Disease. C. N. Davies.—p. 120.
*Incidence of Bladder Tumours in Dyestuffs Factory. T. S. Scott.—p. 127.
Urinary Coproporphyrins in Lead Poisoning. J. E. Kench, R. E. Lane and H. Varley.—p. 133.
Studies on Cotton Dust in Relation to Byssinosis: Part I: Bacteria and Fungi in Cotton Dust. G. Furness and H. B. Maitland.—p. 138.
Cardiovascular Disease in Cotton Workers: Part II: Clinical Study with Special Reference to Hypertension. R. S. F. Schilling, N. Goodman and J. G. O'Sullivan.—p. 146.

Bladder Tumors Following Exposure to Dyes.—The occurrence of bladder tumors in workers in the dyestuffs industry was observed by Rehn in Germany as early as 1895 and by the third decade of this century exposure to beta naphthylamine and to benzidine had been accepted as a cause of bladder tumors by many European observers. American investigators, however, are less certain of the carcinogenicity of benzidine, and stress that it cannot be accepted as such until it is known that benzidine workers without exposure to beta naphthylamine have developed tumors of the bladder. This paper reviews 66 cases of bladder tumors in workers engaged in the manufacture of dyes. Thirty of the 66 patients had been exposed only to benzidine and never to beta naphthylamine, and for this reason benzidine is considered as dangerous a carcinogen as beta naphthylamine. In 15 patients the tumors are attributed to beta naphthylamine alone and in others to mixed exposures. For those entering the industry under 30 years of age the average age of onset of tumor was 42 and of death was 44; of those between 30 and 40 the average age of onset was 51 and of death 54; and in those over 40 the average age of onset was 58 and of death 66. According to the registrar general's figures for 1947 the average age of death of 1,417 males in England and Wales who died of neoplasms of the bladder, urethra, and ureter was 67.5 years. Men under the age of 30 should not be employed in the manufacture or handling of substances known or suspected to be bladder carcinogens and, when possible, healthy men over 40 should be employed on these processes. Methods of prevention are discussed, and it is pointed out that a different method of manufacture for beta naphthylamine has been introduced.

British Medical Journal, London

1:987-1042 (May 10) 1952

*Splenic Puncture. J. B. Chatterjea, C. M. Arrau and W. Dameshek.—p. 987.
Sarcoma of Spleen. P. Story and W. E. Gibb.—p. 990.
Histological Study of Onchocerciasis Treated with Hetrazan. F. Hawking.—p. 992.
Further Observations on Capillary Resistance and Adrenocortical Activity. H. N. Robson and J. J. R. Duthie.—p. 994.
*Responsiveness to A.C.T.H. and Cortisone in Rheumatoid Arthritis. J. H. Kellgren, O. Janus, R. Moore and D. S. Jackson.—p. 997.
Effect of Repeated Injections of A.C.T.H. upon Bone Marrow. G. Hudson, G. Herdan and J. M. Yoffey.—p. 999.
Noradrenaline, Adrenaline, and Human Circulation. H. J. C. Swan.—p. 1003.
Statures of Group of University Students and of Their Parents. J. V. G. A. Durnin and J. B. de V. Weir.—p. 1006.

Splenic Puncture.—Splenic aspiration was performed in 43 patients with different diseases of the blood associated with enlargement of the spleen. The cellular pattern of the aspirated material was studied under oil immersion. Evidences of myeloid metaplasia were observed in patients with polycythemia vera and chronic granulocytic leukemia, and in one patient with cirrhosis of the liver. The association of a fibrotic or sclerotic bone marrow with extensive myeloid metaplasia of the spleen is an absolute contraindication to splenectomy, since the spleen in these cases represents the major hematopoietic tissue. The increased number of erythroblasts found in some of the patients with chronic lymphatic leukemia, acquired hemolytic anemia, and cirrhosis of the liver is probably indicative of small ectopic foci of erythropoiesis. Their presence in association with a cellular or hypercellular marrow does not necessarily contraindicate splenectomy, and the recommendation for splenectomy in these cases should be based only on the fundamental disease and on the presence of associated hemolytic anemia. Splenic puncture is useful in the diagnosis of atypical or cryptic types of aleukemic leukemia and leukosarcoma when the marrow picture may not be completely diagnostic or may even be normal. This is particularly true in primary lymphosarcoma and in reticulum cell sarcoma of the spleen in which splenomegaly may be the only abnormal finding. In congestive splenomegaly due to various causes such as cirrhosis of the liver, corroborative evidence in favor of the diagnosis may often be provided.

Corticotropin and Cortisone in Rheumatoid Arthritis.—A course of corticotropin (ACTH), consisting of 12 injections of 25 mg. intramuscularly every six hours, was given to 17 patients with rheumatoid arthritis. After a rest period of at least four days a similar course of cortisone was given, using an oral dose of 50 mg. of aqueous suspension every six hours. The clinical and eosinopenic responses of the patients to these courses were studied. Four patients responded fully to both drugs, four gave no response with either drug, four responded fully to cortisone but only partially or not at all to corticotropin, while one responded fully to corticotropin and only partially to cortisone. The remaining patients gave only partial responses to either or both drugs. The authors' observations suggest that with cortisone and corticotropin great differences in responsiveness may commonly be encountered in patients with rheumatoid arthritis, and the individual patient's responsiveness should be considered before any treatment with these drugs is begun. The single dose test with 50 mg. of cortisone orally and with a single intramuscular injection of 25 mg. of corticotropin may give a useful indication of this, though the three day test would be more reliable. The occurrence of withdrawal deterioration in three patients in whom the drug had had no apparent effect suggests that the differing response of individual patients to administered cortisone and corticotropin may be partly a reflection of the level of endogenous production. If the administered dose of hormone greatly exceeds endogenous production, it causes some degree of hypercorticism, with eosinopenia and suppression of the inflammatory reaction, resulting in a clinical response; but, if the administered dose is less than the amount produced endogenously, the pituitary-adrenocortical system merely adjusts itself to a lower level of production and the net result is no change, though there may be a temporary withdrawal deterioration while endogenous production is being resumed.

Deutsche medizinische Wochenschrift, Stuttgart

77:605-636 (May 9) 1952. **Partial Index**

General Resistance and Chemotherapy. W. Pfannenstiel.—p. 609.
Syphilitic Infants from Healthy Mothers? A. Gaase.—p. 611.
Acute Infection and Manifestations of Vitamin B_2 Deficiency. G. Rewerts.—p. 612.
Neurologic Diagnosis and Prolapse of Intervertebral Disk. K. Römer.—p. 614.
Clinical Aspects and Pathogenesis of Periarthritis Coxae (Gluteal Form). W. Belart.—p. 617.
*Reduction in Blood Pressure by Hexamethonium Bromide to Prevent Surgical Bleeding. P. Wilflingseder.—p. 619.

Hexamethonium Bromide to Prevent Surgical Bleeding.—Wilflingseder reviews observations on more than 50 operations carried out at the University of Innsbruck with drug-induced blood pressure reduction. Extensive operations on face and neck were greatly facilitated by the use of hexamethonium bromide. Transplantation of large skin flaps is largely dependent on whether the formation of a hematoma can be prevented, and this is possible by means of blood pressure reduction with hexamethonium bromide. Fenestration operations, antrotomy, and operations on the neck are also facilitated by blood pressure reduction. In operations for hernia of the nucleus pulposus blood pressure reduction is helpful, provided the patient can be put in proper position, with head and upper body lowered. Thoracic surgery was likewise facilitated by the use of hexamethonium. In intra-abdominal and proctologic operations the method does not seem indicated, because putting the patient into a position that will drive the blood away from the surgical field is difficult or impossible, but at the Innsbruck clinic the method was tried in urologic operations such as prostatectomy and electroresection, so far with favorable results. From 20 to 50 mg. of hexamethonium bromide is administered intravenously as soon as regular respiration has set in following induction of anesthesia. Then the surgical field is elevated and up to 200 mg. more is given or the surgical field is elevated further until the surgical field is free of blood. If muscular relaxation is desirable for intubation or operation, decamethonium should be given rather than curare, in order to avoid antagonism of the hexamethonium. Generally from three to five minutes are required until pressure has been reduced to 65 to 55 mm. Hg. If this low pressure cannot be reached, even values of 90 to 80 mm. Hg will be helpful. As a rule, surgery should not last more than two hours, but the surgical posture must be maintained for a few hours after operation to avoid postoperative hemorrhage. The method is contraindicated in children, and in patients whose general condition is poor, who are subject to anginal attacks or cerebral thrombosis, or who have chronic pulmonary disorders. The physiology of the method and all possible risks are not yet fully explained.

Die Medizinische, Stuttgart

Year 1952: 681-716 (May 17). **Partial Index**

*Partial Renal Infarction and Hypertension Resulting from Ligature of Aberrant Renal Vessels. H. Boeminghaus and F. J. Götzen.—p. 681.
*Myocardosis—New Concept in Evaluation of Cardiac Disturbances. W. Rosenblatt.—p. 684.
β,β'-Dichlorodiethyl Sulfide Poisoning. H. Dickel, A. Fincke and H. Gohr.—p. 686.
Prophylaxis of Pericardial Adhesions by Oleopericardium According to Technique of Kux. R. Günther.—p. 690.

Renal Infarction and Hypertension.—In 38 patients between the ages of 17 and 63 operated on for various renal disturbances, a true aberrant renal vessel, i. e., an artery supplying the parenchyma of the kidney or a vein of the parenchyma, was either sectioned or ligated. Permanent hypertension developed in 7 (19.4%) of the 38 patients. As a rule pulsating, aberrant vessels should not be sectioned and the intervention should be limited to nephropexy, ureterolysis, or transplantation of the ureter. Despite favorable results of a clamping test, ischemia of a large area of the kidney may result from sectioning of an aberrant vessel. Excision of the ischemic area should then be performed to prevent hypertension. In cases of considerable permanent hypertension, nephrectomy is the method of choice. Experience showed that the younger the patients and the shorter the duration of the hypertension the better the chances for the pressure to be restored to normal. Nephrectomy must be performed before irreversible changes in the vessels of the other kidney occur that will maintain the hypertension. In one of the authors' patients, as early as within three months of the onset of the hypertension arteriosclerotic changes were microscopically demonstrated in the portions of the kidney not involved in the infarction. In cases in which hypertension has continued for some time, it seems advisable to combine nephrectomy with lumbotransdiaphragmatic sympathectomy. Since malignant hypertension may result from partial renal infarction, blood pressure measurements should be taken for prolonged periods in cases in which ligature of aberrant renal vessels could not be avoided. The authors believe that the occurrence of hypertension depends on a hereditary predisposition to hypertension, with an abnormal vascular reaction.

New Concept of Myocardosis.—In evaluating cardiac disturbances in his patients the physician may be aided by a new concept of myocardosis suggested by Wuhrmann. According to this Swiss worker, myocardosis may be considered a syndrome in patients with cardiac disturbances associated with changes of the blood proteins (dysproteinemia). The latter changes may very likely be the cause of the cardiac disturbances. This new concept will be useful in all cases in which the dysproteinemia can be easily demonstrated by combined simple methods, such as determination of total protein content of the blood, Takata test, cadmium sulfate test, Weltmann's serum test, thymol turbidity test, and blood sedimentation rate, and in which no other cause of the cardiac disturbances can be found. Myocardosis may frequently be associated with disease due to malnutrition, but is usually masked by dystonia.

Edinburgh Medical Journal

59:213-260 (May) 1952

Clearance Tests of Renal Function. C. P. Stewart.—p. 213.
Pathology of Foetal and Neonatal Asphyxia. A. R. Macgregor.—p. 229.
Assessment of Inadequate Lactation. R. A. Miller.—p. 238.
Vitamin B Complex as Supplement in Infant Feeding. J. A. Chalmers.—p. 247.

Journal de Chirurgie, Paris

68:329-408 (May) 1952

Postoperative Syndrome of Potassium Deficiency. R. Montant, H. C. Plattner, R. Faller and G. Mottironi.—p. 329.
*Possibilities and Limits of Methods of Removing Obstruction of Common Bile Duct by Administering Chemicals or Pharmacodynamic Substances. J. Sénèque, M. Roux and C. Chatelin.—p. 340.

Use of Chemical and Pharmacodynamic Substances in Obstruction of Common Bile Duct.—Pribram's method of treatment for residual calculi combined with intravenous administration of pharmacodynamic substances following cholecystectomy, cholecystostomy, or choledochotomy was used by the authors in nine patients. Two or three times a day, 3 or 4 cc. of a mixture of 8 parts of ether and 2 parts of alcohol were instilled through an indwelling tube in the common bile duct. This was followed by instillation of 5 to 10 cc. of tepid olive oil. In the morning and in the evening the patients in addition were given slow intravenous injections of aminophylline or of sodium dehydrocholate. Excellent results were obtained in eight of the nine patients, while one patient was a therapeutic failure. These results suggest that postoperative medical treatment of obstruction of the common bile duct is indicated if the presence of small calculi has not been recognized during surgical intervention performed under radiomanometric control. In all cases with moderate dilatation of the biliary passages and slightly increased pressure values, the calculi may be removed as a rule by instillations of olive oil alone, or only by injections of aminophylline, sodium dehydrocholate, or even atropine. Such treatment may be likewise effective in cases in which, because of the general condition of the patient, a simple cholecystostomy was done and calculi deliberately were not removed. Besides, if small calculi have been detected closely adhering to the wall of the common bile duct because of concrete biliary sediment, simple drainage of the common bile duct may be deliberately carried out in the presence of insignificant distention of the bile duct and pressure values below 20, and secondary treatment according to Pribram's technique then may be considered. The only disadvantage of this type of treatment consists in the long duration that occasionally may be required, but it is not associated with any risk. The duration of the treatment can be shortened if the calculi are free and the papilla patent. It is emphasized that the treatment should be given outside of the digestive periods, and aspiration of several cubic centimeters of bile from the bile ducts should always precede instillation of the drugs.

Klinische Wochenschrift, Heidelberg

30:433-480 (May 15) 1952. **Partial Index**

Methods and Results of Metabolic Studies by Means of Radioiodine in Diagnosis of Thyroid and Extrathyroid Diseases. W. Horst.—p. 439.
*Functional and Morphologic Liver Tests in Tuberculous Patients Treated with Thiosemicarbazones. E. Böhlke and H. G. Henkel.—p. 449.
*Relations Between Blood and Bone Marrow Effect of Corticotropin, Particularly as Regards Eosinophils. R. Gross and U. Siecke.—p. 456.
Investigations on Metabolism of 5-Iodo-2 Thiouracil. E. Klein.—p. 462.
Is the Eosinophil Test Specific? A. Hitzelberger, W. Ruppel and L. Weissbecker.—p. 470.

Liver Changes Resulting from Treatment with Thiosemicarbazone.—The damaging effects of the thiosemicarbazones on the liver were studied with the aid of 124 puncture biopsies carried out on 86 patients who were receiving these drugs in the treatment of tuberculosis. In a number of patients, the liver, which had already been impaired by the tuberculous toxin, sustained added damage, which became evident particularly in increased peripheral fatty lobular degeneration and in an increase in periportal round cell infiltrations, in those who were treated with "tebethion" (*p*-acetylaminobenzaldehyde-thiosemicarbazone). Comparisons between certain functional tests of the liver and the puncture biopsies indicate that the functional tests are not sensitive enough to detect mild parenchymal liver lesions. Only a third of the functional tests indicated any pathological structural changes in the tuberculous patients. Fatty degeneration and focal lesions such as tubercles were not revealed by the chemical functional tests. Impaired feeling of well-being on the part of the patient, loss of appetite, and gastrointestinal disturbances are better indicators of the severity of the hepatic lesions than the functional tests, because, with increasing severity of liver damage, the intolerance for the chemotherapeutic substances increases.

Action of Corticotropin on Blood and Bone Marrow.—Blood and bone marrow changes were studied in 12 healthy men after a single injection of 50 mg. of corticotropin and in a woman who had received a total of 1,200 mg. of corticotropin for chronic polyarthritis. It was found that the decided decrease in blood eosinophils was not accompanied by an equal reduction of bone marrow eosinophils. On the contrary, there was an increase, which during prolonged corticotropin therapy became considerable and involved chiefly immature forms. In a patient who had high blood and bone marrow eosinophilia because of Löffler's eosinophilic pulmonary infiltrate, administration of 125 mg. in 24 hours did not cause a reduction in the immature eosinophils. The sharp decrease of eosinophils in the blood under the influence of glucocorticoids is explained on the basis of one or several of the following factors: destruction of cells in the peripheral blood or in certain organs; temporary retention of the cells in certain circulatory regions (disturbance in distribution); and prevention of maturation or of release of cells in the bone marrow.

Minerva Medica, Turin

43:585-616 (March 22) 1952. **Partial Index**

*Late Prognostic Findings in 140 Patients Following Acute Myocardial Infarction. P. Trivella.—p. 586.
*Atypical Forms of Generalized Carcinosis. E. Cacciapuoti.—p. 597.

Prognosis of Myocardial Infarction.—A follow-up was made of 140 patients 3 months to 15 years after an acute attack of myocardial infarction. At the time of the attack 12 patients were less than 40 years old, 47 were between 40 and 49, 42 between 50 and 59, and 39 were more than 60 years old. Of these patients 52% were living in good cardiocirculatory condition after two years, 37.7% after three years, and 18.8% after six years. Angina had appeared in 25 (17.1%), and heart decompensation in 14 (11%). Prognosis of myocardial infarction is favorable in young persons who, as a rule, are healthy at time of the attack, while older persons may have hypertension and angina. In these the infarct is generally asymptomatic, and may be attributed to endocardial factors, whereas in young persons it may occur because of lipid concentration or a tendency to increased blood coagulation. Of these patients 74 had infarction of the anterior wall, 57 of the posterior wall. In two the infarction could not be localized and in seven it did not involve the entire thickness of the ventricle wall. The site of the infarct is not as important as its size. Preexisting hypertension and angina make the prognosis difficult if they are associated with fibrosis and cerebral arteriosclerosis. When the anatomic condition of the heart is the same at the time of the attack, heart decompensation is likely to develop in the patient with hypertension, while in the patient with angina a recurrent infarct is more probable. The shorter the period of time between the two attacks the less the probability of survival. In seven of the nine patients who died the second attack had occurred within one year. Thus a myocardial infarction may be followed by decompensation, angina pectoris, or restoration of good function of the myocardium.

Atypical Generalized Carcinosis.—Two atypical cases of generalized carcinosis are reported. A few days after giving birth to a child, a 41-year-old woman had a temperature elevation and symptoms that suggested puerperal sepsis, which, however, was not confirmed by gynecological examination. Results of serum and blood tests were normal, but roentgen examination revealed small miliary nodules in the lungs of metastatic nature. A latent microscopic neoplasm of the uterus that became active at time of pregnancy was considered. Swelling of the left inguinal lymph nodes occurred, and biopsy revealed epithelial carcinoma. Had the swelling not appeared a correct diagnosis would have been impossible, since autopsy was not permitted. The second patient was a 38-year-old single woman from a family in which five of eight members, including the mother, had died of neoplastic disease. The tumor always appeared when the persons were between 35 and 40 years old. First symptoms in this patient were abdominal effusion, followed later by pleural effusion in the right side, intermittent temperature elevation, sweating, and little involvement of the general condition. Polyserositis of tuberculous nature was considered and at first seemed to be supported by the fact that the pleural effusion receded almost completely, to reappear only toward the end of the disease. Suddenly the symptoms became aggravated, edema of the legs became severe, and abdominal edema regressed only slightly after tapping. The change of fibrinous serum to hemorrhagic serum, the presence of palpable masses in the abdomen, and swelling of the lymph nodes in the axillary region suggested carcinoma. Biopsy of the swollen lymph nodes revealed metastasis. The hypothesis is advanced that the neoplasm was latent and was stimulated by tuberculous disease. In both patients the course was unusual and an early correct diagnosis was not possible.

Nederlandsch Tijdschrift v. Geneeskunde, Amsterdam

96:857-924 (April 12) 1952. **Partial Index**

Osteoid Osteoma. G. Bosman and H. N. Hadders.—p. 865.
*Isolation of Herpes Simplex Virus from Brain of a Patient with Acute Encephalitis. H. A. E. van Tongeren and J. G. Y. de Jong.—p. 871.
Commotio Cerebri. A. Verjaal.—p. 877.

Herpes Simplex Virus in Brain of Patient with Acute Encephalitis.—The four-year-old boy whose history is presented had been ill with headache, vomiting, elevation of temperature, and apathy for four days before he was hospitalized. On the sixth day after onset, the boy lapsed into coma and had convulsions, tremors, a varying degree of spasticity of the legs, and disturbed reflexes. There were also signs of gastrointestinal disturbance. Papilledema with hemorrhages of the fundus oculi were seen shortly before death. No labial herpes or any other form of herpes simplex infection was observed on the skin or mucous membranes. Necropsy corroborated the diagnosis of encephalitis. Virus was isolated from the brain with the aid of mouse passages. Cross immunization tests carried out between the freshly isolated virus strain and an established strain of herpes simplex showed them to be immunologically identical. The etiological connection between herpes simplex virus and meningoencephalitis had been demonstrated by Levaditi and Harvier in 1920 and subsequently by others, but the total number of cases in which such a relationship has been proved is small.

Ugeskrift for Laeger, Copenhagen

114:555-588 (May 1) 1952. **Partial Index**

*Cause of Death in Diabetes Mellitus. F. Heintzelmann.—p. 560.
Status Asthmaticus Treated with Ether Anesthesia. O. Storm.—p. 568.
Thrombocytopenia in Diseases of Liver: Case of Cirrhosis of Liver with Severe Thrombocytopenia and Esophageal Varices. G. Thymann. —p. 571.

Cause of Death in Diabetes Mellitus.—Heintzelmann analyzes the causes of 2,260 deaths of diabetics in Denmark in 1947-1949. In most cases death was due to degenerative heart disorders. This cause of death predominates mainly in the age group of 45 to 64, but is an important cause among men in the age group from 15 to 44, in which death is also often caused by renal diseases, which is more common among men than among women, and by tuberculosis, and in a number of cases by the diabetes itself (diabetic coma). The last-named is almost exclusively the cause of death among diabetic children. In diabetics in the age group over 65 death is most frequently due to senile disorders (cardiac disease, cerebral hemorrhage, and senility). The relatively rare occurrence of cancer as the cause of death in diabetics is stressed. Tuberculosis is the cause of death in diabetics somewhat oftener than in the total population, heart disease and arteriosclerosis are the cause much oftener, and death from renal disease is relatively frequent among diabetics. Among the oldest diabetics death from pyelonephritis is more frequent in men than in women.

BOOK REVIEWS

Cellular Changes with Age. By Warren Andrew, Ph.D., M.D., Professor of Anatomy and Chairman of Department, George Washington University School of Medicine, Washington, D. C. Publication number 127, American Lecture Series, monograph in American Lectures in Anatomy, edited by Otto F. Kampmeier, M.D., Ph.D., Professor of Anatomy and Head of Department, University of Illinois College of Medicine, Chicago. Cloth. $2.50. Pp. 74, with 25 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto 2B, 1952.

This is a brief but comprehensive statement of the anatomic aging changes in living cells. It discusses what is meant by aging of cells in general and specifically in cells of plants, protozoa, and other invertebrates, arthropods, lower vertebrates, and man. It discusses in detail nervous, muscular, skeletal, glandular, fibrous, connective, and surface epithelial tissues and blood in man. Everyone interested in these tissues in health or disease will get valuable and stimulating information. The author suggests answers to some current clinical problems and raises still more questions.

With a broad medical background, Dr. Andrew presents facts primarily as an anatomist and a zoologist. He makes his point that what happens to the cell happens to the man. Studies of aging cells reveal the nature of aging in the man. He points out the great diversity in cells and cell aging processes in man. Some age quickly, breed their kind, and die (blood cells). Some serve best after ceasing to be cells (epithelium); some cells never divide or multiply and never are replaced. Some cells in lower animals live a few hours; in plants, hundreds or thousands of years. The several common varieties of anatomic changes in aging are described, including the variations in mitotic and nuclear changes, accumulations of inert matter, vacuolizations, desiccation, atrophy, degeneration, rejuvenation, and generation. He discusses chemical changes in various parts of the cell and intercellular substances. The works of the leaders in cytology and aging, Lansing, Kountz, Korenchevsky, and Calkins, are cited, and the author's own extensive researches in this field are presented. He notes the significant differences of chronological, physiological, and pathological aging in cells. He stresses the fact that the reason persons and their organs age at different rates and in different ways is due to the aging process in the cells. The book is of much value to medical science and service.

Trompa de Falopio, esterilidad de causa tubaria y asuntos conexos. Por Eduardo Bunster M., médico-jefe de la sección ginecologica del Hospital "El Salvador," Santiago de Chile. Tesis para optar al título de Profesor extraordinario de ginecología en la Universidad de Chile. Cloth. Pp. 549, with 315 illustrations. Guillermo Kraft Limitada, Reconquista 319-27, Buenos Aires, 1951.

This book is intended for physicians interested in gynecology and particularly in sterility. The author, in a compact volume, makes a complete study of the fallopian vestibule and tubes, discussing history, anatomy, histology, and physiology. The physiological aspect is extensively treated, and the author substantiates the opinion that the so-called physiological impermeability of the tubes during the premenstrual stage is erroneous.

There is an orderly study of the different procedures that are used to demonstrate the permeability of the tubes, including hydrointubation, which is seldom mentioned in the English literature. The author modifies the original procedure by the addition of antibiotics which, in his opinion, will make the procedure safer and, at the same time, in some instances assist in treating tubal obstruction.

The surgical treatment of sterility is extensively discussed, and the different procedures are analyzed, taking into account their advantages and disadvantages.

The book is written in Spanish; the subjects are discussed concisely but effectively. This volume, divided into 14 chapters, contains an extensive bibliography.

Surgical Forum: Proceedings of the Forum Sessions, Thirty-Seventh Clinical Congress of the American College of Surgeons, San Francisco, California, November, 1951. Surgical Forum Committee: Owen H. Wangensteen, M.D., et al. Cloth. $10. Pp. 667, with 290 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; 7 Grape St., Shaftesbury Ave., London, W.C.2, 1952.

The forum on fundamental surgical problems was established in 1941, as a permanent feature of the Clinical Congresses of the American College of Surgeons. It was created expressly to give an opportunity to the younger men engaged in research on surgical problems to present their work before the older members of the profession. The publication of this type of literature has two advantages. It gives the younger men an opportunity to present their research, and it stimulates the older men preoccupied with practical clinical work. The 1952 volume covers most of the physiological problems in connection with modern operative procedures. These reflect definitely a shift from purely mechanical aspects of surgery to the more intricate chemical and physiological problems involved. This attractively published volume deals with many problems in the field of surgery. These brief and important papers should be of great interest to the researchers in the surgical field as well as to the older surgeons engaged in clinical work.

Il cancro. Di Mario Musella. Biblioteca moderna Mondadori CCLXIV. Boards. 250 lire. Pp. 156. Off. graf. veronesi dell'editore Arnoldo Mondadori, Verona, 1951.

This book, written by a well-informed layman, is to be read by educated laymen. Mr. Musella is well known in Italy as a "scientific reporter" and has published other scientific monographs for the general public. In this small volume, the author tries to explain simply but accurately what the modern trends are in cancer research, diagnosis, and treatment. In his rapid review, he touches such varied fields as the interpretation of statistics on cancer morbidity and mortality, the importance of constitutional or inherited predisposing factors, the influence of environment, the role played by the different hormones and by enzyme systems, nutritional factors, and the most recent views on the use of enzyme antagonists. He discusses many of the important "early signs" and gives proper emphasis to the enormous importance of early diagnosis, diagnosis at a time at which cancer is still a local disease. Cancers of the different systems and organs are discussed separately in a manner that should be easy to understand for the average educated person. In other chapters, for example, those dealing with hormones, chemotherapy, and chemistry of nutrition, the discussion is probably above the level of the general public. There are some unavoidable controversial statements, but, as a whole, this volume is well worth reading for one who understands the Italian language. The preface to the book is written by Prof. Hans R. Schinz, director of the radium therapy department of the University of Zurich, Switzerland.

Neurosurgery: An Historical Sketch. By Gilbert Horrax, M.D., Sc.D., Chief of Neurosurgical Service, The Lahey Clinic. Publication number 117, American Lecture Series, monograph in American Lectures in Surgery. Edited by Michael E. De Bakey, M.D., Professor of Surgery and Chairman of Department of Surgery, Baylor University College of Medicine, Houston, Texas, and R. Glen Spurling, M.D. Clinical Professor of Surgery, University of Louisville, Louisville, Kentucky. Neurosurgical Division, editor: Barnes Woodhall, M.D., Professor of Neurosurgery, Duke University, Durham, North Carolina. Cloth. $3.75. Pp. 135, with 41 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto, 2B, 1952.

This is a brief and enjoyable summary of the development of neurological surgery. It has been written by one of the most able of America's neurological surgeons. Dr. Horrax has been an active participant for more than half of the 66 years that constitute the modern period of this specialty. He has written, therefore, with a background of personal knowledge of his subject and of personal acquaintanceship with practically every man

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The reviews here published have been prepared by competent authorities and do not represent the opinions of any official bodies unless specifically stated.

who has been responsible for its modern development. In addition to the modern period, Dr. Horrax has dealt briefly with the earlier periods, including the prehistoric era. He stresses the development of neurological surgery on the firm foundations of a thorough knowledge of the nervous system and its diseases and a mastery of sound surgical principles and operative technique. Dr. Horrax has written with the affection of one who has enthusiastically devoted his entire life to his subject.

Manual de radiología clínica. Por el Dr. Juan José Quezada R., profesor de radiología clínica y de fisioterapía en la Escuela nacional de medicina. Cloth. $8. Pp. 254, with 230 illustrations. Impreso por la editorial E.C.L.A.L., Constitución 18, Tacubaya, *México, D.F., 1951.*

This manual partially fills a need for books of this type, particularly in the field of radiology. The majority of the reference books in this field are written in English, German, or French. This résumé on radiology is concise but practical and helpful to the busy practitioner who wants a quick glance at radiological technique or diagnosis. It should also prove helpful to the intern studying radiology and the technician.

Almost every one of the radiological techniques discussed is preceded by a small anatomic description of the part of the body involved. There are numerous graphs and reproductions of radiographs; this makes the understanding of the manual easier and more interesting. The quality of the reproductions is unusually good. There is a bibliography, and an alphabetical index.

The Management of Bronchial Asthma: A Guide to Treatment. By Herbert G. J. Herxheimer, M.D., L.R.C.P.Ed., L.R.C.S.Ed., Research Assistant, University College Hospital Medical School, London. Cloth. $3.45. Pp. 107, with 16 illustrations. Butterworth & Co., Ltd., 4-6 Bell Yard, Temple Bar, London, W.C.2, 1952.

The author attempts, with much success, to guide the inexperienced through the confusion caused by the many methods used in the treatment of bronchial asthma. He points out why one drug will help in certain cases and fail in others and gives detailed information regarding the use of epinephrine, ephedrine, aminophylline, and iodides. He thinks highly of inhalations of isopropylarterenol (isuprel®) hydrochloride, but the average American physician probably prefers inhalations of 1:100 epinephrine. Much space is given to vital capacity tests but none to those breathing tests based on forced breathing over a period of 20 to 30 seconds (the so-called voluntary ventilation capacity). The drugs to be used are tested by vital capacities before and after use. In some cases, the offending allergen is inhaled and any resultant asthma is relieved by inhalation of isopropylarterenol or epinephrine. These inhalation tests are dangerous, as severe attacks of asthma can result.

His discussion on the treatment of psychosomatic factors in asthma is good, especially his statement that "One of the main tasks of the physician must be to strengthen the confidence of the patient," and his note that the patient "should be told that there are good prospects of his improvement, but that it will take time for the correct method of treatment and the individual dosage to be found. This cautious prognosis is usually received well; it prevents disappointment if progress is slow at the beginning."

This volume is small; hence, much is omitted. There is nothing on preventive treatment, which is especially important for children, and no mention of therapy in a hospital. Discussion of the technique of hyposensitization is largely omitted, and the author seems to have little confidence in it; this is in opposition to the experience of most American allergists, for whom the best results in treating bronchial asthma come from carefully observing details regarding avoidance of allergens and hyposensitization against those that act by inhalation. Skin tests are apparently not too useful to the author, and, on page 91, he advises against skin tests "if the history shows definite evidence of polyvalent allergy of the bronchi"; however, it may be questioned why the skin tests, which may give valuable information and which offer no danger if carried out with due precautions, should not be done. Another statement seems wrong: "An examination of the blood for eosinophils is usually not necessary." A minor criticism might be his recommendation of high dosages of drugs, e. g., ephedrine. Despite these objections, there is much in the book that is valuable, especially his ideas on the cause of asthma and his drug therapy.

The Hair and Scalp: A Clinical Study (with a Chapter on Hirsuties). By Agnes Savill, M. A., M.D., F.R.C.P.I., *Consulting Physician to Fitzroy* Square Skin Hospital, London. Fourth edition. Cloth. $5.50. Pp. 316, with 60 illustrations. William Wood & Company (division of Williams & Wilkins Company), Mount Royal and Guilford Aves., Baltimore 2; [printed in Great Britain by Butler & Tanner, Ltd., Frome and London], 1952.

Some of the material in this edition has been reviewed and rewritten, and a few additions have been made. These additions include material on the influence of vitamins and endocrine glands on the condition of the hair and a section on the cold permanent wave. The book is written in an informal, readable style and is by no means a didactic reference work. Unfortunately, a considerable portion of the text consists of comments and case reports based on limited scientific facts, such as a four page discussion of the somewhat antiquated practice of singeing. The volume is completely indexed, and a limited number of references to the recent literature are cited. The book provides a readily accessible source of information of value to the student and to the general practitioner. It cannot be considered as a postgraduate course for the expert.

The Pathology of Diabetes Mellitus. By Shields Warren, M.D., Sc.D., Director of Division of Biology and Medicine, U. S. Atomic Energy Commission, Boston, and Philip M. LeCompte, M.D. Third edition. Cloth. $7.50. Pp. 336, with 115 illustrations. Lea & Febiger, 600 S. Washington *Sq., Philadelphia 6, 1952.*

This edition contains new and revised material on experimental diabetes, diabetes and cancer, diabetes as it affects the kidneys, and on the etiology and pathogenesis of diabetes. A noteworthy addition is the detailed study of 50 autopsies on infants of diabetic mothers. This volume is almost 100 pages longer than the previous edition and contains a good deal of new illustrative material.

The authors have aimed at a complete study of the pathology of diabetes mellitus, based on the analysis of data from more than 800 autopsies of definitely diagnosed cases. Among the subjects covered are diabetes mellitus and the pancreas, pathological evidences of abnormal fat metabolism, the pathology of acidosis and coma, vascular disease in diabetes, the pathology of diabetes in children, pathology of insulin-resistant cases, hyperinsulinism and the pathological effects of insulin, infants of diabetic mothers, cancer and diabetes, experimental diabetes, and medicolegal aspects of diabetes.

There can be no doubt as to the competency of the authors and the adequacy of their treatment of the pathological aspects of the subject. Their pathophysiological considerations are somewhat less adequate, partly because the intricacies of the latter field are undoubtedly less familiar to the authors, trained in another discipline, and partly because they have relied heavily on advice from the Joslin school of diabetes, which does not always correspond with other, equally authoritative views on the subject. With these reservations, however, the pathophysiologic sections serve their purpose in attempting to relate altered structure to abnormal function, and they outline and give references to the important observations that warrant further reading of the original sources. This volume can hardly be recommended as essential for the library of every general practitioner, but it is an excellent reference book and should be owned by every physician who claims a special interest in or knowledge of diabetes.

A Symposium on Essential Hypertension: An Epidemiologic Approach to the Elucidation of its Natural History in Man. Commonwealth of Massachusetts [Recess Commission on Hypertension]. Cloth. $3.95. Pp. 373, with illustrations. Wright & Potter Printing Company, Legislative Printers, 32 Derne St., Boston, 1951.

This book contains reports and discussions that took place at a symposium held Feb. 1 through 3, 1951. It makes available to the student and the clinician interested in hypertension a good summary of the knowledge of the 24 authorities who wrote chapters and of others who participated. Although the epidemiological approach is emphasized, the authors have also covered the problems of etiology, diagnosis, classification, and treatment. The informal questions and answers aid interpretation. The symposium is well worth reading by those interested in this field of study.

STREPTOMYCIN AND DEAFNESS

To the Editor:—*A patient, aged 60, suddenly lost his hearing, while under treatment with streptomycin. What treatment do you suggest to restore his hearing?* *M.D., Michigan.*

Answer.—Neurotoxic effects occurring after the administration of streptomycin or dihydrostreptomycin are not uncommon. Individual sensitivity to both drugs varies greatly. In general, dihydrostreptomycin has a more profound effect on the auditory mechanism than does streptomycin. Streptomycin damages the vestibular apparatus early, while hearing is affected late; in contrast, dihydrostreptomycin causes severe deafness early, while the vestibular function is affected late. In many instances the hearing loss is progressive even after the discontinuance of medication. The prognosis is guarded and distinctly unfavorable in patients having a pronounced hearing loss. There is currently no treatment for this condition.

ELECTRIC SHOCK AND TUBERCULOSIS

To the Editor:—*A man while on a pole came in contact with a 6,600 volt line wire with his left forearm when his right thigh was in contact with the ground wire. He was able to climb down the pole to the ground. He received electrical burns of the left forearm and right thigh. He strained his back in breaking contact with the wire. A routine roentgenogram of the dorsal vertebral column taken the day after injury showed evidence of some lesion in the right upper lung lobe, and roentgenograms of the lung indicated that this lesion was a fibrotic type of tuberculosis. One month later the sputum was found positive for tubercle bacilli. There is a history of blood spitting two and one-half months before injury and of an attack of pneumonia two and one-half years previously. Could the injury described be responsible for activating the tuberculous process present in the lungs?*

Albert H. Winters, M.D., Pittsburgh.

Answer.—High voltage current is destructive of pulmonary tissue in proportion to the amount of current passing through the cells. The higher the voltage, the greater is the destruction. In this case the voltage was high, and no doubt the amperage also was great. It is problematic, however, whether much current went through the lungs at all since a slight contact against the heart would completely disorganize the cardiac rhythm and result in death. Currents of that nature usually zig-zag their way from point of entry through the tissues to the point of exit. The terrific shock and toxicity resulting, however, would in itself be enough to cause an exacerbation of a tuberculous lesion. It seems probable that the patient had latent tuberculosis at the time of the shock, but the shock contributed to or was the direct cause of the exacerbation.

CUSHING'S SYNDROME

To the Editor:—*In* The Journal (148:1166 [March 29] 1952) *an answer is given to a query regarding the treatment of Cushing's disease, which I think is acceptable and logical. With the experience that we have had, however, and the presence of mental disturbance in this patient, I would strongly urge a more radical approach as soon as possible, particularly in view of the fact that the excretion of 17-ketosteroids has attained a level of 29 mg. per 24 hours. In our clinic with proper preparation, I should advise that bilateral adrenal exploration be carried out and, if no tumor is found, one adrenal removed and a very appreciable portion of the second, also.*

The hormone therapy before and after adrenalectomy is of paramount importance. The need for urgency rests on the mental disturbance, which can become permanent if the Cushing's syndrome is not relieved completely in the immediate future. X-ray treatment, in our experience, helps in a very small percentage of cases, and the response, of course, is slow. Without mental symptoms, it is certainly justifiable to proceed serially with the less radical procedures.

George W. Thorn
Peter Bent Brigham Hospital
Boston.

URETHRAL STRICTURE FOLLOWING PROSTATECTOMY

To the Editor:—*I think the strictures Dr. Heins is asking about in the query on urethral stricture following prostatectomy (J. A. M. A., March 29, 1952, page 1167) are the ones that occur mostly in the pendulous urethra. They are probably due to trauma from an electrotome sheath that is inadequately lubricated or to injury to the mucosa produced by tension of the urethra about the sheath, which prevents circulation in the mucosa during the period of the transurethral resection. It is necessary to withdraw the sheath and lubricate it frequently if it tends to be tight in the urethra and does not move back and forth easily. Careful calibration of the urethra should be done before a transurethral resection so as to be sure that there are no places tight enough to fit the electrotome sheath too tightly. If I find a urethra too tight for the no. 28 French sheath, I prefer to use the no. 26 French sheath rather than divulse the constricted area with sounds. The no. 26 French instrument is adequate except for large prostates. These I do not remove transurethrally.*

Montague L. Boyd, M.D.,
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Atlanta, Ga.

IODIDES AND TUBERCULOSIS

To the Editor:—*Concerning the query on iodides and tuberculosis* (J. A. M. A. **147**:1091 [Nov. 10] 1951), *considerable damage may result in allowing the answer to stand as published. First, the question of unsuspected tuberculosis of the lung rests in the efficiency of the examining physician, who should, in the light of current education and knowledge, be able not only to suspect but to recognize tuberculous disease if it is present. It would appear to be negligence not to recognize it. Three to four decades ago, before x-ray films were a common or routine procedure in chest examinations, physicians ceased using potassium iodide in 2-grain dosage to increase the sputum and produce a sputum positive for tubercle bacilli. My book, "Clinical Tuberculosis," contains the following statement:*

"Iodine has been used in the form of its alkaline salts, the sodium or potassium, or as the tincture or Lugol's solution which contains these salts in addition to iodine. The use of the alkaline iodine salts in the exudative or caseous pneumonic lesions causes an increase in the blood supply in and about the site of the tuberculous disease. This increases the secretions in this area and tends to break down and carry necrotic tissue from the area of tuberculous disease into the bronchial tract. It thus allows a 'spilling over' of tuberculous infection into uninvolved tissue. It is this action of the iodide salts which for many years, as a diagnostic procedure, was used to produce sputum containing tubercle bacilli, where a questionable tuberculosis existed. This procedure is to be condemned and never employed. Extension of tuberculous disease and hemoptysis have been encountered in many instances following its use. Employing iodine in tuberculosis for a number of years, [I] have used a solution of iodine, in glycerine and alcohol, as below:

Iodine resublimed	*90.0*
Alcohol 95%	*400.0*
Glycerine	*500.0*

Allow to stand for two weeks

This is without the addition of the iodide salts. No deleterious results have been seen from this solution, even in exudative cases where it has been used to treat, or as a diagnostic aid in associated thyrotoxicosis. Iodine is used in pleurisy, especially with effusion, and in some instances of chronic fibroid or the scirrhous type of pulmonary disease, which frequently have associated asthmatic manifestations. The average dose of 10 minims of the above solution may be given in milk three times daily and increased, if necessary, or if symptoms of iodism appear, to as high as 30 to 60 minims three times daily in a glass of milk, preferably after meals."

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MEDICAL PROBLEMS CREATED BY A NATIONAL BLOOD PROGRAM

CHAIRMAN'S ADDRESS

Carl V. Moore, M.D., St. Louis

The development of effective transfusion services is one of the major medical accomplishments of the past three decades. During this time, methods have been developed for storing blood; techniques have been greatly improved; additional blood groups of clinical importance have been recognized so that determination of compatibility can be more specific; and plasma has been dried and fractionated. These technical and scientific advances made possible the formation and development of blood banks. Physicians were quick to use the blood as it became more readily available, because mortality was thereby lowered both in civilian and in military practice. This experience stimulated an even more rapid growth of organized transfusion services, both in hospitals and on a community basis. There are now several state-wide transfusion programs, and that of the American Red Cross extends throughout many communities of the nation as a whole. A similar expansion has occurred in many other countries.

The extent and effect of this growth have not been generally appreciated. The second survey on blood banks made by the American Medical Association indicated that, in 1950, hospital blood banks procured more than 1,600,000 pints of blood, Red Cross Regional Centers obtained almost 500,000, and nonhospital blood banks collected nearly 400,000 units.[1] In addition, the survey estimated that roughly 500,000 pints of blood were obtained for immediate transfusion without prior storage. The total is about three million units of blood per year for the United States. Even this high figure, however, needs to be revised upward because of the change since the Korean conflict began. From July, 1951, through April, 1952, the American Red Cross obtained 2,986,600 pints for both civilian and military purposes, while independent banks cooperating in the national blood program obtained an additional 453,000 for military purposes.[2] These figures average slightly more than four million units per year. If it is assumed that hospital and nonhospital blood banks have continued their collections unchanged, then the amount of blood that will be obtained from the American people during the year 1951-1952 will be from six to seven million pints.

This is a record of which medicine, the blood banks, the military establishment, and the persons who give their blood can very rightly be proud. With the phenomenal growth of transfusion services, however, have come two problems: 1. The ready availability of blood has led to its increasing use, without adequate realization of the hazards involved, when no clear indication for transfusion exists. 2. The repeated donation of blood, particularly by women, constitutes a severe drain on iron reserves, so that hypochromic anemia more readily develops. Serious attention should be directed to the desirability of providing iron supplements to women donors in amounts sufficient to replace the iron lost.

UNNECESSARY USE OF BLOOD TRANSFUSIONS

Whenever a blood bank is established in a hospital or in a community, the number of transfusions given often increases several fold. The Red Cross has found that, after a regional center is organized, the amount of blood required by the community is always much greater than had originally been estimated by the physicians and hospitals in that community. The experience in the blood bank that serves the Washington University Group of Hospitals is illustrative (fig. 1). The first full year of operation for this bank was in 1943, when 2,700 transfusions were given; currently almost 8,000 transfusions are given per year. Because the number of hospital beds has increased, and the average stay of patients in the hospital has become shorter, the data in fig. 1 have been charted as transfusions per bed and transfusions per 100 hospital admissions. It is evident that the increase is striking in each instance. The number of transfusions per bed has increased nearly 2.5 times, per admission or per number of patient days nearly two times. Many factors

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From the Department of Medicine, Washington University School of Medicine.

The iron tablets distributed to donors were supplied by Burroughs Wellcome & Company, Inc., Tuckahoe, N. Y., and Wyeth, Inc., Philadelphia.

Read before the Section on Experimental Medicine and Therapeutics at the 101st Annual Session of the American Medical Association, Chicago, June 11, 1952.

[1] Dickinson, F. G., and Welker, E. L.: Second Survey of Blood Banks: A Report to the Committee on Blood Banks, Bulletin 83, American Medical Association, Bureau of Medical Economic Research, 1951.

[2] Report, American National Red Cross, April, 1952.

are unquestionably responsible for this rise. Patients who could have profited from blood *probably* were not given transfusions or did not receive as many, when it was less readily available. The character of surgery has changed to include more thoracic and cardiac operations and a more radical approach to the surgical treatment of abdominal carcinoma. In some instances, however, unnecessary transfusions are being given.[3]

It is almost impossible to determine what percentage of transfusions can be classified as unnecessary. There are numerous instances, however, in almost every hospital in which patients are "prepared" for operation by being given one or two transfusions, even though their nutritional status is satisfactory, their red blood cell levels are above 3,500,000 cells, and their hemoglobin values are above 10.5 gm. Similarly a transfusion is often begun immediately prior to what promises to be an uncomplicated surgical procedure, even though the nutritional and hematological status is good. Women after delivery are occasionally given transfusions before their release from a hospital in order to hasten the return of hemoglobin values to normal, even when only very mild degrees of anemia are present. On medical services, transfusions are also given to patients with only slight decreases of red cells, presumably to speed convalescence or in the vague hope that a sense of fatigue will be relieved. Interns and residents are asked to transfuse blood for its "tonic" effect. There is no need to cite specific cases as examples. Almost every active practitioner can supply illustrations from the hospitals of his own affiliation. Objection to my criticisms may be made because of honest difference of opinion as to the indications for transfusion. Physicians who give blood under these circumstances, however, must be cognizant of the risks involved, and must be prepared to justify the risk.

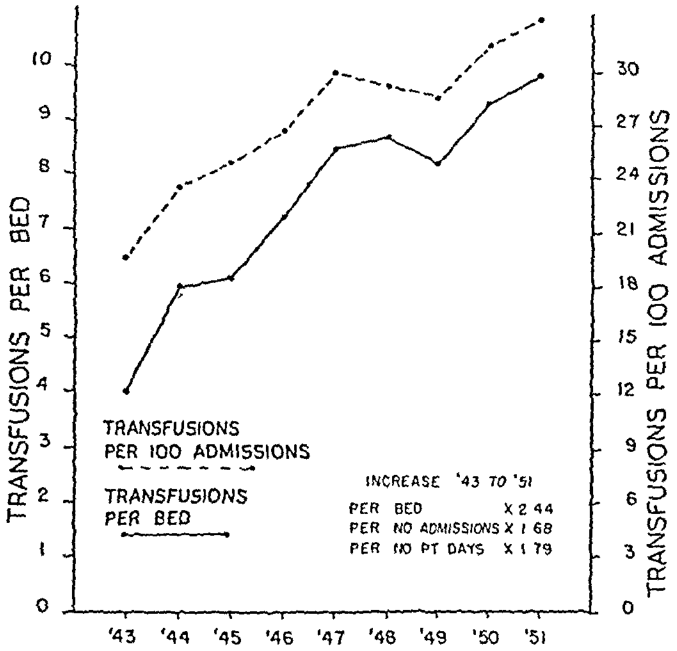

Fig. 1.—Increase in transfusions given in the Washington University Hospitals, including Barnes, St. Louis Maternity, and McMillan Hospitals, after the blood bank was established.

Sufficient emphasis has not been given to the fact that the mortality rate from transfusions probably varies from 1 in 1,000 to about 1 in 3,000.[4] These figures probably represent minimum rates, because they were accumulated in hospitals where one or more physicians have been particularly interested in transfusion services, and have carefully supervised techniques. The reasons for transfusion deaths are many; they include human error, severe hemolytic reactions because the patient has previously been sensitized to one of the rare blood groups, circulatory overload, contamination of the blood, and serum hepatitis. The factor of error is particularly difficult to overcome completely. A blood bank refrigerator, not equipped with a constant recording thermometer, may go off during the night because of power failure. Power is restored, but not until after the blood has become warm. The blood is recooled and the hemolysis that occurs when the red cells are brought to body temperature at the time of transfusion may not be detected unless a sample is centrifuged. Severe hemolytic reactions may therefore result. Not infrequently, patients with very similar names but with different blood types may be in the hospital at the same time; I know of at least one instance when incompatible blood was given because an intern confused the identity of patients under these circumstances. Errors in labeling of bottles or pilot tubes in typing or crossmatching continue to occur. There seems to be an irreducible minimum human error that the most constant vigilance is not able to avoid. Transfusion reactions in the operating room are apt to be particularly severe because the usual early signs are masked by the anesthesia. It is only rarely that one sees a physician or a house officer so aware of the dangers of producing circulatory overload with pulmonary edema that he checks the venous pressure regularly during transfusions to old or severely anemic patients. The reasons for fatal transfusion reactions are too numerous to be discussed in greater detail here; they have been reviewed by numerous authors.[5] The important thing to emphasize is the mortality figure itself. If one accepts the mortality rate of one death in 3,000 transfusions, then for the approximately 3,000,000 transfusions given in this country per year, there must be about 1,000 deaths. The claim has been made that the mortality rate from transfusions approaches that of appendicitis or of ether anesthesia.[3]

Unquestionably, transfusions save many times this number of patients. The risk, when clear indication for transfusion exists, is minimal compared to the risk of not using blood. Some of the deaths, however, certainly occur among patients who do not need transfusions. The best way to avoid such accidents is to discourage the practice of giving blood unnecessarily. A blood transfusion should not be regarded as a tonic.

Of related importance is the fact that the incidence of serum hepatitis from whole blood transfusions is estimated to be from 0.5 to 0.8%.[6] The mortality rate from this cause alone may be as high as 1 for every 6,000 whole blood transfusions.[3] Other factors that make this complication a very serious one are the long illness, the possibility of residual hepatic damage, and the probability of increasing the number of carriers of the serum hepatitis virus.

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[3] Abuse of Transfusion Therapy, editorial, New England J. Med. **245**: 745-746 (Nov. 8) 1951.

[4] Kilduffe, R. A., and DeBakey, M. E.: The Blood Bank and the Technique and Therapeutics of Transfusions, St. Louis, the C. V. Mosby Company, 1942, p. 558. Wiener, A. S.: Blood Groups and Transfusion, ed. 3, Springfield, Ill., Charles C Thomas, Publisher, 1943, pp. 113-114.

[5] DeGowin, E. L.; Hardin, R. C., and Alsever, J. B.: Blood Transfusion, Philadelphia, W. B. Saunders Company, 1949, chap. 12, pp. 266-301. Klendshoj, N. C., and Witebsky, E.: Reactions to Single and Multiple Transfusions: Comparison Between Homologous and Conditioned O Blood Transfusions, Blood **5**: 123-128 (Feb.) 1950. Lange, R. D.: Blood and Blood Derivatives, J. Missouri M. A. **48**: 111-122 (Feb.) 1951. Soutter, L.; Allen, F. H., Jr., and Emerson, C. P., Jr.: Blood Grouping, Blood Banking and Blood Transfusion, New England J. Med. **245**: 410-424 (Sept. 13) 1951. Borden, C. W., and Hall, W. H.: Fatal Transfusion Reactions from Massive Bacterial Contamination of Blood, ibid. **245**: 760-765 (Nov. 15) 1951. Mollison, P. L.: Blood Transfusion in Clinical Medicine, Springfield, Ill., Charles C Thomas, Publisher, 1951, pp. 271-345.

SUPPLYING IRON SUPPLEMENT TO WOMEN DONORS

The second problem focuses attention on the donor rather than the recipient, and is concerned with the advisability of supplying iron supplements to women donors. The effect of blood donations on iron balance becomes evident from the following considerations. Each 500 cc. of blood with a hemoglobin level of 15 gm. per 100 cc. contains approximately 250 mg. of iron. If this loss is spread evenly throughout one whole year, it is equivalent to an average daily loss of 0.68 mg. of iron. With three donations, the average daily loss would be 2.08 mg., and with five donations, 3.4 mg. (fig. 2). These figures may at first not seem very high, but recent studies on the absorption of food iron have indicated that probably not more than 10%, and certainly less than 20%, of food iron is absorbed.[7] The amount of food iron that would have to be ingested per day in order to compensate for the loss in donated blood, then, even if it is assumed that absorption is 20% complete, would have to be 3.4 mg., 10.2 mg., and 17 mg. for one, three, and five donations, respectively. These requirements, furthermore, must be added to those normally needed to maintain iron balance. There is reason to believe that a dietary intake of 12 mg. of food iron per day maintains the normal woman during her sexually active life in iron equilibrium, but provides only a small margin of safety.[7] Women, therefore, would need 12 mg. of food iron per day plus the additional requirement produced by blood donation.

That many women find it difficult to meet these added demands is indicated, first, by the fact that slightly more than 12% must be refused when they report the first time to give blood because their hemoglobin level is below the minimal standard of 12.3 to 12.5 gm.[8]; and, second, by the fact that the rejection rate because of low hemoglobin values rises with repeated donations. There admittedly are a few women, who, for some unknown reason, are able to give blood regularly and repeatedly without difficulty, but they certainly are the exceptions. The desirability of replacing the iron lost in blood donations by giving iron supplements to women donors was recognized during World War II. Four of the Red Cross centers, those in New York City, Detroit, St. Louis, and Columbus, provided iron supplements to women donors during the last year of the plasma procurement program. The effect of this replacement on the hemoglobin levels of these women has never been analyzed in detail, but there was a definite impression that rejection rates among women giving repeated donations became less. About nine months ago, a similar program was begun at the Red Cross Regional Centers in Columbus and in St. Louis. The medical societies in these two cities approved the plan to supply women donors with iron tablets, since replacement rather than therapy was involved. Iron is never given to a donor until after her hemoglobin level has been found to be acceptable and she has donated blood. It is important to emphasize that, if her hemoglobin value is low, she is referred to her physician for diagnosis and therapy. The donors themselves have accepted the program well, have not been frightened by it, and have reported very few gastrointestinal symptoms from the iron.

NUMBER OF DONATIONS	Fe LOSS AV./DAY	INCREASE IN FOOD Fe REQUIREMENT	
		IF 10% FOOD Fe ABSORBED	IF 20% FOOD Fe ABSORBED
1 PER YEAR	.68 mg.	6.8 mg.	3.4 mg
3 PER YEAR	2.04 mg.	20.4 mg	10.2 mg
5 PER YEAR	3.40 mg.	34 0 mg	17.0 mg.

Fig. 2.—The effect of donation of blood on iron loss and requirement in women. If the hemoglobin level is 15 gm. per 100 cc., 500 cc. of blood contains approximately 250 mg. of iron.

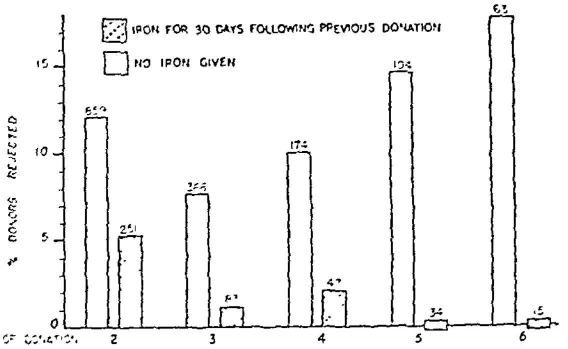

Fig. 3.—Chart showing the effect of iron replacement on rejection rate due to low hemoglobin values in 1,110 women. The number at the top of each column represents the total number of donors on which the observations were made, i. e., of 859 women donors who had not taken iron and who reported for a second donation, 12% were rejected because of low hemoglobin. The dotted columns for donations 5 and 6 are charted as far below as above the base line to indicate that there were no rejections in this small group of women

Each woman donor is given an envelope containing 100 tablets, each of which contains approximately 0.3 gm. of a simple ferrous salt. The following statement is printed on the envelope:

> A normal healthy adult can donate a pint of blood with the assurance that his health will not be injured. This involves, however, the replacement of (1) fluids, (2) proteins, and (3) iron, which is used to regenerate the hemoglobin. The average diet is usually quite adequate to maintain balance in these three relationships. An additional supplement of iron, however, will frequently accelerate the replacement of hemoglobin and red cells.
>
> For those donors who care to act upon this information, inorganic iron in amounts calculated to replace that in donated red cells is available in this sample.
>
> Take one tablet before breakfast the first day, one before each of two meals the second day, and one before each meal daily thereafter.

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6. Neefe, J. R.: Viral Hepatitis: Problems and Progress, Ann. Int. Med. **31**: 857-870 (Nov.) 1949. Lehane, D.; Kwantes, C. M. S.; Upward, M. G., and Thomson, D. R.: Homologous Serum Jaundice, Brit. M. J. **2**: *572-574 (Sept. 10) 1949.*

7. Moore, C. V., and Dubach, R.: Observations on the Absorption of Iron from Foods Tagged with Radioiron, Tr. A. Am. Physicians **64**: 245-256, 1951.

8. Hervey, G. W.; McIntire, R. T., and Watson, V.: Low Hemoglobin Levels in Women as Revealed by Blood Donor Records, J. A. M. A. **149**: 1127-1128 (July 19) 1952.

Note: Keep these tablets in a safe place where small children may not mistake them for candy.

The caution about keeping the tablets out of the reach of small children was added because severe toxic reactions have occurred in children who have taken large amounts of iron.[9] It is difficult to obtain exact information about the effectiveness of the supplements, since only a small percentage of Red Cross donors report regularly for repeat donations. The effect on those who fail to return is unknown. Data analysed by Dr. Claire Ramsey for the St. Louis Regional Red Cross Center indicate that the refusal rate among those who do return is dramatically decreased when iron is taken. This information is charted in fig. 3. Except for the unexplained decrease in the rejection rate of third time donors, the percentage of women whose hemoglobin level was below 12.4 gm. increased steadily as the number of donations increased. When iron supplements were provided, however, this trend was reversed, and the rejection rate was strikingly reduced. The results are preliminary but show exactly what one would expect. By administering iron one prevents the development of iron deficiency anemia and thereby very effectively performs a service of preventive medicine. At the same time a continuing supply of acceptable donors is assured.

SUMMARY

Medicine has every right to be proud of the excellent transfusion services that have been developed in this country and in many areas of the world. From research now in progress there is reason to believe that transfusion programs will continue to grow and to become more effective. The greater use and availability of blood have unquestionably led to very significant decreases in mortality rates, in both military and civilian medicine. With this greater use and availability of blood have come two new problems: 1. Transfusions are given when there is no clear indication for their use. However, the risk of serious, even fatal, transfusion reactions is great enough to contraindicate this practice, and it should be vigorously discouraged. 2. Blood donation is a severe drain on the iron reserves of women donors. Iron supplements should be given in amounts sufficient to replace the loss.

600 Kingshighway Blvd.

[9] Iron Poisoning, editorial, J. Pediat. **40**: 141-142 (Jan.) 1952.

ENZYME DEBRIDEMENT OF INDOLENT INFECTED CUTANEOUS ULCERS

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and
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Within the past few years there have been many reports [1] on the enzymatic débridement of fibrinopurulent exudates in empyema, hemothorax, miscellaneous chronic ulcerative lesions, and local infections, such as osteomyelitis and sinusitis. The present study was undertaken to evaluate the efficacy of the lysing enzymes, trypsin, streptodornase, and streptokinase, in the treatment of various indolent infected cutaneous ulcerations. The majority of the ulcerations treated with these substances were of the varicose stasis type, although decubitus, traumatic, arteriosclerotic, and other types of cutaneous ulcers were included in this series.

Streptokinase and streptodornase ("varidase") are used together, although their physiological action is somewhat different. Purified crystalline trypsin (tryptar®) was sometimes used on a patient who was also given streptokinase-streptodornase or on alternating patients. Tillett and his associates [2] showed that hemolytic streptococci produce a fibrin-lysing substance known as streptokinase that acts as a catalytic agent. The fibrin-lysing system is activated by streptokinase, which causes liquefaction of fibrin. Streptokinase also inhibits the change of fibrinogen to fibrin. This product is abundantly excreted into the culture medium and is readily obtainable from broth cultures of hemolytic streptococci in bacteria-free filtrates. Streptokinase has maximal activity at pH 7.3 to 7.6; at pH 5.0, it is inactivated, and at pH 9.0 or higher it is irreversibly inactivated.

These investigators also demonstrated that the bacteria secrete or excrete a second enzyme, streptodornase. This substance acts on desoxyribose nucleoprotein, which is found in purulent and pleural exudates; streptodornase with fibrin accounts for the coagulum and coarse sediment of such material. Streptodornase (desoxyribonuclease) can catalyze the breakdown of desoxyribonucleoprotein and desoxyribonucleic acid. Miller [3] found that it acts only on the extracellular nucleoprotein or on nuclei in degenerating cells but does not attack the nuclei or nucleoprotein of living cells. The action of streptodornase is one of depolymerization by which the long-chained chemical structure of the nucleoprotein is severed at many points. Coincident with this molecular rupture, the inspissated quality of the purulent material is transformed into a thin solution. Streptokinase and

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Director of the Department of Dermatology and Syphilology, Ancker Hospital (Dr. Madden) and Director of the Division of Dermatology and Syphilology, University of Minnesota (Dr. Michelson).

[1] Tillet, W. S., and others: Streptococcal Enzymatic Debridement, Ann. Surg. **131**: 12 (Jan.) 1950. Adie, G. C., and Childress, W. G.: Experiences with Streptokinase and Streptodornase, ibid. **134**: 659 (Oct.) 1951. Miller, J. M.; Ginsberg, M.; Lipin, R. J., and Long, P. H.: Streptokinase and Streptodornase in Treatment of Pilonidal Cysts, U. S. Armed Forces M. J. **2**: 1423 (Oct.) 1951. Morrel, C., and Weinberg, J. A.: Streptokinase and Streptodornase in Suppurative Lesions, West. J. Surg. **59**: 524 (Oct.) 1951. Tillett, W. S.; Sherry, S., and Read, C. T.: The Use of Streptokinase-Streptodornase in the Treatment of Postpneumonic Empyema, J. Thoracic Surg. **21**: 275 (March) 1951. Reiser, H. G.; Patton, R., and Roettig, L. C.: Tryptic Debridement of Necrotic Tissue, A. M. A. Arch. Surg. **63**: 568 (Oct.) 1951. Wright, L. T., and others: Use of Streptokinase-Dornase in Certain Surgical Conditions, J. Internat. Coll. Surgeons **15**: 286 (March) 1951. McVay, L. V., Jr., and Sprunt, D. H.: Streptokinase and Streptodornase in the Treatment of Diabetic Gangrene, A. M. A. Arch. Int. Med. **87**: 551 (April) 1951.

[2] Tillet, W. S.; Sherry, S., and Christiansen, L. R.: Streptococcal Desoxyribonuclease: Significance in Lysis of Purulent Exudates and Production by Strains of Hemolytic Streptococci, Proc. Soc. Exper. Biol. & Med. **68**: 184 (May) 1948.

[3] Miller, J. M.; Ginsberg, M.; Lipin, R. J., and Long, P. H.: Clinical Experience with Streptokinase and Streptodornase, J. A. M. A. **145**: 620 (March) 1951.

streptodornase cannot digest mucin, collagen, or fibrous tissue, and the presence of large amounts of such substances will seriously interfere with effective enzymatic débridement of fibrinous and purulent exudates. In many cases a combination of enzymatic and mechanical débridement is of value.

Purified crystalline trypsin is a proteolytic enzyme derived from mammalian pancreas. It digests fibrin clots and dead tissue. The products of trypsin action are peptides and some individual amino acids. Trypsin digests the slimy and viscid coating of desoxyribose nucleoprotein of the purulent exudate and then attacks dead tissue in the lesion. The exudate becomes thin and serous, and fresh viable leukocytes appear that help increase the phagocytosis of bacteria. Bacteria rapidly decrease in number and may disappear completely. Trypsin is not bactericidal, and the disappearance of bacteria seems to be caused by a strengthening of the humoral elements of defense and a clearing of debris. By clearing the wound of debris and slough, trypsin denies a medium to the bacteria. Trypsin activity is optimal within a pH range of 6.8 to 7.5. Roettig[4] and his associates stated that it will lyse only dead tissue and will not attack a living cell. On surface lesions the pH of the surface should be tested, and if it is extremely alkaline or moderately acid, trypsin in a solution of Sorensen's reagent should be used as a wet dressing. At temperatures lower than body temperature trypsin is rapidly inactivated; therefore, external heat should be applied to the wet dressing. When allowed to stand in solution at room temperature, trypsin is said to lose 75% of its activity in three hours, and, therefore, continuous effectiveness should not be expected unless the dressings are changed every three or four hours. The activity of trypsin may be stopped by irrigating the treated area with sodium bicarbonate solution.

Twenty-seven patients with cutaneous ulcers were treated in this series. Sixteen of the group had varicose stasis ulcers, four traumatic ulcers, and four decubitus ulcers. One patient was hypertensive; one had a chemical burn; and one had dry gangrene of the toes. The object of the treatment with the enzymes was to remove the adherent fibrinopurulent exudate from the ulcer surface initially in order to proceed with further healing therapies. After a few trial cases it was felt that it would be best to discontinue the enzymatic treatment as soon as the ulcer surface was clean. Continuation of the medication beyond this stage did not enhance the complete healing of the ulcers.

Both streptokinase-streptodornase and purified crystalline trypsin were used in the forms of wet compresses and powders applied to the ulcers. Streptokinase-streptodornase was used in ointment form on three patients. As is shown in the accompanying table the majority of patients were treated with enzymatic wet compresses. When used as a wet compress, 100,000 units of streptokinase and 25,000 units of streptodornase were dissolved in 20 cc. of isotonic sodium chloride solution. This wet pack was applied directly to the ulcer. Moisture was maintained by applying a large isotonic sodium chloride solution compress over the enzyme-medicated pack, which was changed every 12 hours. In three cases streptokinase-streptodornase was used in a water soluble jelly (lubafax®) adjusted to a pH of 7.5. This ointment was used exclusively in two of the patients and alternately with the wet compresses in the other patient. In four other patients, streptokinase-streptodornase powder was sprinkled on the ulcer. The powder was applied to the ulcer every six hours, and an isotonic sodium chloride solution pack was applied over the powder. The powder and the wet pack forms of treatment seemed to be equally effective, although the powder form of treatment was simpler.

Purified crystalline trypsin was also used by direct application of the dry powder to the surface of the ulcer and also as a wet dressing and by infiltration of purified crystalline trypsin solution made by dissolving 250 mg. in 25 cc. of Sorensen's reagent. Purified crystalline trypsin solution had to be freshly prepared every three hours because of loss of potency. Antibiotic therapy, either locally or systemically, was utilized in several patients to prevent spread of infection. In deep decubitus ulcers in which there was a firmly adherent eschar, the débridement was aided by surgical and mechanical removal.

The stasis and traumatic ulcerations of the legs responded most rapidly and dramatically to enzymatic débridement of the purulent exudate on the surface. The results were obtained in 2 to 14 days, the average time for treatment being 6.5 days. Several ulcerations had resisted treatment with many other medications, including wet compresses and antibiotics, and in these cases the responses were significantly impressive.

The decubitus ulcerations were the slowest to respond, and all required the aid of surgical débridement, but at the end of the treatment period the ulcerations were clean, with healthy red granulations in evidence. One patient (case 24), who had extensive and multiple decubitus ulcers, failed to respond to treatment, but this patient had been chronically ill and severely debilitated. She required supplemental feedings and blood transfusions. In another patient (case 7), who had a long-standing history of recurrent stasis ulcers on the lower legs and feet, a surrounding cellulitis developed following treatment with streptokinase-streptodornase packs. Later, purified crystalline trypsin powder was used but only intermittently as it caused pain. The purulent exudate cleared after 10 days' treatment. The patient (case 26) with arteriosclerotic dry gangrene of the toes showed no change following treatment with streptokinase-streptodornase wet compresses for 10 days and streptokinase-streptodornase in ointment form for 5 days.

In one patient (case 10) with large stasis ulcerations on both lower legs we simultaneously compared the two enzymes. After five days the leg ulcerations treated with purified crystalline trypsin seemed much cleaner than those on the opposite leg, which were treated with streptokinase-streptodornase. The powder form of the two enzymes was used with the application of isotonic sodium chloride solution compresses.

Bacteriological studies made on this group of patients revealed only the usual variety of bacterial flora found on cutaneous lesions. No adverse reactions from treatment were noted other than pain on application of the purified crystalline trypsin powder in a few patients. In one patient, a mild cellulitis developed during treatment with streptokinase-streptodornase (see table on next page).

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[4] Roettig, L. C.; Reiser, H. G.; Habeeb, W., and Mark, L.: The Use of Trypsin in Chest Disease, Dis. Chest 21: 245 (March) 1952.

Results of Treatment with "Varidase" and Tryptar®

Case	Sex	Age	Type of Ulcer	Location and Size	Medication	Days of Treatment	Bacteriological Study	Results
1	F	56	Stasis	Left leg, outer, lower, 3 × 5 cm.	Tryptar® powder	5	Not done	Ulcer cleaned rapidly
2	F	76	Stasis	Right ankle	"Varidase" ointment	7	Not done	Cleaned in 7 days
3	M	68	Stasis	Left outer ankle	"Varidase" packs	4	Not done	Cleaned rapidly
4	F	75	Stasis	Left inner ankle	"Varidase" packs	2	Not done	Cleaned dramatically, "Varidase" continued 14 days, ulcer healed
5	F	79	Stasis	Both ankles, 3 × 7 cm.	"Varidase" packs	14	Staphylococcus Streptococcus Gram-negative rods	Cleaned but became painful until frequency of applications reduced
6	M	68	Stasis	Left lower leg, 2 cm.	"Varidase" packs	8	Not done	Cleared slowly
7	M	64	Stasis	Dorsum of right foot 5 × 7 cm., left leg, medial tibial surface, 4 × 6 cm.	"Varidase" powder Tryptar® powder Tryptar® packs	6 7 3	Hemolytic Staphylococcus (coagulase positive)	"Varidase" caused cellulitis, tryptar® cleaned ulcers somewhat, terramycin also used
8	M	77	Stasis	Left leg, anterior and medial aspects, 8 × 13 cm.	"Varidase" packs	3	Hemolytic Staphylococcus (coagulase negative) Coliform bacillus	Cleaned up rapidly, recurrence 3 months later with same results
9	M	32	Stasis	Left leg, anterior	"Varidase" packs	5	Hemolytic Staphylococcus (coagulase positive)	Cleared rapidly
10	M	61	Stasis	Lower legs, medial	"Varidase" powder, left leg Tryptar® powder, right leg	5	Negative	Tryptar® treated leg cleaner at end of 5 days
11	M	70	Stasis	Left lower leg, anterior	"Varidase" packs	5	Hemolytic Staphylococcus (coagulase negative)	Cleared rapidly
12	F	81	Stasis	Right lower leg, medial, 5 × 5 cm.	"Varidase" packs and (11 days) powder on recurrence (4 days)	15	Hemolytic Staphylococcus (coagulase negative)	Cleared rapidly on recurrence
13	F	57	Stasis	Malleolus on right ankle, medial, 7.5 cm. diameter	Tryptar® powder	5	Not done	Ulcer cleared rapidly
14	F	67	Stasis	Left ankle, posterior	Tryptar® packs "Varidase" packs	7 4	Not done	No response to either tryptar® or "varidase"
15	F	69	Stasis	Right leg, posterior	Tryptar® packs	4	Not done	Cleared rapidly
16	F	65	Stasis	Lower right leg	Tryptar® packs	7	Not done	Cleared
17	F	65	Traumatic	Upper calf of left leg, posterior 5 × 5 cm.	Tryptar® powder	7	Negative	Cleaned rapidly
18	F	50	Traumatic	Lower right leg, anterior, 1 cm.	"Varidase" ointment	14	Negative	Cleared, erythema developed around ulcer
19	F	58	Traumatic	Lower left leg, anterior, 5 × 12 cm.	Tryptar® powder and packs	6	Gram-positive bacilli and cocci	Cleared (patient diabetic)
20	F	39	Traumatic	Lower left leg, anterolateral	"Varidase" packs	3	Hemolytic Staphylococcus (coagulase positive)	Cleared rapidly
21	M	46	Hypertensive	Left leg, medial, malleolus, 4 cm.	"Varidase" packs	21	Coliform bacillus	Cleaned up slowly
22	F	79	Decubitus	Sacrum, 15 × 15 cm.	Tryptar® instillations	24	Not done	Cleaned up with aid of surgical débridement (patient diabetic)
23	F	60	Decubitus	Sacrum, 5 × 7 cm.	Tryptar® powder and infiltrations	17	Not done	Cleaned up slowly, surgical débridement necessary
24	F	71	Decubitus	Feet, ankles, and buttocks	"Varidase" packs (10 days) "Varidase" powder (17 days)	27	Gram-negative bacillus of coli-aerogenes group	Cleaned up slowly, surgical débridement necessary, patient severely debilitated
25	M	35	Decubitus	Sacrum, right buttock, 18 cm. diameter	Tryptar® infiltration and powder	14	Not done	Cleaned up slowly
26	M	68	Dry gangrene	Toes, left foot, 3d, 4th, and 5th; right foot, 5th	"Varidase" packs (10 days) Ointment (5 days)	15	Not done	No change after 3 weeks
27	M	28	Chemical burn	Lower leg, 4 × 4 cm.	Tryptar® packs	10	Not done	Cleaned up and healed, slough separated in 5 days

CONCLUSIONS

In this series of patients treated with the enzymes streptokinase and streptodornase ("varidase") and purified crystalline trypsin (tryptar®) favorable results were obtained in 23 of the 27 cases. Eleven patients treated with purified crystalline trypsin responded favorably and two unfavorably. Thirteen patients treated with streptokinase-streptodornase responded favorably and four patients unfavorably. It must be emphasized that the specific action of the enzymes is directed against the necrotic fibrinopurulent exudate seen on the surface of the ulcerations. By their catalytic and enzymatic properties it is now possible to bring about lysis and digestion of fibrin and desoxyribose nucleoprotein when these enzymes are brought into contact with fibrin, pus, and dead tissue. Thus, not only is the pus and debris eliminated, but the formation of healthy granulation tissue is facilitated. It is felt that streptokinase-streptodornase and purified crystalline trypsin should be considered as an adjunct in the treatment of indolent infected cutaneous ulcers. These applications are a valuable aid in removing debris from necrotic lesions that require further competent surgical and medical management.

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VIRAL AND BACILLARY DYSENTERY

A DUAL EPIDEMIC

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Mild, infectious, gastrointestinal disorders, according to many observers, are more numerous and widespread than any other enteric disease, especially in late summer and autumn. Epidemics of nausea, vomiting, and diarrhea, each of several weeks' duration among medical students, nurses, and hospital personnel were studied in 1943 and 1944.[1] The distribution and clinical behavior of the disease suggested filtrable agents to be the cause,[2] and a similar disease was transmitted to volunteers by inhaled [1b] and ingested [3] filtrates of stools obtained from patients. There appear to be at least two similar etiological entities, one febrile and the other not.[3a] Many subsequent outbreaks were reported, with a probable viral cause in mind.[4]

Several inadequate descriptive terms are given to the disease as can be noted in the appended references. The term viral dysentery was proposed to imply bowel trouble caused by one or more filtrable agents. Adoption of the term would favor an orderly nosologic separation of enteric infections caused by viral, bacterial, fungal, or protozoal agents in keeping with parallel etiological classification of other system diseases.

Viral dysentery is in certain respects like the common cold. It is seasonal, contagious, sporadic, and occurs in epidemics. As a rule, it is mild, brief, afebrile, and bedrest is seldom necessary. It seems to be spread by contact or by air-borne particles, not by food or water, and bacteria are not implicated; however, overt signs and symptoms are usually absent. Most victims do not mention diarrhea willingly, and medical aid is seldom sought. Thus, epidemics may be unnoticed, except in closely observed groups or unless special effort is made to detect them.

In mid-September, 1951, a nurse was admitted to the Jefferson Hospital six hours after the onset of severe headache, anorexia, nausea, fever, colic, and diarrhea. The next day, 4 nurses with similar complaints sought treatment, and, on the third day, 7 more; in a week, there were 27 patients. In contrast to viral dysentery, in which patients are seldom sick enough to need special care, the large number of relatively severe cases occurring over several days, limited to nurses, suggested some other cause and led to investigation of the epidemic.

EPIDEMIOLOGY

Inquiry was made on Sept. 20, in the classroom of 320 medical students who lived and ate in various places. About 20% had or had had mild, brief, diarrheal disease over the same period. All were ambulatory. Several students and nurses reported similar disease in members of their families who lived at a distance. For example, a nurse visited her rural home on Sept. 14. On Sept. 16, she awakened with colic and diarrhea and later vomited. On Sept. 17, she returned to the hospital. She was ill en route and was admitted to the hospital with a temperature of 102 F and rhinorrhea. On Sept. 18, at her home,

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Dr. John R. Griffith of the Jefferson Medical College and Hospital, Philadelphia, aided in the clinical and epidemiological studies.

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her mother became ill with rhinorrhea and complained of aches followed by colic, nausea, and diarrhea for three days. On Sept. 21, her father became similarly ill. In another instance, a student who lived at home had diarrhea, sore throat, and chills for five days. His wife had sore throat the next day followed by three days of abdominalgia, anorexia, and diarrhea. Small epidemics were current in other hospitals in the city and its environs and in the practices of interrogated physicians, especially among children. The diverse, widely-scattered groups involved ruled out a common food or water supply as the source of infection. No obvious epidemics of minor disease of the respiratory tract were present.

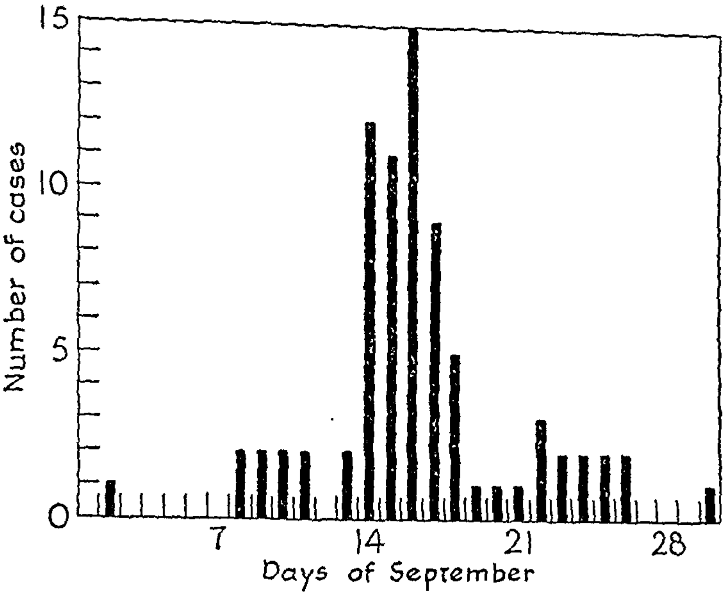

Chart 1.—Days of onset of disease in 78 patients with acute enteritis. All victims were ambulatory except between Sept. 13 and 19, when 27 patients with severer cases were admitted to the hospital; Shigella sonnei was isolated from 12.

On inquiry, it was learned that the first case in the group studied occurred on Sept. 2. Between Sept. 7 and 13, two new ambulatory cases occurred daily, until the admission of 27 nurses to the hospital between Sept. 13 and 19, aroused attention. The date of onset of illness in 79 patients during September is shown in chart 1. Thereafter, between one and three patients with mild cases appeared daily until Sept. 26, and sporadic cases occurred in October. Seventy-five patients were observed, including nurses, students, and other personnel. Many cases, no doubt, were undetected. Except for the "peaking" in the curve of incidence caused by the sudden increment of relatively severe cases, the course of the epidemic resembled the ones referred to previously.[1a]

Of 211 nurses, 54, or 25%, were ill. Half of this group needed care in bed, and they and several other hospital personnel who dined in the same room accounted for the peak of the curve between Sept. 13 and 19. This large proportion of incapacitated patients is not characteristic of viral dysentery, with which only the exceptional victim seeks bedrest. Evidently, the outbreak among nurses was caused by a severe form of viral dysentery, by another enteric infection, or by a combination of the two.

Specimens of stools of 27 nurses confined to bed were cultured routinely in the hospital laboratory, but only colon bacilli, staphylococci, and streptococci were found. On special study, shigellae were isolated from the stools of 12 of 17 patients who took sick between Sept. 14 and 17. After this discovery, it was obvious that the sharply limited outbreak was one of bacillary dysentery. It was unlikely, however, that Shigella colitis accounted for the many mild cases in our group and scattered throughout the city and its suburbs. Cultures of stools, made with an improved technique in the hospital laboratory, of all succeeding patients until Sept. 26, failed to reveal additional cases of Shigella infection. Shigellae were not recovered from stool samples obtained by rectal swabs from 50 nurses who had not been ill or from 146 employees who handled food in the hospital. It seemed, therefore, that a dual epidemic had occurred, composed of widespread viral dysentery with a slowly rising incidence and decline over a month or more and a superimposed sharply defined outbreak of bacillary dysentery lasting about a week and limited to nurses (chart 1).

CULTURAL STUDIES

Stool specimens from 17 patients were cultured by Dr. Kenneth Goodner, who isolated Shigella from 7. Subcultures were sent for identification to the Dysentery Registry in New York City under the direction of Dr. Joseph Felsen and Dr. A. J. Weil. The seven strains acidified a sucrose and lactose medium in a week and were classified as S. sonnei. They grew richly on all mediums, and five decomposed urea in three to five days.

Attempts to transmit viral dysentery experimentally to volunteer students by the inhalation or ingestion of fecal filtrates as described in previous experiments [1b] were not made because of the now known hazard of poliomyelitis, viral hepatitis, or Coxsackie disease. Instead, samples of feces, preserved frozen in carbon dioxide, from the same 17 patients were tested by Dr. Joseph Smadel and Dr. Ross L. Gauld at the Army Medical Center. Intraperitoneal or intracerebral injection of 10% suspensions of the supernatant fluid of centrifugated stools failed to cause significant disease in guinea pigs or in suckling and adult mice. Culture of the stools on McConkey's medium revealed S. sonnei S and R from 11 of the 17 samples. Because of the apparent dual nature of the epidemic, studies were made separately of patients from whom

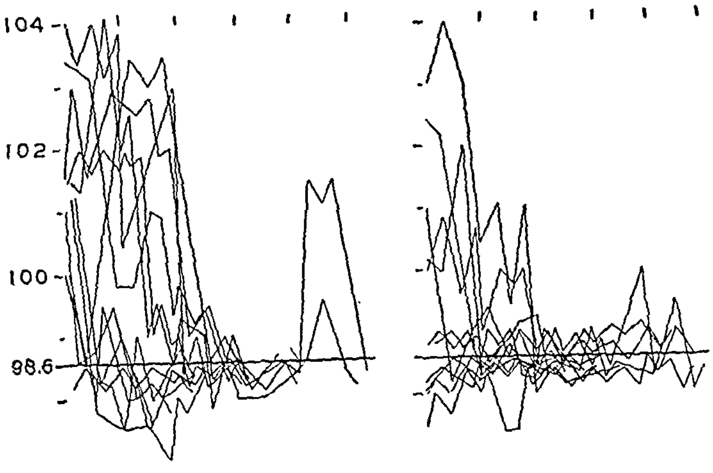

Chart 2.—Superimposed temperature curves of 22 patients ill between Sept. 13 and 19. Left, curves of patients with bacillary dysentery. Right, curves of patients from whose stools Shigella was not isolated.

Shigella was obtained to see whether clinical differences from viral dysentery other than degree of severity could be detected.

BACILLARY DYSENTERY

The disease began between Sept. 14 and 17, in 12 patients from whom S. sonnei was cultured. The initial symptoms were colic, and/or diarrhea in seven, headache in two, nausea in one, vomiting in one, and malaise and a chill in one. During the course of the disease, all patients had diarrhea; colic occurred in 10, nausea in 8, and vomiting in 8. Two had a shaking chill, and six experienced chilliness or chills. A temperature of 100 F or over occurred at the onset or during the course of the disease in 10 patients and lasted two or three days (chart 2). Two had a recurrence. Leukocytosis of 18,000 occurred in only one person. There were from 4 to 20 watery bowel movements a day. Mucus and pus were not observed, but in only one or two patients streaks of blood were present at times. Leukocyte counts roughly estimated in smears of stools stained by Wright's method were between 0 and 70 cells per oil-immersion field. Four patients had rhinitis or pharyngitis. The duration of symptoms averaged four days; in two patients, diarrhea lasted 9 days and in one, 14 days. Nine patients were classified as "severely" ill for a day or two, and three had mild attacks. The word severe is used only in relation to the majority of cases of mild disease in ambulatory patients. Severe bacillary dysentery as described in textbooks, with prolonged fever, prostration, blood, pus, and mucus in the stools, and sequelae did not occur. A composite record of the fever curves of 11 patients is illustrated in chart 2. Streptomycin, aureomycin, chloramphenicol, or terramycin were given to one or two patients each. The short duration of the acute stage in all patients did not allow critical evaluation of antibiotic therapy. Several cases are cited as examples of the degrees of severity encountered.

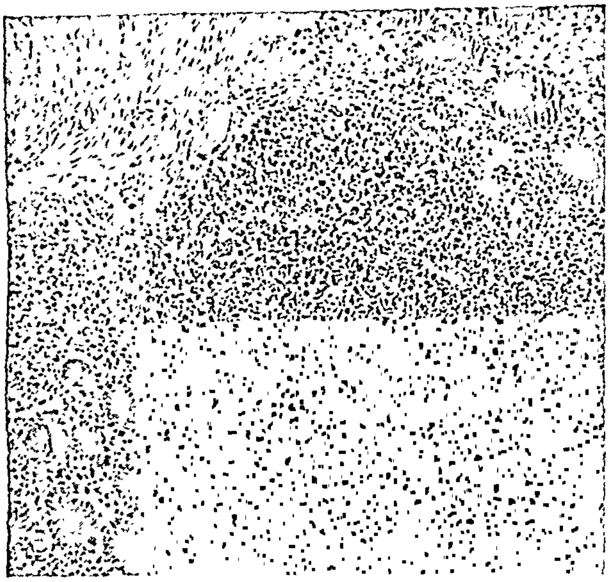

Fig. 1 (patient B. D.).—Mild inflammatory reaction, with edema, congestion, superficial focal areas of hemorrhage, and infiltration of plasma cells and polymorphonuclear cells of the rectal mucosa in a patient with bacillary dysentery. The epithelium was eroded in a few areas. Some of the crypts contained leukocytes. A hyperplastic lymph node in the mucosa is shown (× 88.55).

REPORT OF CASES

Severe Disease.—B. D. noticed nasal obstruction, rhinorrhea, sore throat, and cough between Sept. 9 and 17. On Sept. 15, she had sudden colic, nausea, and six watery stools. The next day she vomited, felt chilly, and had diarrhea, but she continued to work. She became so ill that she entered the hospital on Sept. 17, with a temperature of 100 F. She appeared ill, had nasal obstruction, rhinorrhea, and an injected edematous pharynx. The leukocyte count was 7,000. The number of leukocytes in a sample of stool was estimated as 70 per microscopic field. During the next three days, her fever rose to 103 F. Seven to 13 watery stools a day required two intravenous infusions of 1,000 cc. of isotonic solution of sodium chloride and 5% glucose for rehydration. The leukocyte count was 12,000. On the fifth day, her temperature again rose to 102 F, and rehydration was necessary. Terramycin was given between Sept. 21 and 25, without obvious effect; diarrhea persisted for 14 days.

Sigmoidoscopy performed on Sept. 19, revealed perianal inflammation and moderate inflammation up to the limit of vision, 17 cm. The mucosa was dusky and erythematous, with moderate edema. There were numerous small petechiae but no ulcers. Biopsy revealed inflamed rectal mucosa (fig. 1).

A. S. had a headache on Sept. 14; this was soon followed by three diarrheal stools and colic, nausea, and vomiting. On admission to the hospital on Sept. 15, she had a temperature of 101.5 F that rose to 104 F but lasted only a day; her pulse rate was 120 to 140 per minute. Diarrhea persisted; she vomited four times and was dehydrated. The leukocyte count was 7,000. About 60 leukocytes per microscopic field were present in a sample of stool. An intravenous infusion of 1,000 cc. was given. On Sept. 16, her temperature became almost normal, but dehydration from diarrhea again required intravenous infusion. Recovery occurred after nine days. Sigmoidoscopy on Sept. 20 revealed proctitis with diffuse granularity, erythema, and many submucosal hemorrhages. The mucosa bled easily. Biopsy showed moderately severe inflammation (fig. 2).

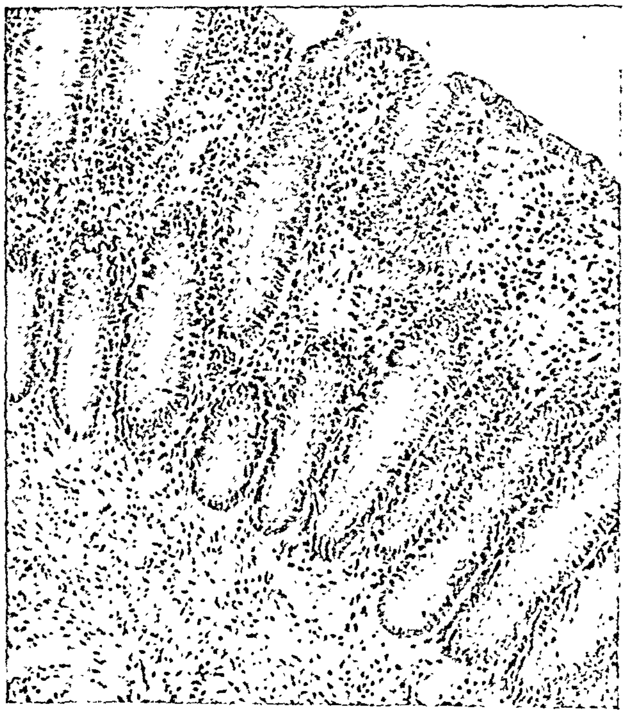

Fig. 2 (patient A. S.).—Moderate inflammatory response of the rectal mucosa as seen in a patient with bacillary dysentery. The supportive tissue is edematous and shows punctate hemorrhages, polymorphonuclear leukocytic invasion, engorged capillaries, and an occasional hyperplastic lymph follicle In many areas the surface was denuded of epithelium (× 100).

Mild Disease.—E. S. had sudden abdominal pain, colic, and diarrhea on Sept. 16. She suffered from nausea and had four watery stools during the night. She was admitted to the hospital on Sept. 17, with a temperature of 101 F and a pulse rate of 120 per minute. She vomited once. Her abdomen was tender, and peristalsis was audible. She had seven watery stools on Sept. 18, and two on Sept. 19. About five leukocytes were seen in a microscopic field of stained stool. The illness lasted three days.

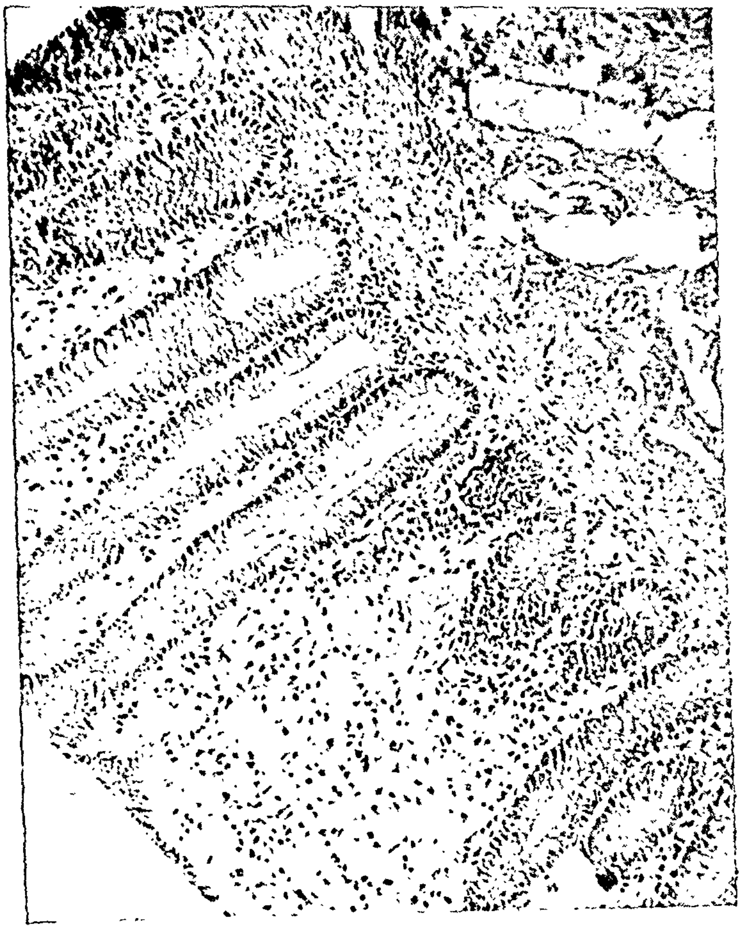

Fig. 3 (patient I. I.).—Shigella was not found in this patient with dysentery that was probably viral in origin. A mild, inflammatory reaction of the rectal mucosa with much edema, engorgement of vessels, punctate hemorrhages, and a diffuse infiltration of plasma cells and polymorphonuclear cells can be seen (× 100).

VIRAL DYSENTERY

The onset of disease in 11 patients from whose stools Shigella was not cultivated occurred between Sept. 14 and 20. For comparison with those of proved bacillary dysentery they are grouped tentatively as cases of viral dysentery (chart 2). Shigella, of course, may have been operative but undetected in some. Variation in grades of severity characterize both diseases and renders clinical diagnosis impossible in individual cases. The clinical characteristics of the cases grouped here were similar to those described for the severe form of viral dysentery [1a]; the temperature did not exceed 99 F in six patients; it reached 104 F in only one and lasted more than two days in only two patients, in contrast to those from whom Shigella was isolated (chart 2). All the patients had diarrhea; colic was noted in nine, nausea in seven, vomiting in seven, and shaking chill in six, chilliness in five, and a temperature over 99 F in five. Leukocytosis of 12,000 and 13,000 occurred in two. Eight had rhinitis and/or pharyngitis. Diarrhea lasted from 3 days in two patients to 12 days in one. The disease in patients ill before and after the middle of September was like that described as viral dysentery by many observers and need not be repeated in detail. All were ambulatory. Nasal obstruction, rhinorrhea, sore throat, or cough were noted in 26% of the patients.

REPORT OF CASES

Severe Disease.—I. I., on Sept. 16, suddenly noted colic, which was shortly followed by a liquid stool and abdominal pain. She suffered from anorexia and passed five watery stools. The next day, she had anorexia, intermittent colic, and six watery stools; during the night, 12 stools were passed. She vomited but kept working. A shaking chill occurred on Sept. 18, with a temperature of 101 F, giddiness, and weakness. She was sent to bed with a temperature of 102 F and a pulse rate of 120 per minute and was given two infusions for rehydration. The nasal and pharyngeal mucosa were injected. She was giddy and had hyperperistalsis and abdominal tenderness. Diarrhea persisted for two days, necessitating four more intravenous infusions. She recovered on the fifth day.

Sigmoidoscopy on the fourth day revealed irregularly distributed areas of inflammation and moderate edema of the mucosa. There were many submucosal ecchymoses. Biopsy showed inflammation, congestion, and edema of the mucosa (fig. 3).

Mild Disease.—S. D. had a sore throat on Sept. 17. The next morning she had sudden colic and one loose stool. Thereafter, she had anorexia and 16 watery bowel movements in 24 hours, but she continued to work. On the third day, there were eight stools, nausea, vomiting, weakness, and general aching but no fever. She was sent to bed on Sept. 20. The nasal and pharyngeal mucosae were injected. The leukocyte count was 6,000. She vomited, but the diarrhea ceased, and she returned to duty. Sigmoidoscopy revealed the rectal mucosa to be slightly reddened, and biopsy showed mild inflammation (fig. 4).

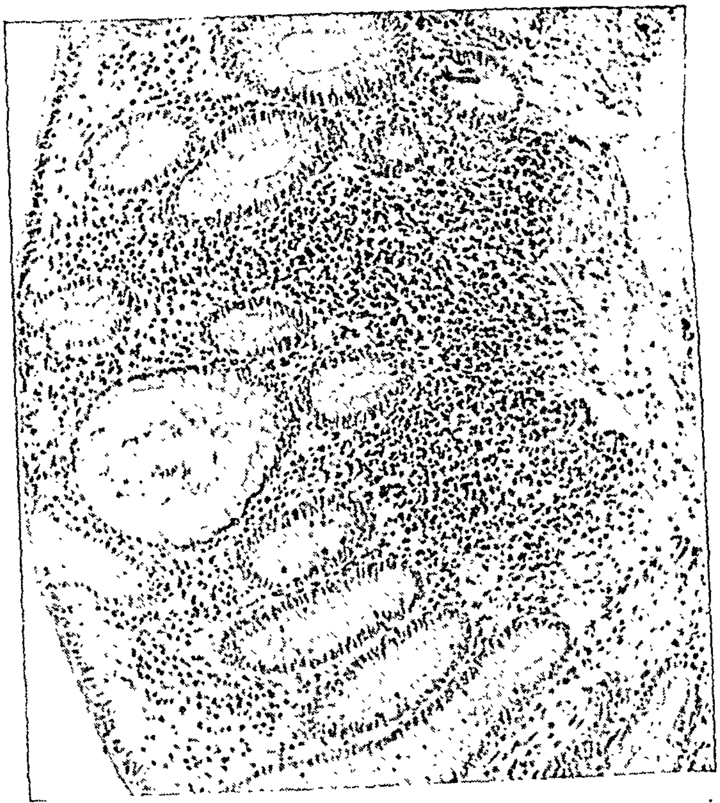

Fig. 4 (patient S. D.).—Inflammation of the rectal mucosa as seen in a patient with mild dysentery, probably viral. The epithelium is hyperplastic in areas. Distended glands are separated by edema of the supportive tissue. Infiltration of tissue with plasma cells and an occasional polymorphonuclear cell can be seen. There is engorgement of blood vessels in mucosa and submucosa (× 100).

COMMENT

According to the observations described, viral dysentery again appeared to be rampant in autumn, but, in the group studied, the sudden peaking over five days, as shown in chart 1, was caused by the coincidence of a small epidemic of bacillary dysentery. Similar circumstances were observed in three epidemics described by English physicians.[5] Another extensive epidemic of diarrheal disease, which lasted over a month in a city in Kansas, in 1942, was regarded as water-borne bacillary dysentery because Shigella was isolated from a few patients;[6] however, a similar mild disease prevalent in nearby communities was called "intestinal influenza."

In the outbreak described, shigellae probably were spread by an undetected carrier who, over a short period, contaminated food served in the nurses' dining room. Probably all nurses who partook of the food ingested an inoculum, but only a few became ill, and Shigella was recovered from 12. Viral dysentery apparently was present among the nurses and may have predisposed some of those who had it to superinfection with Shigella. Otherwise, Shigella may not have been invasive. Other mild enteric disturbances induced by catharsis or by avitaminosis[7] are known to predispose to cholera and to bacillary dysentery. In the healthy volunteers, of Shaughnessy's experiments,[8] large inoculums of Shigella often failed to cause dysentery, and, in many instances, caused only mild, brief diarrhea within 24 hours. There is reason to suppose, therefore, that intestinal disturbance caused by viral dysentery, at times, may render persons susceptible to shigellosis in a manner in which minor viral infections of the respiratory tract predispose to bacterial pneumonias. That such is not always the case is indicated by information furnished by Dr. William W. Frye. A characteristic, large epidemic of presumed viral dysentery affected military personnel and prisoners of war on a Korean island in the summer of 1951. Bacillary dysentery was common in Korea at the time, but it was not detected among the victims of viral dysentery.

The frequency of nasopharyngitis in patients with viral dysentery, as noted in the epidemics of 1943, 1944,[1a] the present one, and by Bradley,[9] Enders,[2] and Beadenkopf[10] is confusing. Many observers make no mention of it or note its absence. The respiratory tract was suggested as a possible portal of entry,[1a] and, in experiments, mild diarrheal disease seemed to be transmitted to volunteers by the inhalation of nebulized, filtered oropharyngeal secretions from patients.[1b] It is not clear, at present, whether nasopharyngitis is an integral part of viral dysentery. If nasopharyngitis represents a separate infection, three diseases were concurrent in the present epidemic. Influenza, colds, and viral dysentery apparently concurred in an epidemic in Los Angeles in 1947.[11] Further uncertainty is added by the occurrence of the opposite circumstance, namely, the presence of gastrointestinal disorders in about 10% of patients with minor infections of the respiratory tract.[12]

Differentiation between viral and bacillary dysentery obviously is difficult. Epidemiologically, "peaking" is unusual for viral dysentery, but it may occur,[13] and bacillary dysentery may appear in low-grade, prolonged epidemics. In this study it is not likely that Shigella was implicated in the many cases scattered throughout the city and its environs during the autumn. Except by the isolation of Shigella, it was impossible, in individual cases, to differentiate the two diseases. The severe form of bacillary dysentery, often portrayed as "typical" in textbook descriptions was not seen, and the symptoms in most cases[14] were the same as those of viral dysentery. However, as suggested by the clinical description and illustrated in chart 2, Shigella was apt to cause severer disease with higher fever, as was also noted by Martin and Wilson.[4d]

The pathological changes in the rectal mucosa are not specific. The mild inflammatory response observed in viral dysentery is indistinguishable from that in mild bacillary dysentery and is the same in Salmonella infection.[15] Yet a predominance of plasma cells or lymphocytes may be more suggestive of a viral than of a bacterial infection. The mild inflammation may not reflect changes possibly present higher in the intestinal tract. They are similar, however, to those in the small intestine in fatal cases described by Britten.[4i] Evidence of inflammation in the intestine may be caused partly by the irritation of hyperperistalsis and in the pharynx by vomiting.

As illustrated by the experience described, diagnosis depends on the accuracy of bacteriological procedure. Bacillary dysentery would not have been diagnosed had dependence rested on the first negative reports from the general laboratory. Shigella was detected only after samples of stools were submitted to experts. Until the cause of viral dysentery is discovered and etiological diagnosis is made possible, differentiation rests on the nature of the epidemic and on the predominance of mild cases suggesting a viral cause on one hand and on the greater number of severer cases and the isolation of Shigella on the other.

SUMMARY

As in previous years, an outbreak of viral dysentery occurred in the early autumn among hospital personnel, students, and the community at large and lasted over a month. A short epidemic of Shigella sonnei colitis limited to nurses was superimposed. Viral dysentery may render patients susceptible to enteric bacillary infections, as viral infections of the respiratory tract predispose to bacterial pneumonia. Differentiation between viral and bacillary dysentery rests on the nature of the epidemic, the greater severity of shigellosis, and the isolation of Shigella.

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[5] Brown, Crawford, and Stent.[4b] *Barnard.*[4c] *Martin* and Wilson.[4d]

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[11] Perri, A. M.: Epidemic Nausea, Vomiting and Diarrhea, Philadelphia Med. **43**: 1125 (March 20) 1948.

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A CLINICAL GUIDE TO PROGNOSIS IN STRESS DISEASES

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and
Harold G. Wolff, M.D., New York

A guide to prognosis in stress diseases has been devised on the basis of experience in the treatment of 739 patients in the Medicine A clinic of the New York Hospital. Its validity has been tested in three selected groups of patients with essential hypertension, migraine, and asthma. An earlier report of the experience of this clinic [1] emphasized a description of the general methods of treatment used, an appraisal of the effectiveness of each, and an estimation of the important prognostic factors. In the present report a more systematic appraisal of prognosis is offered in the form of a score sheet on which a patient can be rated after the first three or four visits to the physician.

Most of the patients were seen at approximately weekly intervals during the first month of attendance at the clinic and at biweekly or monthly intervals thereafter. The average number of visits per patient was 15; the average duration of treatment was nine months. Among these patients 90 had essential hypertension, 62 irritable colon, 67 bronchial asthma, 54 headache due to sustained muscle contraction, 52 migraine, 35 peptic ulcer syndrome, 25 urticaria, 21 vasomotor rhinitis, and 18 ulcerative colitis.

The first step in construction of the scale was to summarize the data in each case under 49 main headings as follows:

Age
Admission information (sex, marital status, and employment)
Race, nationality
Religion
Doctors in charge
System review
Past medical history
Diagnostic headings
Family (general information)
Spouse
Children
Whether contraception used and what type
Childhood
School attendance
School adjustment
Mother
Father
Family values
Siblings
Employment
Employment adjustment
Sex, childhood
Sex, adult
Housing
Character attitude
Character behavior
Estimate of previous life experience
Stress situation in past
Previous treatment
Life situation precipitating present illness
Estimate of information
Duration of illness
Intensity of illness
Patient's attitude toward illness on admission
Patient's attitude toward illness during treatment
Patient's attitude toward treatment on admission
Patient's attitude toward treatment during course of treatment
Course
Mood during course
Therapy by physician
Therapy by social workers
Reasons for leaving clinic
Result on leaving clinic
Appraisal of those still in clinic
Follow-up first year
Follow-up second year
Number of visits
Number of months
Clinic financial rating

These 49 headings were subdivided into 852 items that pertained to the life experience and personal attributes of the patients. The selection of these items was based on the general experience of the entire group of 18 clinic physicians and on their particular experience with the patients of Medicine A clinic over the period of three years. The appropriate item under each heading was checked by the physician in a manner suitable for analysis with an IBM machine.

Independent of the construction of the prognostic scale, an appraisal of the clinical status of each of the 739 patients was made by his physician at yearly intervals or on the patient's discharge or departure from the clinic. The rating was based on changes in signs and symptoms and changes in the way in which the patient saw himself in relation to his life problems and how he handled and reacted to them. Evidence of change in such basic attitudes was adduced from observation of the patient's responses to new situations of significance similar to those that had earlier been associated with symptoms.

On the basis of these clinical appraisals the patients were classified into three groups: 1. Unimproved; these patients displayed no change in either symptoms or attitudes. 2. Moderately or symptomatically improved; these patients displayed a lessening or absence of symptoms without change in basic attitudes. 3. Basically improved; these patients not only were symptomatically improved but displayed a change for the better in their way of dealing with problem situations. The 739 patients fell into the three groups as follows: unimproved, 249, or 34%; symptomatic improvement, 311, or 42%; and basic improvement, 179, or 24%.

These clinical progress appraisals were entered on IBM cards together with the data on life experience and personal attributes as noted above. The various categories, unimproved, symptomatically improved, and basically improved, were then analyzed by the IBM sorting machine so that we might determine which of the coded items were most prevalent in each. Whenever there was a correlation between one of these items and the percentage of patients that fell into the improved groups, the item was selected as possibly significant. The number of items was reduced until those that appeared to be the most significant remained. The prognostic scale was made up of these remaining items, which were weighted in a more or less arbitrary way. It was not possible to evaluate statistically the significance of the various correlations, in view of the extreme variability in numbers and often the very small numerical differences. In addition to the items selected from the code, certain others of presumed significance were arbitrarily added and suitably weighted on the basis of the general clinical impression of the physicians involved.

Groupings were made under three main headings: (1) objective data relating to such items as the patient's age, social status and past medical history (20 points); (2) factors concerning the patient's family and interpersonal

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From the New York Hospital and the Department of Medicine, Cornell University Medical College.

The following physicians gave us the benefit of their clinical judgment with respect to their own patients: Drs. Jacob Bornstein, George Draper, Charles Duncan, William J. Grace, David T. Graham, Andrew D. Hart Jr., Lawrence E. Hinkle Jr., Thomas Holmes, Robert Marcussen, Herbert S. Ripley, Edward Shepard, Harry Stalker, Ian Stevenson, Leonard Straub, Arthur Sutherland, and Theodore Treuting.

1. Ripley, H. S.; Wolf, S., and Wolff, H. G.: Treatment in a Psychosomatic Clinic: Preliminary Report, J. A. M. A. **138**: 949 (Nov. 27) 1948.

relationships obtained from verbatim statements made by the patient (30 points); and (3) evaluations of the patient's past performance, personality structure, and attitude toward his illness, made by the physician (40 points). Table 1 shows the prognosis scale form, with an example of how one patient was scored.

Certain items commonly considered of prognostic importance did not show a positive relationship with clinical appraisals. These items included the sex of the patient, position of the child in the family, bottle or breast feeding, nail biting, night terrors and enuresis in childhood, and the number of visits to the clinic-or the frequency or interval between visits. The following factors were used in making up the prognostic scale.

TABLE 1.—*Prognostic Scoring of a Patient in the Group Studied*

Objective Data	Maximum Score	Actual Score	Comment
Age, up to 40 yr.	2	2	Age 34
Not divorced or separated	2	2	Married
Third generation American, white, gentile	2	2	American family both sides several generations; gentile
High school education complete	2	2	
Both parents living at home until patient was 12 yr. old	4	4	Father died when patient was 18; mother still alive
No surgical treatment for symptoms	2	0	Caldwell-Luc operation in past
No previous formal psychiatric care	2	2	
Steady employment	2	2	Employed 15 yr. same company
Duration of present illness less than 5 yr.	2	0	Onset of asthma 14 yr. previously
Total	20	16	
Previous health good	1	1	Well until onset of asthma
Some religious education	1	1	Family members of parish (religion no source of conflict)
Emotional support from both parents	4	2	Father and mother "fine people"; mother hypercritical, lives with patient
Congenial siblings	2	1	Brother "more successful"; many tastes in common
Emotional support from spouse	2	2	Wife most understanding and helpful
Sexual compatibility with spouse	2	2	
No serious sexual problem	3	2	One extramarital affair with conflict
Spouse adequate economic provider *	2	..	Not applicable
Children not rejected	2	2	Adopted child after death of own son
No housing problem	2	2	Five-room apartment
Occupational satisfaction	2	2	Proud of his work
Congenial working conditions	2	1	Difficulties with boss and fellow workers
Moderately adequate salary	2	2	Able to meet rent and food costs and furnish home
Goal	3	3	Hope of regaining health and buying home
Total (females) (males)	30 28	23	

Objective Data	Maximum Score	Actual Score	Comment
Evidence from past performance: Not exaggerated sensitivity to traumatic situation Good interpersonal relationships (including sexual) Evidence that the activities, achievements, and other life experiences have afforded satisfaction	10	5	Adoption of child after death of first Immature handling of sexual problem, mother, and fellow workers Job satisfaction
Evidence from personality structure: Moderate flexibility Minimal and short-lived hostility Capacity to face and solve personal problems Active participation in the give and take of daily affairs Moderate orderliness and reliability Good judgment and evidence of adequate discrimination regarding human values and goals	10	5	Stubborn Nurtures small hurts Inability to face or resolve dependency; hostility conflict Reliable in meeting financial responsibilities Reasonable life ambition
Prevailing attitudes of the patient toward illness and his problems at time of admission to clinic: Recognition of failure of present patterns of adjustment and willingness to adopt others Willingness to consider possibility that life stress, attitudes, and feeling states are relevant to the illness Confidence in physician, hospital, and its methods Capacity and willingness to assume responsibility in treatment	10	6	Desperate Willing to try anything This hospital last resort Capacity to assume responsibility for treatment doubtful
Extent to which a specific aspect of the life situation was a factor in precipitating the patient's illness: Extent to which this specific feature is modifiable with regard to the involved person, other persons, and circumstances	10	5	No specific life situation apparently related to onset of illness; wife, child, and job considered assets; mother considered a liability
Total	40	21	

* Scored only for female patients.

OBJECTIVE DATA

The following objective data were rated two points each, except where otherwise indicated.

Age, up to Forty Years.—Patients in their forties and fifties (185) were the ones who showed the least evidence of improvement. The greatest degree of change was found among those from 14 to 40 years of age (537). Those in their sixties (12) and seventies (6) were too few to permit evaluation of them as a group, although great improvement was observed in some.

Not Divorced or Separated.—Only 5% of the 739 patients were either divorced or separated. However, in these two groups (divorced and separated) the percentage of no improvement was 47 and 40, respectively.

Third Generation American, White, Gentile.—The obviously increased adjustment problems of subjects who are not of the dominant group in our society prompted the assignment of two points to third generation American, white, gentile, although the preponderance of patients meeting these criteria among the improved group was slight.

High School Education Completed.—There was a slight preponderance of those with completed high school education among the improved group.

Both Parents Living at Home Until Patient Was Twelve Years Old (Four Points).—Twenty per cent of the patients had suffered the loss of one parent at an early age. In these patients the degree of improvement was much less than for the group as a whole.

No Surgical Treatment for Symptoms.—There were 120 patients among the 739 who had never had any surgery; the incidence of improvement among these was much higher than among the 117 who had had one or more operations for the symptoms under consideration.

No Previous Formal Psychiatric Care.—The proportion of improved patients was less among the 44 who had had previous psychiatric care than among the group as a whole.

Steady Employment.—There were 412 patients who were old enough to have been employed for several years and from whom adequate past employment histories could be obtained. The proportion of improved patients among the 280 who had been steadily employed was greater than that among the 132 who changed jobs frequently.

Duration of Present Illness Less Than Five Years.—There were 154 patients in whom symptoms referable to the present illness were of less than one year's duration on admission to the clinic; 75% of these were in the improved group. Of the 224 patients whose illness had lasted more than one year but less than five years, 69% were improved. In the 361 whose illness had lasted more than five years, only 63% were improved.

DATA DEPENDENT ON PATIENT'S INTERPRETATION

The following criteria distinguished those patients who showed the greatest improvement. The categories reflect the patient's own interpretation of his background and experience.

Previous Health Good (One Point).—Those who had had the usual childhood illnesses but no serious diseases or traumatic accidents were among those who showed most improvement.

Some Religious Education (One Point).—Among the improved, those patients who had had some religious education appeared in greater numbers. Fewer of those with a "strict" religious background (attendance at parochial schools) and family stress on religious observation or with no religious education and no family adherence to religious custom were found among the improved group.

Emotional Support from Both Parents (Four Points).—By emotional support from both parents was understood loving care of the child combined with predictable and consistent discipline in keeping with prevailing cultural patterns.

Congenial Siblings (Two Points).—This implied fondness among brothers and sisters, with competition for parental affection and recognition at a minimum.

Emotional Support from Spouse (Two Points).—Credit was given for evidence that the spouse customarily endorsed the patient in public and private and for evidence of mutual interdependence with a minimum of belittling criticism.

Sexual Compatibility with Spouse (Two Points).—Credit depended on an expression of satisfaction by the patient in his intimate sexual relations with his or her spouse.

No Serious Sexual Problem (Three Points).—Serious sexual problems were defined as deviations from the accepted form of sexual behavior in the form of homosexual relations or unusual sexual appetites when they were sources of anxiety and guilt.

Spouse Adequate Economic Provider (Two Points).—This item was included only for the female married patient on the basis of her evaluation of her husband's effectiveness as a provider.

Children Not Rejected (Two Points).—Evidence of rejection included both overt and implicit expression of hostility on the part of patients toward their children.

No Housing Problem (Two Points).—In evaluation of the patient's housing situation, the yardstick was not the standard of housing authorities or the physician, but the patient's own appraisal of his home surroundings. Living in a substandard tenement in a poor neighborhood, if not indicated by the patient as being a source of conflict or disturbance, was not considered a housing problem.

Occupational Satisfaction, Congenial Working Conditions, Moderately Adequate Salary (Two Points Each).—The exact nature of the work done, hours of work, salary obtained, and attitude toward fellow workers and superiors provided evidence of the patient's attitude toward his work. Satisfaction and pride in terms of individual needs were stressed. Scoring is intended to reflect the patient's attitudes toward various aspects of his working life. The score was not based on Department of Labor standards or on the physician's appraisal of the job and salary.

DATA DEPENDENT ON PHYSICIAN'S INTERPRETATION

Many individual attributes were appraised by the physician and found relevant to the patient's course in the clinic. These separate items have been classified into four main groups: (1) past performance, (2) personality structure, (3) attitude toward the present illness, and (4) potential modifiability of the current situation. Each group is weighted 10 points.

Past Performance.—In the evaluation of the patient's reaction to stress, the manner in which adversity had been met in the past was taken into account. The capacity for entering into and maintaining warm interpersonal relations and achieving a sexual adjustment was credited under the category of past performance. Also included was an estimate of the degree to which the patient had been able to derive satisfaction from the vocations and avocations of everyday life.

Personality Structure.—This was evaluated through an analysis of the balance of drives and characteristic behavior as revealed by a review of the life history as a whole and through a discussion of ordinary incidents in daily life. Evidence was sought to illustrate the patient's degree of flexibility and capacity to adapt to change. Indications of extreme hostility, overt or unexpressed, were considered of poor prognostic significance, especially if there were instances of asocial behavior.

A favorable interpretation was placed on indications of resourcefulness and stamina in dealing with personal problems as well as evidence of participation in civic, club, and church activities. Moderate but not excessive orderliness and reliability were also scored as good signs; finally, credit was given for indications of maturity of values and goals.

Attitude Toward the Present Illness.—The physician's evaluation of the patient was focused on an appraisal of the patient's attitude toward his illness. A "declaration of bankruptcy" in which the patient indicated a recognition of failure of past patterns of adjustment and his need for help in making new ones was considered the most favorable attitude. Another helpful indication was the willingness to consider the pertinence of stressful life experiences as factors in bringing about illness. Confidence in the physician and the hospital and a capacity and willingness to assume some responsibility in treatment were other items in this group.

Modifiability.—Attempts were made to determine whether the onset of the patient's illness was contemporaneous with an environmental change, i. e., change of job, school, home environment, death or illness of a parent figure, or return from military service. If such were the case, the possibility of manipulating the environmental situation for the individual patient was evaluated.

METHOD OF APPLYING SCALE

The scoring of one patient is shown in table 1. He achieved 69%, and it was predicted that he would achieve symptomatic improvement without basic change. At the time of writing he had been seen in the clinic over a period of 27 months on 42 occasions—14 in the last 12 months as contrasted with 10 in the first 3 months. His course was a fluctuating one, varying not with the seasons but with the stressful situations at home. He had not missed more than three days at work during this period, compared with the weeks and months of absence in previous years.

TABLE 2.—*Comparison of Prognostic Scale Results with Appraisals of Patients' Clinical Status* *

Results	No. of Patients	Prognosis Rating: Range	Prognosis Rating: Average
Migraine †			
Group 1	18 (35%)	41-61	52
Group 2	30 (58%)	60-74	65
Group 3	4 (7%)	79-83	81
Hypertension ‡			
Group 1	37 (41%)	25-62	51.8
Group 2	44 (49%)	51-58	63
Group 3	9 (10%)	80-89	82.5
Asthma §			
Group 1	18 (27%)	25-59	49.5
Group 2	33 (49%)	52-65	61
Group 3	16 (24%)	70-83	80

* Group 1, unimproved; group 2, symptomatically improved; and group 3, basically improved.

† Fifty-two patients. ‡ Ninety patients. § Sixty-seven patients.

TESTING OF THE PROGNOSTIC SCALE

The scale was applied to 67 patients with asthma, 52 with migraine, and 90 with essential hypertension treated by this medical group. Patients with these diagnoses were selected because they have a well-defined clinical syndrome in which changes in severity can be fairly reliably appraised. When the prognostic scale results were compared with the separately recorded appraisals of the patients' clinical status, a close correlation was observed (table 2).

The 73 patients with hypertension, asthma, or migraine headaches, who averaged a score below 60% on the prognosis score, were found in the unimproved group; only 5 who had been considered unimproved scored over 60%. The symptomatically or moderately improved group comprised 107 patients whose average score was 63%. Only one patient in this group scored above 73%, while five scored below 60%. In the third group of 29 basically improved patients the average score was 81%, with only one scoring below 71%.

Because in retrospect the criteria for improvement or lack of it appeared somewhat rough and not in sufficient detail, an attempt was made in the 90 cases of hypertension to appraise more carefully and in greater detail the clinical status at the time this report was prepared.

The 90 patients fell into four groups: Group 1, or "no change," included 37 patients, or 41% of the total. These patients showed no detectable change in blood pressure, in basic attitudes as defined earlier, or in other stress reactions.

Group 2, or "minor change," was made up of 38 patients, or 41% of the total. This group was further divided into three subgroups: subgroup A, with blood pressure the same, basic attitude only slightly changed or the same, and other stress reactions significantly reduced; subgroup B, with blood pressure lower, basic attitude the same, and other stress reactions reduced; and subgroup C, in which blood pressure became normal, other stress reactions were reduced, but the basic attitude remained the same.

Group 3, or "moderate change," included eight patients, or 9% of the total. In these the blood pressure was the same or slightly reduced, basic attitudes were moderately altered, and the other stress reactions were definitely reduced.

Group 4, or "major change," was made up of nine patients, or 10% of the total. In these the blood pressure had fallen to normal, the basic attitude was much altered, and other stress reactions were greatly reduced.

From an examination of these results, reported in detail in another publication,[2] it would appear that some alteration could be anticipated as a result of a constructive physician-patient relationship in approximately 60% of patients with essential hypertension who complain of symptoms. Most of these, or about 40% of the total, were those changed in that they had fewer symptoms, less outward expression of anxiety, and greater tranquillity than before the experience, although in most of these the blood pressure was not appreciably lowered. However, even in this group a slight lowering of the blood pressure was associated with a decline in symptoms and a restitution of some degree of tranquillity.

The symptom loss was not directly related to a change in the blood pressure level, although in group 4 the loss of symptoms was accompanied by a lowering of the blood pressure. Tremulousness, insomnia, and restlessness were most readily modified. Headache, mainly related to more or less superficially suppressed anger or frustration and to fatigue, was also modifiable and, as mentioned elsewhere,[3] was not related to the level of the blood pressure. These reactions were more easily modified than the blood pressure elevation, which was associated with more deeply repressed anger or frustration. Thus a consideration of life situations, attitudes, and feeling states reduced or eliminated symptoms and other stress reactions in the majority of those with essential hypertension and reduced the blood pressure to normotensive levels in a few. The prognostic scale was applied to the revised data obtained from the more detailed analysis of the clinical state of each patient with hypertension. The results corresponded even more closely than those listed in table 2.

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[2] Wolff, H. G., and Wolf, S.: Hypertension: A Symposium, Minneapolis, University of Minnesota Press, 1951, p. 457.

[3] Wolff, H. G.: Headache and Other Head Pain, New York, Oxford University Press, 1948.

blood pressure was 17 mm. and in diastolic 12 mm. When only the patients who had a decrease, whether uniphasic or biphasic, in blood pressure following the intravenous administration of regitine® were considered the mean fall was 20 mm. systolic and 14 diastolic (table 2).

Table 2.—*Effects of Regitine® Given Intravenously on Blood Pressure of 107 Patients Who Did Not Have Pheochromocytoma*

	Mean Blood Pressure or Changes, Mm. Hg		Percentage of Patients Showing Change in Blood Pressure	
	Systolic	Diastolic	Systolic	Diastolic
Basal	178 ± 3.3	119 ± 21	..	..
Rise	17 ± 1.0 *	12 ± 0.6 *	74 †	62 †
Fall	20 ± 1.6 *	14 ± 0.8 *	64 †	68 †

* Diphasic responses included; all falls or all rises were included as indicated. The pure depressor responses were excluded from calculating average pressor responses and vice versa.

† Some patients showed both a fall and rise in blood pressure after administration of regitine.®

Only rarely did the fall in blood pressure exceed 35 mm. systolic and 25 diastolic except in cases of proved pheochromocytoma; however, in a few instances of essential hypertension depressor responses of this magnitude did occur; these will be discussed separately as false positive reactions.

Effect on Pulse and on Subjective Symptoms.—Regitine® when given intravenously increased the pulse rate 96% of the 107 patients. The mean increase in pulse rate was 17 beats per minute. A few of the patients experienced a mild sensation of warmth with minimal flushing of the skin after regitine® was administered intravenously. There were no serious untoward side-effects.

Comparison with Piperoxan.—Sixty-one hypertensive patients received both regitine® and piperoxan intravenously. The difference in the effects of the two drugs was striking. Whereas regitine® produced minimal side-reactions, piperoxan almost invariably produced unpleasant subjective sensations of heat, palpitation, and apprehension with objective signs of flushing and sweating. The administration of piperoxan to one patient had to be discontinued because it produced severe substernal pain, yet the patient tolerated regitine® without difficulty. In 97% of the patients, piperoxan produced a pressor response of more than 5 mm., which averaged 34 mm. systolic and 21 mm. diastolic. Regitine,® on the other hand, produced a pressor response in only 75% of the patients and the average rise was only 17 mm. systolic and 12 mm. diastolic (table 3). In only three patients did regitine® have a greater pressor effect than piperoxan. Figure 1 compares the blood pressure responses of two patients to whom regitine® and piperoxan were given.

Table 3.—*Comparison of Effects of Regitine® and Piperoxan Given Intravenously on Blood Pressure of 61 Patients with Hypertension*

Blood Pressure After Intravenous Injection	Mean Blood Pressure or Changes, Mm. Hg		Percentage of Patients Showing Change in Blood Pressure	
	Systolic	Diastolic	Systolic	Diastolic
Regitine®				
Basal	193 ± 2.8	127 ± 2.1	..	..
Rise	17 ± 1.1	12 ± 0.8	75	66
Fall	22 ± 2.0	14 ± 1.0	64	69
Piperoxan				
Basal	196 ± 2.9	126 ± 2.2	..	..
Rise	31 ± 1.8	21 ± 1.4	97	93
Fall	14 ± 1.2	9 ± 1.0	33	30

False Positive Reactions.—Regitine,® whether it is given intravenously or intramuscularly, tends to depress the blood pressure of patients with essential and renal hypertension. Usually the blood pressure of these patients is reduced less than 35 mm. of mercury systolic and 25 diastolic. In 4 of the 107 patients studied, the fall in blood pressure was marked enough after the intravenous administration of regitine® to arouse suspicion that excess circulating pressor substance might be present.

Case 1.—A woman, 27 years old, who had terminal nephritis, retinopathy (group 4 [13]) and uremia, evidenced by 250 mg. of urea per 100 cc. of blood, was given 5 mg. of regitine® intravenously. Within two and a half minutes her blood pressure fell from a basal level of 290 mm. of mercury systolic and 188 diastolic to 240 mm. systolic and 156 diastolic. The response to piperoxan was equivocal. Postmortem examination revealed chronic glomerulonephritis but no evidence of a pheochromocytoma.

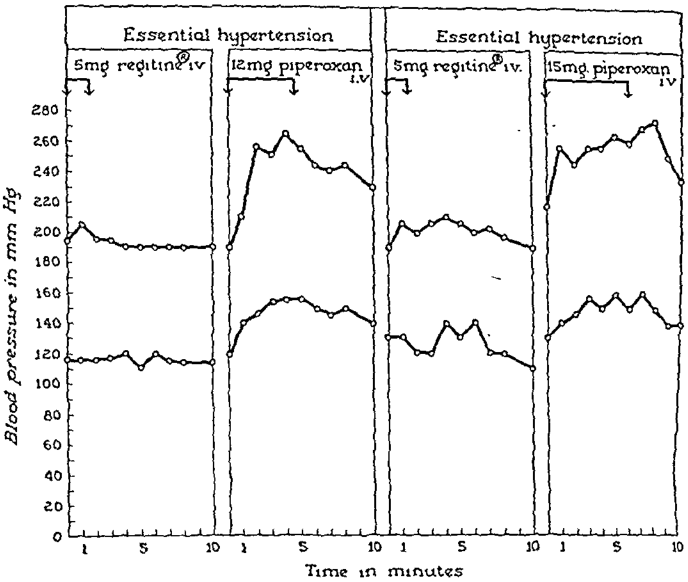

Fig. 1.—Comparison of the changes in blood pressure of two patients after the intravenous administration of 5 mg. of regitine® and later of 12 and 15 mg. of piperoxan respectively.

Case 2.—A woman, 50 years old, gave a short history of paroxysmal attacks of headache and hypertension on admission. Two histamine tests reproduced the headaches, but the elevation in blood pressure following administration of histamine was just about equal to that obtained with the cold pressor test. On one occasion when the patient was observed in a spontaneous attack, she had received 200 mg. of meperidine (demerol®) hydrochloride in the preceding three hours and appeared heavily sedated. Five milligrams of regitine® were administered intravenously, and within three minutes the blood pressure fell from 180 mm. systolic and 90 diastolic to 120 mm. systolic and 66 diastolic. Her headache was temporarily relieved. On two other occasions, when the patient had not received previous sedation, regitine® had no depressor effect on her blood pressure during a spontaneous attack of headache or a histamine-induced attack. Results of a piperoxan test were negative. A thorough abdominal exploration failed to reveal a pheochromocytoma.

Case 3.—A woman, 59 years old, complained of exhaustion, weakness and paroxysmal attacks of constriction in the chest, numbness of the hands, and abdominal bloating. After 5 mg. of regitine® were given intravenously, her blood pressure dropped within two minutes from 210 mm. systolic and 114 diastolic to 148 mm. systolic and 90 diastolic. A similar result with regitine® was obtained on another occasion, but two piperoxan tests gave definitely negative results. An exploratory operation was not done, as her attacks were considered to be due to hyperventilation and not to pheochromocytoma.

CASE 4.—A man, 59 years old, gave a three year history of paroxysmal attacks of hypertension, dyspnea, orthopnea, apprehension, and profuse perspiration. He had a mild case of diabetes. His basal blood pressure was about 120 mm. systolic and 72 diastolic. Provocative tests using histamine, tetraethylammonium chloride, and methacholine hydrochloride failed to induce an attack. He was observed in one spontaneous attack. Unfortunately, he had been heavily sedated with ⅜ grain (0.024 gm.) of morphine sulfate 30 minutes before he was hospitalized. He had acute pulmonary edema, and blood pressure was 180 mm. systolic and 110 diastolic. Ten milligrams (instead of five) of regitine® were given rapidly intravenously, and blood pressure fell to 80 mm. systolic and 60 diastolic within two minutes. Clinically he improved rapidly. Ninety minutes later blood pressure was 90 mm. systolic and 60 diastolic, and the pulmonary edema had largely disappeared. A thorough abdominal exploration failed to disclose a pheochromocytoma.

RESULTS OF INTRAMUSCULAR TESTS

One hundred and fifty-four patients received regitine® intramuscularly. Of these five proved to have pheochromocytomas. Of the remaining 149 patients, 11 were normotensive and 138 were hypertensive. As with regitine® given intravenously there was no essential difference in the blood pressure response to regitine® of patients in either group or among patients in various groups of hypertension, so they will be discussed together.

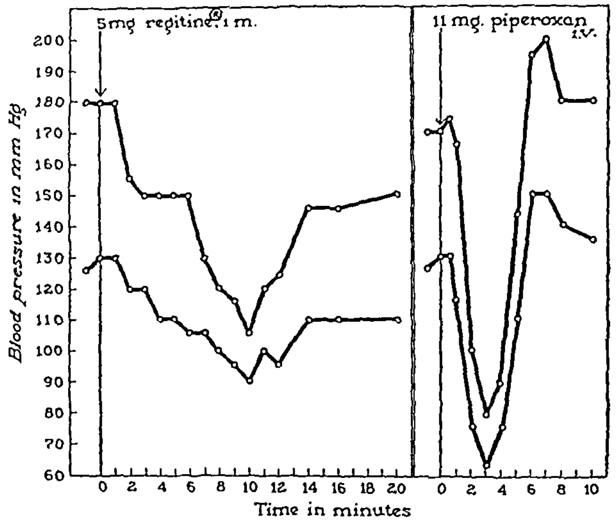

Fig. 2 (case 5).—Changes in blood pressure resulting from the intramuscular administration of 5 mg. of regitine® and on another occasion from the intravenous administration of 11 mg. of piperoxan.

Effect on Blood Pressure.—Except for a slower response following the intramuscular injection of regitine,® the effect on blood pressure was the same regardless of the route of administration (table 4). What has already been said concerning the effects of regitine® given intravenously on blood pressure, applies, in general, to that observed after regitine® was given intramuscularly. When regitine® was given intramuscularly, however, the average time for its maximal effect on blood pressure was 13 minutes, while the maximal effect was noted within 4 minutes after intravenous administration.

Effect on Pulse and on Subjective Symptoms.—The effects of regitine® given intramuscularly on pulse rate were variable. Seventy-nine per cent of the patients showed a rise in pulse rate, the average rise being only 10 beats per minute as compared to 17 following intravenous administration of regitine.® The pulse rate of 68% of the patients decreased an average of 7 beats per minute. As with the blood pressure, many of the patients showed both a rise and fall in pulse rate. None of the patients complained of any unpleasant side-effects following the intramuscular administration of regitine.®

TABLE 4.—*Effects of Regitine® Given Intramuscularly on Blood Pressure of 149 Patients Who Did Not Have Pheochromocytoma*

	Mean Blood Pressure or Changes, Mm. Hg		Percentage of Patients Showing Change in Blood Pressure	
	Systolic	Diastolic	Systolic	Diastolic
Basal...............	180 ± 3.0	116 ± 2.0	..	..
Rise...............	17 ± 0.8 *	13 ± 0.6 *	62 †	58 †
Fall...............	19 ± 1.1 *	14 ± 0.7 *	72 †	63 †

* Diphasic changes included; all falls or all rises were included as indicated. The pure depressor responses were excluded from calculating average pressor responses and vice versa.

† Some patients showed both a fall and a rise in blood pressure after administration of regitine.®

Comparison with Piperoxan.—One hundred hypertensive patients received regitine® by the intramuscular route and piperoxan by the intravenous route. The results were just as striking as when results of the intravenous use of regitine® were compared to those of piperoxan. Unpleasant symptoms that almost invariably accompanied the administration of piperoxan were entirely lacking when regitine® was given intramuscularly. The pressor response following regitine® was much less than that following piperoxan (table 5). In only 1 patient in this group of 100 was the pressor response after administration of regitine® greater than that after piperoxan.

False Positive Reactions.—Three of the 149 patients showed a depressor response following use of regitine,® which suggested that an excess amount of a pressor substance might be present in the circulating blood. In none of these three cases, however, was a pheochromocytoma proved to be present, and, therefore, the reaction must be considered false positive.

TABLE 5.—*Comparison of Effects of Regitine® Given Intramuscularly and of Piperoxan Given Intravenously on Blood Pressure of 100 Patients*

Drugs Administered	Mean Blood Pressure or Changes, Mm. Hg		Percentage of Patients Showing Change in Blood Pressure	
	Systolic	Diastolic	Systolic	Diastolic
Regitine® Given Intramuscularly				
Basal...............	198 ± 2.7	125 ± 3.2	..	..
Rise.....	17 ± 1.1	13 ± 0.9	67	57
Fall...............	20 ± 1.3	14 ± 0.9	73	67
Piperoxan Given Intravenously				
Basal...............	198 ± 2.4	127 ± 1.6	..	..
Rise	34 ± 1.6	20 ± 1.0	98	94
Fall...............	13 ± 1.5	10 ± 0.9	29	19

One patient, who had terminal uremia, was given regitine® intramuscularly as well as intravenously. Following the administration of 10 (instead of 5) mg. of regitine® intramuscularly, her systolic blood pressure slowly dropped from 256 mm. to 198 and her diastolic pressure from 160 mm. to 120 in 40 minutes. Although the maximal fall in blood pressure did not occur in the first 20 minutes after injection, the magnitude of the fall led us to consider this a positive reaction. As previously stated, no pheochromocytoma was found at necropsy.

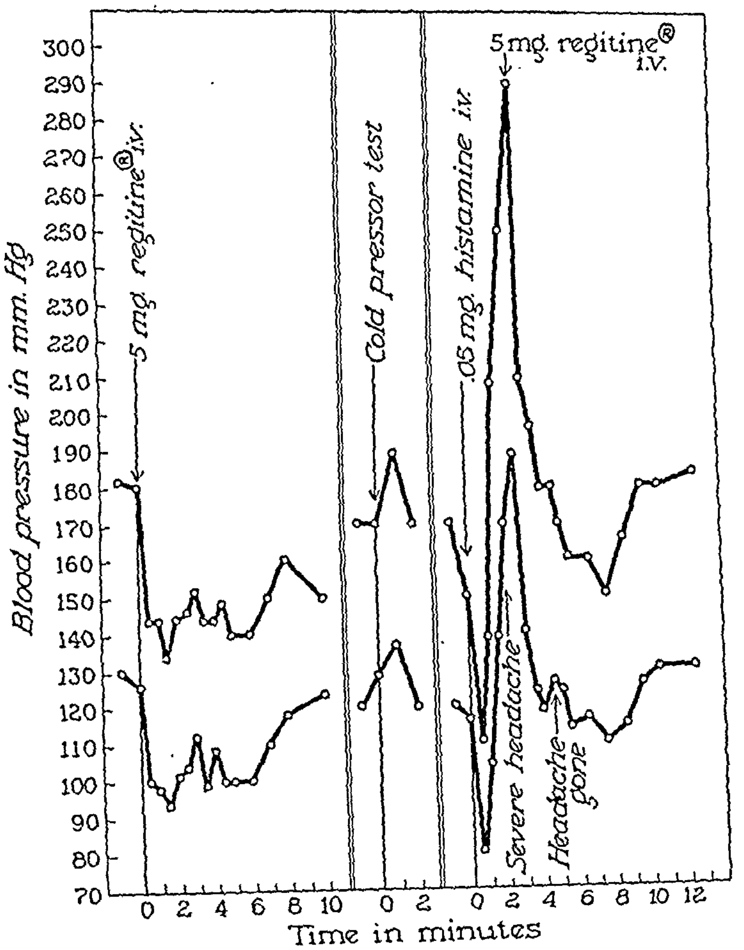

Fig. 3 (case 6).—Changes in blood pressure resulting from the intravenous administration of 5 mg. of regitine®; also from the intravenous administration of 0.05 mg. of histamine, and subsequent intravenous injection of 5 mg. regitine.®

The other two patients who had false positive reactions to intramuscular tests with regitine® had basal blood pressures that were low in the hypertensive range, and after the administration of regitine® the fall in blood pressure, though not of great magnitude, was enough to put them in the normotensive range. In one instance the blood pressure dropped from 164 mm. systolic and 100 diastolic to 130 mm. systolic and 78 diastolic. In the other case the fall was from 148 mm. systolic and 84 diastolic to 120 mm. systolic and 70 diastolic. A negative result of the histamine test in the latter and a negative result of the piperoxan test in the former ruled out pheochromocytoma.

RESULTS OF REGITINE® TESTS ON PATIENTS WITH PHEOCHROMOCYTOMA

Paroxysmal Hypertension.—Two patients with paroxysmal hypertension due to pheochromocytoma were observed. In the normotensive phase, regitine® had no unusual effect on the blood pressure whether given intravenously or intramuscularly. This was not unexpected. Regitine,® when given rapidly into the vein, did abort the attacks induced by histamine. Hightower, Priestley, and Roth [14] also have used regitine® to control the hypertensive crises of these patients at operation as originally suggested by Grimson.[9b,c]

Sustained Hypertension.—Five patients had sustained hypertension due to tumors of the adrenal medulla, which were removed surgically. The results of the pharmacologic tests in four of these cases are presented in figures 2, 3, 4, and 5.

Case 5.—A man, aged 47, had a right adrenal tumor that was removed, but hypertension persisted after operation because of functioning metastatic lesions. Preoperatively his resting blood pressure was 180 mm. systolic and 130 diastolic. A prompt and marked fall in blood pressure was observed after piperoxan was given intravenously and after 5 mg. of regitine® was given intramuscularly (fig. 2). Regitine® was not administered intravenously to this patient.

Case 6.—A pheochromocytoma, weighing 575 gm., was removed from the right adrenal gland of a woman, 46 years old, who had sustained hypertension in addition to a history of paroxysmal attacks of palpitation, tremor, sweating, and pain in the thorax. Before operation, 5 mg. of regitine® given intravenously promptly reduced her blood pressure from a basal value of 180 mm. systolic and 126 diastolic to 134 mm. systolic and 94 diastolic (fig. 3). Five milligrams of regitine® given rapidly intravenously quickly aborted an attack induced by histamine in this patient (fig. 3).

Two patients (cases 7 and 8) were sisters and were found to have bilateral pheochromocytomas at operation. Their case histories will be presented in detail in another paper.[15]

Case 7.—The younger of two sisters, aged 18, had a basal blood pressure of 190 mm. systolic and 140 diastolic. On two occasions regitine® given intramuscularly failed to reduce her blood pressure. But when regitine® was given intravenously, it produced a sharp, prompt fall in blood pressure followed by a marked rise. The reaction to the piperoxan test was likewise positive for pheochromocytoma (fig. 4).

Case 8.—The older of two sisters, aged 28, had extremely labile blood pressure that made the results of pharmacologic tests hard to interpret. Blood pressure fell promptly and markedly after the intravenous administration of both regitine® and piperoxan (fig. 5). Following the intramuscular administration of regitine,® blood pressure fell more gradually; the mag-

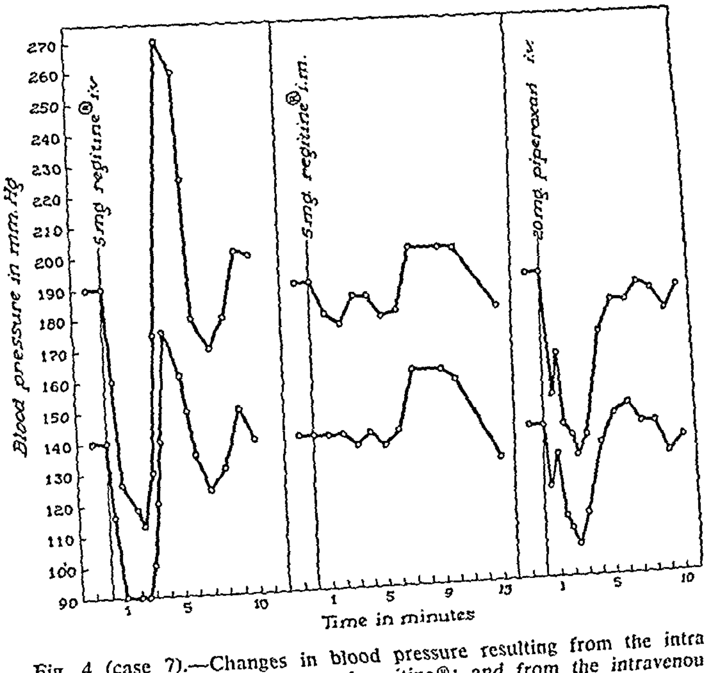

Fig. 4 (case 7).—Changes in blood pressure resulting from the intravenous administration of 5 mg. of regitine®; and from the intravenous administration of 20 mg. of piperoxan. The lack of immediate fall in blood pressure after intramuscular injection of regitine® should be noted.

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[14] Hightower, N. C.; Priestley, J. T.; Roth, G. M., and Kvale, W. F.: The Control of Blood Pressure with Pheochromocytoma During Surgery and the Postoperative Period, unpublished data.

[15] Roth, G. M., and others: Bilateral Familial Pheochromocytoma: Report of Three Cases in the Same Family, to be published.

nitude of the fall was great when compared to the immediate preinjection level but not so great when compared to the lowest preinjection level.

Case 9.—The patient, a woman, 25 years old, had sustained hypertension secondary to pheochromocytoma. Preoperatively two tests using regitine® intravenously gave definitely positive results: the systolic blood pressure was reduced from 190 to 88 mm. and the diastolic from 130 to 62 mm. A test with piperoxan performed one hour before the second regitine® test gave negative results. A test with tetraethylammonium chloride also gave negative results. The pharmacologic diagnosis in this case, therefore, rested entirely on the positive result of the regitine® tests. At operation, four tumors were removed, three from the right adrenal gland and one from behind the liver just below the diaphragm above the usual site of the adrenal gland.

Thus regitine® was given intramuscularly to three of the five patients with sustained hypertension due to pheochromocytoma. Two of the four tests using regitine® intramuscularly on these three patients gave positive results and two gave negative results. For all four of the patients on whom the intravenous test with regitine® was done, results were positive. No false negative reactions were observed when regitine® was given intravenously.

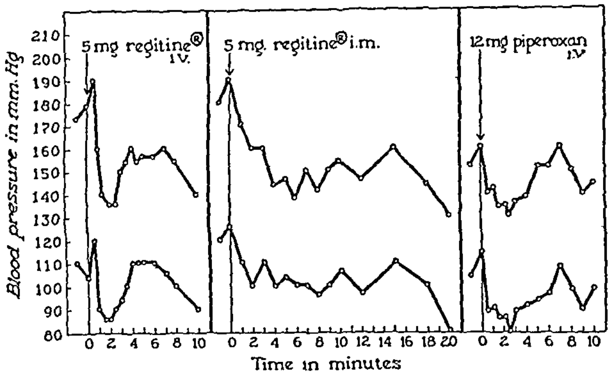

Fig. 5 (case 8).—Changes in blood pressure resulting from the intravenous administration of 5 mg. of regitine®; from the intramuscular administration of 5 mg. of regitine®; and from the intravenous administration of 12 mg. of piperoxan.

COMMENT

Pheochromocytoma associated with sustained hypertension can masquerade as and be clinically indistinguishable from essential hypertension; therefore, most patients with hypertension should have a test for pheochromocytoma. Regitine,® which has been found to be nontoxic, easy to administer, and free of unpleasant side-effects is an excellent drug for screening such patients. From the results just presented it is clear that regitine® should be administered intravenously rather than intramuscularly, since false negative reactions were encountered twice in cases in which pheochromocytoma was present when the drug was given intramuscularly.

Furthermore certain precautions must be considered in the execution and interpretation of this test. Regitine® is not a provocative test drug (such as histamine, tetraethylammonium, or methacholine) and is of no value in the diagnosis of paroxysmal hypertension owing to pheochromocytoma unless it is given during a paroxysm. In this respect it is similar to piperoxan and dibenamine.® As a test drug, regitine® should not be given in doses of more than 5 mg. In both instances in which 10 mg. were given, results were false positive. Emlet and co-workers also have found that 5 mg. is the optimal dose of regitine® for the test.[7a]

The importance of omitting sedation before any of the pharmacologic tests are performed for pheochromocytoma cannot be overemphasized. The patients should have no sedatives or narcotics for 24 hours before the test. In two patients to whom regitine® was given while they were heavily sedated, results were positive and led to a fruitless exploration. In one of these patients, regitine® tests performed without previous sedation gave negative results. The confusing results obtained when these drugs are administered to anesthetized patients[3] only serve to emphasize this necessary precaution. Likewise, patients who have hypertension and uremia are likely to have false positive reactions to regitine® tests.[7a] One such case is included in this report.

Furthermore, falsely positive results with regitine® are likely to be obtained in patients whose blood pressure is in the low hypertensive range (from 140 to 170 mm. systolic and 90 to 110 diastolic). Even a relatively small decrease may bring the blood pressure of these patients into the normotensive range and thus give the false impression of a positive reaction. It is preferable to use a provocative test, such as histamine, for these patients.

Before any of the test drugs are administered, the patient's blood pressure should be allowed to stabilize while he rests in a quiet environment. If the drugs are given too soon, the depressor effect of rest in bed alone may be attributed to the drug. Finally it must be realized that 66% of all patients to whom regitine® was given responded with a fall in blood pressure that averaged 20 mm. systolic and 14 diastolic. A fall in blood pressure of as much as 30 mm. systolic and 20 diastolic was frequently observed in patients with hypertension due to causes other than pheochromocytoma. In spite of all these precautions, hypertensive patients will be encountered occasionally in whom the depressor effect of regitine® will be great enough to simulate that in patients with pheochromocytoma. One such patient was encounterered in this series of 259.

As a screening test to rule out pheochromocytoma, regitine® is preferred to piperoxan because it is a pleasanter drug from the patient's standpoint and because it is almost free of the unpleasant side-reactions caused by piperoxan. Furthermore, regitine® is a safer drug to give to hypertensive patients because it does not produce the alarming rise in blood pressure that is frequently encountered after giving piperoxan.

Regitine® is easier to administer, as the test can be performed by one person, who can give the injection in an instant and then record the blood pressures. In contrast, piperoxan must be given more slowly and hence one person must give the injection while a second person records the blood pressures. For the unaided general practitioner the regitine® test is preferable.

No falsely negative reactions have been recorded as yet from regitine® given intravenously. The fact that false positive reactions occur with disappointing frequency does not detract from its value in a screening test. These false positive reactions can be kept at a minimum if all the precautions mentioned are observed. All positive reactions should be checked with piperoxan or one of the provocative tests. The surprising frequency of false negative results produced by piperoxan must be kept in mind.[6] This all points to the important fact that no single test is infallible in the diagnosis of these tumors.

SUMMARY AND CONCLUSIONS

Regitine® (C-7337) has been used as a test for pheochromocytoma on 259 patients, 7 of whom had pheochromocytoma. The test is most reliable if the drug is given intravenously in doses of 5 mg. When given in this manner, regitine® produced a marked fall in blood pressure in patients who had sustained hypertension secondary to pheochromocytoma. Regitine® had some depressor effect on the blood pressure of 66% of patients who had essential hypertension, but blood pressures of these patients were usually decreased less than 35 mm. of mercury systolic and 25 diastolic.

False positive reactions were obtained in 3 out of 149 patients without pheochromocytomas to whom regitine® was given intramuscularly and in 4 out of 107 patients to whom regitine® was given intravenously. These false positive reactions were attributed to the following factors: (1) sedation before the test, (2) uremia, (3) basal blood pressures that were in the low hypertensive range, and (4) the unexplained depressor effect of regitine® on the blood pressure of some hypertensive patients.

Regitine® is a safer and pleasanter test drug than piperoxan and also is easier to administer. It is an excellent drug to use for a "screening test" on all hypertensive patients to rule out pheochromocytoma. Equivocal reactions with regitine® should be confirmed by pharmacologic tests with other drugs, as no one drug is infallible; however, it must be emphasized that in one of the patients reported herein the preoperative diagnosis was made on the basis of the markedly positive result of the regitine® test and in spite of a negative result of the piperoxan test.

MESENTERIC VENOUS THROMBOSIS ASSOCIATED WITH ACUTE APPENDICITIS IN A YOUNG CHILD

RECOVERY FOLLOWING RESECTION OF INFARCTED INTESTINE

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and
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Mesenteric vascular occlusion is one of the rarest and most uniformly fatal of the intra-abdominal catastrophes. Without surgical therapy, the condition carries an extremely grave prognosis. The recently reported mortality rates following definitive operative treatment vary between 60% and 70%.[1] McClenahan and Fisher,[1c] reviewing the literature up to 1948 and including their own series, were able to report a total of 616 cases of mesenteric vascular occlusion, in 43 (7%) of which the condition was treated successfully by intestinal resection.

Mesenteric vascular occlusion may be arterial or venous in origin. Arterial occlusion is most often associated with arteriosclerosis and cardiac disease. Mesenteric venous thrombosis is usually a complication of acute intra-abdominal infections, such as acute appendicitis, pelvic thrombophlebitis and abscess, and diverticulitis and peritonitis.[2]

The age range of greatest incidence of mesenteric vascular occlusion is between 20 and 70 years, with an average age of 43 years.[1a] The condition is decidedly rare in children. In 1940, Johnson,[3] including his own case, found only 11 cases of mesenteric vascular occlusion in infants and children in the English literature. In the case to be reported, mesenteric venous thrombosis associated with acute appendicitis occurred in a 2½-year-old child. Resection of the infarcted intestine with primary anastomosis resulted in complete recovery. Although instances of mesenteric vascular occlusion have been reported in infants of 1 month[4] and 4 months,[5] review of the literature does not reveal a case of successful intestinal resection for the condition in a child of this age group.

REPORT OF A CASE

On Oct. 12, 1951, a 2½-year-old Negro male child was admitted to the pediatric service of Flower and Fifth Avenue Hospitals with the complaints of sore throat and abdominal pain. The present illness began on the morning of the fifth day prior to admission, at which time the patient complained of a sore throat. His mother noted that he seemed ill, and she reported that his temperature was 102 F at that time. On the following day, the patient's throat pain was worse and he was taken to a local hospital, where the diagnosis of "acute tonsillitis" was made. He was given an injection of penicillin. On the third day of his illness, the patient's fever and sore throat persisted and he was given another penicillin injection. The day before his admission, the patient's temperature rose to 104 F, and, along with the sore throat, he began to complain of diffuse abdominal distress. He was listless and anorexic but did not vomit. His bowel movements were normal, the last one occurring on the day before admission. He was again given a penicillin injection at the clinic of his local hospital. The patient was seen at Flower and Fifth Avenue Hospitals for the first time on the afternoon of Oct. 12 and was immediately admitted to the pediatric ward. His complaints at the time of admission were essentially those of the preceding day: sore throat and diffuse abdominal pain, which was apparently not colicky.

From the Department of Surgery, New York Medical College.

[1] (*a*) Brown, M. J.: Mesenteric Venous Occlusion: A Clinical Entity, Am. J. Surg. **49**: 242-249, 1940. (*b*) Laufman, H., and Scheinberg, S.: Arterial and Venous Mesenteric Occlusion: Analysis of 44 Cases, Am. J. Surg. **58**: 84-92, 1942. (*c*) McClenahan, J. E., and Fisher, B.: Mesenteric Thrombosis, Surgery **23**: 778-785, 1948.

[2] Berry, F. B., and Bougas, J. A.: Agnogenic Venous Mesenteric Thrombosis, Ann. Surg. **132**: 450-474, 1950. Brown.[1a] Cokkinis, A. J.: Mesenteric Vascular Occlusion, London, Wood, 1926. Moore, T.: Mesenteric Vascular Occlusion, Brit. J. Surg. **28**: 347-356, 1941.

[3] Johnson, G. D.: Mesenteric Vascular Occlusion in Children: Report of a Case, Am. J. Dis. Child. **60**: 640-643, 1940.

[4] Laganne, cited by Trotter, L. B. C.: Embolism and Thrombosis of Mesenteric Vessels, London, Cambridge University Press, 1913, p. 96.

[5] McLanahan, B. V.: Mesenteric Thrombosis: Report of Case 4 Months of age, Illinois M. J. **49**: 514-515, 1926.

The neonatal and postpartum courses were normal. There was no history of serious past illness. The child had received the usual immunizations and vaccinations.

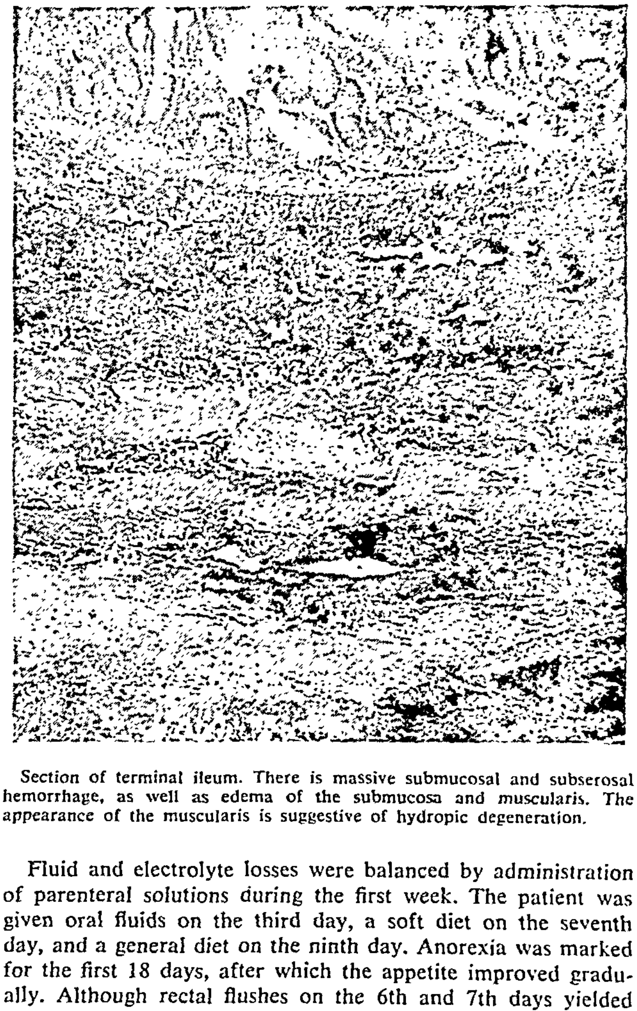
Physical examination revealed a well-developed and well-nourished young child. He was feverish and irritable and resisted examination. His weight was 30½ lb. (13.8 kg.). The temperature was 103.4 F rectally, the pulse rate 96, and respirations 30. The pharynx and tonsils were beefy red and edematous but without exudate. There was diffuse mild tenderness throughout the abdomen but no rigidity. Rectal examination was unsatisfactory but revealed no localized tenderness, mass, or blood.

Laboratory observations were as follows: hemoglobin 11.2 gm. (72%), erythrocytes 3,700,000 per cubic millimeter and leukocytes 18,500, with 81% polymorphonuclear neutrophils. Blood culture yielded no growth. The urine was normal except for a moderate number of hyaline and finely granular casts.

Late in the evening of the day of admission, surgical consultation was requested because the patient was becoming more toxic in appearance, and there had been a rapid increase in the severity of the abdominal pain. On examination at this time, there was extreme tenderness of the entire abdomen, although it was more marked in the right lower quadrant. There was voluntary spasm but no rigidity. The abdomen was not distended. Neither abdominal nor rectal examination revealed the presence of a mass. A flat roentgenogram of the abdomen showed a small amount of gas irregularly distributed throughout the colon. Several loops of small intestine were also slightly distended with gas in the middle and lower portions of the abdomen. Because of the severity and progression of the patient's abdominal signs, exploratory laparotomy was deemed advisable. Codeine, 1/8 grain (8 mg.), and atropine, 1/350 grain (0.17 mg.), were given as premedication.

The patient was anesthetized with cyclopropane, and a plastic cannula was inserted in an ankle vein. Palpation of the abdomen under anesthesia revealed the presence of a doughy elliptical mass, approximately 10 cm. by 4 cm. in size, lying horizontally in the right lower quadrant. The abdomen was entered through a low right rectus muscle-splitting incision. When the peritoneum was incised, a thick, purulent, fecal smelling, nonsanguineous fluid was encountered, and about 200 cc. was removed by suction. Several loops of normal appearing small intestine protruded through the incision. These were packed off to expose the affected bowel. The terminal ileum was edematous and reddish purple for a distance of approximately 17 cm. This segment of bowel was doughy and heavy, lying upon the posterior pelvic brim. The affected ileum was about 5 cm. in diameter but was not distended; the wall was thickened and soggy in texture. The purplish discoloration stopped abruptly at the ileocecal valve. However, the most marked change was observed in a 10-cm. segment just proximal to the ileocecal valve. In this area, the ileum was purplish black, thin-walled, and lusterless. There were several flecks of fibrin on the affected bowel, but no adhesions were noted. The cecum was edematous and red. The appendix, which was necrotic in its terminal third, was adherent to the under surface of the ileocecal mesentery. The mesentery to the terminal ileum and cecum was greatly edematous, its thickness approximating 2.5 cm. There were numerous enlarged pink friable lymph nodes throughout the mesentery. There had been no evidence of internal hernia or intussusception. Therefore, it was our belief that we were dealing with an ascending mesenteric venous thrombophlebitis that was probably secondary to the acute gangrenous appendicitis. The discolored ileum was wrapped in hot packs for 30 minutes, during which the appendix was extirpated. When the ileum was reexamined, its color had improved somewhat but was still bluish; pulsations and contractility were noted in all but the terminal 10 cm. The latter segment was plum-colored, thin-walled, and dull appearing; it did not exhibit peristalsis when stimulated and lacked arterial pulsations. Believing this segment to be nonviable, we elected to perform a resection and primary anastomosis. The distal 30 cm. of ileum, the cecum, and about 5 cm. of ascending colon were resected, well beyond the grossly affected area. The ileum was sectioned obliquely in order that its diameter might be equalized with that of the colon, and an end-to-end ileo-ascending colostomy was performed. A 22 F catheter was threaded through a Witzel enterostomy located about 2 ft. (61 cm.) proximal to the ileocolostomy. The tip of the catheter was placed at the anastomosis, while its opposite end was brought out through a stab wound in the left lower quadrant to provide decompression of the segment proximal to the anastomosis. Drainage of the right lumbar gutter and pelvis was effected by two Penrose drains extending through a stab wound in the right lower quadrant. The operating time was 2 hours 35 minutes, during which the patient's condition was considered good. A slow intravenous infusion of 250 cc. of isotonic sodium chloride solution was administered during the operative procedure.

The postoperative course was remarkably uneventful. Massive doses of antibiotics (including aureomycin, penicillin, and streptomycin) were administered parenterally for the first 10 days. At no time did the temperature rise above 101.8 F. (rectally); normal temperatures were recorded from the seventh postoperative day until discharge. Heparin was administered in 25-mg. doses every four to six hours for the first five postoperative days. Distention was notably absent. The enterostomy tube drained a daily average of 125 cc. of thick bilious fluid and was removed on the sixth postoperative day, with no further drainage.



Section of terminal ileum. There is massive submucosal and subserosal hemorrhage, as well as edema of the submucosa and muscularis. The appearance of the muscularis is suggestive of hydropic degeneration.

Fluid and electrolyte losses were balanced by administration of parenteral solutions during the first week. The patient was given oral fluids on the third day, a soft diet on the seventh day, and a general diet on the ninth day. Anorexia was marked for the first 18 days, after which the appetite improved gradually. Although rectal flushes on the 6th and 7th days yielded formed stools and flatus, it was not until the 14th day that the patient had a spontaneous bowel movement, followed by regular normal bowel action. Barium enema and gastrointestinal examination three weeks postoperatively demonstrated free passage of the medium through the anastomosis.

The patient's weight fell from a preoperative level of 30½ lb. (13.8 kg.) to 27¼ lb. (12.3 kg.) on the ninth day. At the time of dismissal from the hospital, on the 24th postoperative day, his weight was 29 lb. (13.2 kg.). The hemoglobin was 10.5 gm. (70%) on the day following operation; it rose to 12.5 gm. (80%), with 4,300,000 erythrocytes per cubic millimeter, at the time of discharge. The leukocyte and differential counts were normal at this time.

Pathological Observations.—Gross and microscopic examinations of the resected specimen were reported by Dr. F. D. Speer of the department of pathology as follows: The specimen consisted of 28 cm. of the terminal ileum and 8 cm. of the cecum and ascending colon. The ileum was markedly edematous and congested and dark blue. The mucosal and serosal surfaces were intact. The cecum was also markedly edematous throughout its wall but lacked the congestion present in the ileum. The mucosal surface was intact, but the serosal surface was sutured at the appendiceal stump. A small amount of mesentery was present in the region of the ileocecal valve, which contained many firm lymph nodes. The resected appendix measured 11 cm. in length and 0.7 cm. in diameter. Its serosa was hemorrhagically pigmented and covered by thick plastic exudate. On section, the lumen was distended and contained fecal matter and purulent hemorrhagic material. The wall was necrotic.

Study of sections of the appendix showed a large, partially calcified fecalith obstructing the lumen, with extensive necrosis of the mucosa and muscularis. Massive, purulent exudate and thrombosis of the vessels in the intermuscularis and serosa were noted. Study of sections of the terminal ileum showed massive edema and hemorrhage of the submucosa and muscularis (see the accompanying figure). Beginning necrosis of the muscle and inflammatory erosions of the muscosa were seen. There was also massive edema, hemorrhage, and purulent exudate in the serosa of the small bowel and fibrinous inflammation on the serosa.

The diagnoses were as follows: 1. In the appendix there was a large fecalith with extensive gangrenous inflammation and periappendicitis. 2. In the terminal ileum there was beginning hemorrhagic infarction with marked fibrinocellular enteritis and perienteritis. 3. The mesenteric lymph nodes showed nonspecific exudative and hyperplastic lymphadenitis.

COMMENT

Acute appendicitis in children less than 5 years of age carries a mortality rate that is more than three times that occurring in older children with the disease.[6] The greater mortality rate in the younger age groups is related to the high incidence of perforation with diffuse peritonitis. Taylor and Hodges[7] reported a 71.7% incidence of perforation in children less than 5 years of age with acute appendicitis. The frequency of perforation is in a large measure due to delay in definitive treatment. Difficulties in diagnosis, failure of parents as well as physicians to recognize that acute appendicitis does occur in the very young, the tendency to give cathartics for abdominal distress, and the lack of realization that appendicitis as well as gastroenteritis is frequently associated with respiratory infections in children all contribute to the delay that may allow perforation. Our case of mesenteric thrombosis illustrates to what extremes the complications of acute appendicitis and abdominal sepsis in children may ultimately progress. It further supports the view that immediate operation is the treatment of choice in young children with unrelenting abdominal distress and tenderness.

Treatment of an acute respiratory infection without consideration of associated abdominal distress may prove fatal. It should be emphasized that upper respiratory infections precede acute appendicitis in children in approximately 20% of cases.[8] Vigorous therapy of pharyngitis or tonsillitis with antibiotics may mask the symptoms of progression of smoldering appendicitis. Therefore, when antibiotics are being administered, one should maintain a high index of suspicion of appendicitis when confronted with vague abdominal complaints. It has been demonstrated that penicillin therapy may alter the coagulability of the blood[9]; this may have been an additional factor in the development of the mesenteric vascular thrombosis in the reported case.

Several points in the surgical management of mesenteric vascular occlusion as related to our case deserve mention. Palpation of the abdomen under anesthesia revealed the presence of a mass that was not noted in the previous physical examination. The position of the mass helped us in deciding on the location of the incision. Young children do not tolerate enterostomies well; for this reason, we elected to do a resection and primary anastomosis. This case illustrates the value of resecting well beyond the grossly affected bowel when dealing with mesenteric vascular occlusion as has been emphasized by others.[10] Because of the need for immediate effective decompression of the segment proximal to the anastomosis, a Witzel enterostomy was employed, with the catheter extending to the anastomosis. It was considered that this method would be preferable to long-tube intestinal intubation in order to preclude the delay involved in advancement of the double lumen tube. Finally, following the suggestion of d'Abreu and Humble,[11] we administered heparin postoperatively in an effort to prevent further thrombosis. It is difficult to assess the effectiveness of this measure in our case since the coagulation times were not within therapeutic ranges, although large doses of the drug were administered.

SUMMARY

A case of mesenteric venous thrombosis occurring in a 2½-year-old child is reported. It is believed that the condition was secondary to acute gangrenous appendicitis. Wide resection of the infarcted ileum with primary ileo ascending colostomy resulted in complete recovery. The frequency with which serious complications attend acute appendicitis in the very young emphasizes that immediate operation is mandatory when there is a suspicion of an "acute abdomen."

521 Park Ave. (Dr. Miller).

6. (*a*) Allen, P. D.: Acute Appendicitis in Children, J. A. M. A. **109**: 121-125 (July 10) 1937. (*b*) Bockus, H. L., and others: Gastro-enterology: The Small and Large Intestines and Peritoneum: Diagnosis and Treatment of Disorders of the Small Intestine, Colon, Peritoneum, Mesentery, and Omentum, Philadelphia, W. B. Saunders Company, 1944, vol. 2, p. 886. (*c*) Hudson, H. W., Jr., and Chamberlain, J. W.: Acute Appendicitis in Childhood: Statistical Study of 848 Cases from Children's Hospital, Boston, J. Pediat. **15**: 408-425, 1939.

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8. Allen.[6a] Hudson and Chamberlain.[6c] Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1941.

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10. Bintcliffe, E. W.: Mesenteric Vascular Occlusion, Brit. M. J. **1**: 50-53, 1947. Brown.[1a] McClenahan and Fisher.[1c]

11. d'Abreu, F., and Humble, J. G.: Mesenteric Venous Thrombosis: Recovery after Resection with Heparin, Lancet **1**: 534-536, 1946.

Artificial Respiration.—The restoration of breathing and of efficient blood circulation invariably is an emergency procedure. . . . One minute after cessation of breathing, if artificial respiration is given, the chances for survival are excellent, assuming of course, that the patient's heart and circulation are in good condition. After two minutes of asphyxia the chances for survival are less, but still excellent, but when five minutes have elapsed the chances for recovery are small—only about 25%. As more time passes, even though artificial respiration has been performed effectively at each of these stages, the chances for recovery grow less and less, and by the twelfth minute of asphyxia they are practically absent.—Cecil K. Drinker, M.D., Physiologic Principles of Resuscitation and Oxygen Therapy, *Postgraduate Medicine*, June, 1952.

PROPYLTHIOURACIL AND METHIMAZOLE THERAPY

COMPARATIVE EXPERIENCES

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and
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It is the purpose of this paper to report the results of the use of methimazole ("tapazole") in 45 patients and propylthiouracil in 54 patients in the management of hyperthyroidism at the Indiana University Medical Center and the Indianapolis General Hospital. All these cases were followed by one or more of us. They were treated in the outpatient thyroid clinics unless the severity of toxicity or complications necessitated admission to the hospital.

The diagnosis of hyperthyroidism in these cases was based on the characteristic features of the history and the results of physical examination and the usual laboratory studies. In the majority of cases three basal metabolism tests were made prior to treatment. Radioactive iodine (I^{131}) uptake with excretion studies and protein-bound iodine determinations were also made in many cases. Generally the patients were seen every two weeks in thyroid clinics, but in some cases the visits were every four weeks. A white blood cell and differential count were done at each clinic visit, and in most instances a basal metabolism test was also performed.

DOSAGE

It was our experience that the original daily recommended dose of 75 to 150 mg. of propylthiouracil was inadequate. If the clinical degree of hyperthyroidism was moderate or severe, our policy has been to use 300 mg. for small goiters, 400 to 500 mg. for those of moderate size, and 600 to 800 mg. for large goiters, estimated to weigh 250 gm. or more. We have arbitrarily used 15 mg. of methimazole daily when the goiter was small, 30 to 45 mg. daily when the goiter was of moderate size, and 60 mg. daily when the gland was large. Propylthiouracil has been given in three equally divided doses during the day. We have stressed that the methimazole should be taken in equally divided doses every eight hours. Iodine was not used concomitantly except in the occasional patient with severe hyperthyroidism in the propylthiouracil series. In occasional cases of severe hyperthyroidism in which iodine had been given prior to the propylthiouracil therapy, the iodine therapy was continued. When patients were euthyroid, strong iodine (Lugol's) solution was given for two or three weeks prior to surgery. In most cases the antithyroid drug was continued until the time of operation.

COMPARISON OF CASES

Table 1 summarizes the clinical data for the patients receiving propylthiouracil and for those receiving methimazole. A greater percentage of patients had received either iodine or propylthiouracil prior to treatment, and the clinical classification of severe hyperthyroidism was relatively greater in the group receiving methimazole. Otherwise the two series were similar, except that the incidence of nodular goiter in the group treated with propylthiouracil was greater than that in the group treated with methimazole.

Table 1.—*Summary of Data for Patients Given Propylthiouracil and Those Given Methimazole*

	Propylthiouracil	Methimazole
Total no. of patients	54	45
No. carried to euthyroid state	43	35
No. lost to the clinic before reaching the euthyroid state	3	4
No. not yet euthyroid	0	4
No. in which treatment was discontinued	7	2
No. hyperthyroid at time of surgery	1	0
Average age of patients, yr.	48.7*	46.2†
Sex incidence	F, 43 M, 11	F, 40 M, 5
Type of goiter		
Nodular	36	27
Diffuse	18	18
Average duration of goiter, yr.		
Nodular	19.2	17.2
Diffuse	1.8	3.5
Average duration of symptoms, mo.	15.8‡	15.5§
Clinical classification as to degree of hyperthyroidism		
Severe	13	29
Moderate	32	14
Mild	9	2
Average basal metabolic rate prior to therapy, %	+46.3‖	+58.8¶
Previous therapy, no. of patients (euthyroid)		
None	33	17
Iodine	5	7
Thiouracil	1	1
Propylthiouracil	4	4
Iodine and propylthiouracil	0	5
Unknown	0	1

* Range, 21 to 74 years.
† Range, 19 to 74 years.
‡ Based on 41 cases.
§ Based on 39 cases.
‖ Range, +20% to +93%.
¶ Range, +21% to +100%.

Table 2 summarizes the data concerning dosage and the time required before a euthyroid state was reached in the two groups of cases. The average daily dose of methimazole, 38.8 mg., was essentially one-tenth the average daily dose of propylthiouracil, 355.4 mg. For the 43 patients treated with propylthiouracil and followed to a euthyroid state, it required 3.2 days of treatment for each unit of elevation in the basal metabolic rate. In the 35 patients controlled with methimazole therapy, the time required was only 1.5 days per unit. In five patients to whom iodine was administered before propylthiouracil, both the daily dose of the drug and the time required to reach the euthyroid state were less than the average for the entire propylthiouracil group. However, in seven who received iodine prior to methimazole, both the dose and the time for control were increased above the average for that series. Previous propylthiouracil or thiouracil therapy caused a slight reduction in

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Methimazole was supplied by Dr. Don Carlos Hines of Eli Lilly & Company.

From the Department of Medicine, Indiana University Medical Center and Indianapolis General Hospital.

the daily dose and the time before euthyroidism was reached in the group receiving propylthiouracil, while there was a significant increase in both values in the group receiving methimazole.

Table 3 summarizes the toxic effects of and the unsatisfactory responses to treatment with the two drugs. The incidence of significant toxic reactions was similar for both drugs. One of the important differences between the two drugs was the 16.6% incidence of a slow response to propylthiouracil therapy as against no instances of slow response to methimazole therapy. In addition, an analysis of all patients followed to the euthyroid state revealed that 15 patients receiving propylthiouracil required four months or longer to be controlled, whereas only three patients treated with methimazole required four months or longer for control.

UNSATISFACTORY RESPONSE TO PROPYLTHIOURACIL THERAPY: REPORT OF CASES

The main objection to the use of propylthiouracil was that some patients required an extended period of treatment with 500 to 800 mg. daily before symptoms were controlled, whereas others failed to reach a euthyroid state with this dose. Not many reports in the literature stress this finding.

Case 1.—A 31-year-old man with a diffuse goiter (estimated weight of 200 gm.) had a severe degree of hyperthyroidism. Both the goiter and the symptoms were of approximately two years' duration, and he had received strong iodine solution for two months prior to admission. He was given 600 mg. of propylthiouracil daily for 85 days, and then the amount was increased to 800 mg. daily for 258 days. Treatment with the iodine solution was not continued. At the end of this time there was still considerable hyperthyroidism and thyroidectomy seemed inadvisable. I^{131} was administered with satisfactory control.

Table 2.—*Summary of Data with Respect to Doses and Time Required for Attainment of Euthyroid State with Propylthiouracil and with Methimazole Therapy*

	Propylthiouracil	Methimazole
Average daily dose*	355.4 mg.	38.8 mg.
Average no. of days to euthyroid state†	148.6	85.6
Patients without previous therapy		
Average daily dosage, mg.‡	365	27.1
Average days to euthyroid state	153.5	72.1
Patients treated with iodine previously		
Average daily dose, mg.§	302.9	47.9
Average no. of days to euthyroid state	129	104
Patients treated with propylthiouracil or thiouracil previously		
Average daily dose, mg.‖	351	42.1
Average no. of days to euthyroid state	136	113
Patients treated with both iodine and propylthiouracil previously		
Average daily dose, mg	0	33.7¶
Average no. of days to euthyroid state	0	84
Definitive treatment		
Surgery	30	19
Medical maintenance	11	9
I^{131}	7	3
Changed to methimazole	3	..
No definitive treatment	0	4

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* Range, 150 to 800 mg. (propylthiouracil) and 15 to 75 mg. (methimazole).

† Range, 14 to 434 days (propylthiouracil) and 30 to 182 days (methimazole).

‡ Thirty-three [illegible] and 17 cases (methimazole).

§ Five cases [illegible] ven cases (methimazole).

‖ Five cases in each group.

¶ Five cases.

Case 2.—A 55-year-old woman with a large nodular goiter and marked toxicity received an average daily dose of propylthiouracil of 388 mg. for 341 days. At the end of this period she still had severe hyperthyroidism. There had not been any previous iodine therapy, and initially she was given 500 mg. of propylthiouracil daily for 35 days. Because of rather severe leukopenia, the dose was reduced to 300 mg. daily for 172 days. Then the amount was gradually increased to 400 mg. for 45 days, then 500 mg. for 62 days, and, finally, 600 mg. for 27 days. She then received 60 mg. of methimazole for 182 days, at which time she was considered to be euthyroid, and a subtotal thyroidectomy was done. The surgical specimen weighed 350 gm.

Table 3.—*Summary of Toxic Effects of and Unsatisfactory Responses to Propylthiouracil and Methimazole Therapy*

	Propylthiouracil	Methimazole
Total no. of patients treated	54	45
No. of patients with toxic manifestations	5 (9.3%)	3 (6.6%)
No. of patients in whom treatment was discontinued because of toxic manifestations	2 (3.7%)	2 (4.4%)
Reason for discontinuance		
Agranulocytosis	1	0
Fever and rash	1	1
Nausea and vomiting	0	1
Toxicity manifestations when treatment was not discontinued		
Leukopenia (i. e., less than 4,000)	3	1
Unsatisfactory response to treatment	9 (16.6%)	0
Treatment discontinued	5 (9.3%)	

Case 3.—A 21-year-old man with a large diffuse goiter (estimated weight, 200 gm.) and severe hyperthyroidism presented the complication of rheumatic heart disease with mitral stenosis and congestive heart failure. He had been treated by his local physician with 300 mg. of propylthiouracil daily for four months and then with strong iodine solution alone for an additional three months prior to admission. We administered 600 mg. of propylthiouracil plus 30 drops of strong iodine solution daily for 103 days, without improvement. Then methimazole was given, and the hyperthyroidism was controlled after 126 days of treatment with an average daily dose of 45 mg.

Case 4.—A 73-year-old woman with a large nodular goiter (estimated weight, 250 gm.), severe hyperthyroidism of six months' duration, and congestive heart failure received 600 mg. of propylthiouracil daily for 120 days, with little improvement. There had been no previous iodine or antithyroid therapy. She made satisfactory progress when 60 mg. of methimazole was given daily.

Case 5.—A 50-year-old woman with a small recurrent diffuse goiter (estimated weight, 60 gm.) was admitted with extreme hyperthyroidism. She had received strong iodine solution for a few days prior to admission. Iodine was given intravenously after admission, and oral administration of strong iodine solution was continued. She received 300 mg. of propylthiouracil daily for eight days, then 450 mg. daily for 25 days, and, finally, 600 mg. daily for eight more days. There was slight improvement evident, but the patient was still critically ill, with fever, diarrhea, and congestive heart failure. All medication was discontinued, but the fever persisted. Radiation therapy was then given over the thyroid without improvement. I^{131} was given cautiously in small multiple doses and resulted in a satisfactory response.

Case 6.—A 72-year-old woman with a large nodular goiter and severe hyperthyroidism received 500 mg. of propylthiouracil daily for 405 days before the clinical and laboratory findings of control were obtained. There was no history of previous iodine therapy.

Case 7.—A 60-year-old woman with a small diffuse goiter had severe hyperthyroidism of about three months' duration. She had received strong iodine solution prior to antithyroid therapy. We administered 300 mg. of propylthiouracil daily for 225 days. The dose was then increased to 400 mg. daily for an additional 134 days before the euthyroid state was reached. Iodine was not used concomitantly with the antithyroid therapy. It was felt that this dosage was adequate since the gland was small. The surgical specimen, estimated to be 80% of the thyroid, weighed 28 gm.

CASE 8.—A 59-year-old woman with a nodular goiter (estimated weight, 200 gm.) required an average daily dosage of 450 mg. of propylthiouracil for 277 days before control was obtained. This patient had hypertensive heart disease with congestive heart failure, and the added problem of prolonged hyperthyroidism was unfortunate.

CASE 9.—A 42-year-old woman with a small nodular goiter required an average daily dose of 408 mg. of propylthiouracil for 212 days before reaching a euthyroid state. She had received strong iodine solution prior to antithyroid treatment, and the iodine therapy was stopped when the latter was instituted. The surgical specimen weighed only 40 gm.

In summary, there were nine cases of the 54 (16.6%) in which the response to propylthiouracil was not satisfactory. In five of these cases treatment with the drug was discontinued because of the slow response. In the remaining four a prolonged period of treatment was required before control was reached.

COMMENT

The potency of methimazole and relatively quick control of symptoms with its use have led to its popularity as an effective antithyroid drug. In reporting our series of cases in which methimazole was used, we have compared the results with those obtained in a similar group in which propylthiouracil was used as a method of testing the newer drug's efficacy. The propylthiouracil study extended over a period of four years, whereas the methimazole study covered 17 months. Although additional factors will become evident with longer use, several things are worthy of notice at this time. The value of an antithyroid compound is based on its ease of administration, rapid induction of control, and freedom from toxic reactions. The proved toxicity of former drugs has pressed the search for a more potent one,[1] with the hope that the rapidity of response would be increased and the toxicity decreased.

The dosage of methimazole required is about one-tenth that of propylthiouracil, as reported by Bartels and Sjogren,[2] Hallman and Bondy,[3] and Beierwaltes.[4] That the amount required for control of symptoms may be more than initially recommended [5] is indicated by our average daily dose of 38.8 mg. (table 2). The average time required for patients treated with methimazole to reach a euthyroid state was 85.6 days and for propylthiouracil it was 148.6 days (table 2). The rate of fall of the basal metabolic rate was about twice as rapid with methimazole as with propylthiouracil therapy. As a general rule the subjective improvement in the patient was prompt with methimazole treatment, a delay being seen in those patients with large nodular goiters and severe toxicity. Other recent reports concerning the use of methimazole include those of Taylor and co-workers,[6] who treated 23 patients; Kendrick and co-workers,[7] who treated 32 patients; and McGavack,[8] who treated more than 50 patients.

Propylthiouracil therapy has been unsatisfactory at times because of the extremely slow response of some patients to large doses. As did Bell and Mishtowt,[9] we encountered patients who had been treated inadequately. In these latter patients the administration of adequate doses under close supervision led to good results. The cases reported above indicate that our patients had received what are generally accepted as large doses of propylthiouracil over prolonged periods and failed to respond or responded slowly. The dose ranged from 400 to 800 mg. daily. In four of our patients (cases 6, 7, 8, and 9), the time of treatment ranged from 212 to 405 days before hyperthyroidism was controlled. Propylthiouracil therapy was discontinued in two patients (cases 1 and 2) after an average of 342 days. In the remaining three patients (cases 3, 4, and 5), the seriousness of the hyperthyroidism and the slowness of response to treatment prompted the discontinuation of propylthiouracil therapy and the institution of other therapy. It is interesting to speculate as to whether a daily dose of 1,000 mg. of propylthiouracil would have produced a satisfactory response in these cases and whether this amount would have increased the incidence of toxicity.

Our cases are too few to permit us to draw significant conclusions concerning the effect of previous iodine therapy on antithyroid drug action. In those patients who had received iodine prior to propylthiouracil therapy, the daily dose of the drug and the time required for them to reach the euthyroid state were less than the averages for the entire group treated with propylthiouracil (table 2). Five of the patients with unsatisfactory responses to propylthiouracil therapy had received iodine previously; iodine therapy was continued in two and discontinued in three patients. The data for the group treated with methimazole show that there was an increase in the dose requirement and the time interval for control in those who had received iodine before treatment (table 2), and in none of these cases was iodine therapy continued during the antithyroid therapy. In our experience, previous iodine therapy did not influence the effect of the antithyroid drug so much as did the size of the goiter and the degree of toxicity. A longer time was required to control severe hyperthyroidism in patients with large goiters in spite of large doses of antithyroid compounds.

Toxic reactions occurred in 9.3% of the group treated with propylthiouracil, cessation of treatment being necessary in 3.7% of the patients. In the group treated with the other drug the incidence of toxicity was 6.6%, treatment with the drug being stopped in 4.4% (table 3). This latter figure agrees with the rate of toxic reactions reported by Bartels and Sjorgen.[2] One case of serious agranulocytosis has been reported.[10] It is realized that a wider experience than ours is needed to permit one to determine the incidence of toxicity due to methimazole therapy. Hines[11] reported that he has now received reports on approximately 1,300 patients treated with methimazole. He found the total incidence of side-effects to be 4.6%, as compared with an over-all incidence of 2.7% in about 2,000 patients treated with propylthiouracil. The incidence of serious side-effects, such as agranulocytosis and fever, was almost the same in the group treated with methimazole as in the group treated with propylthiouracil, whereas the incidence of skin rash and urticaria was higher in the patients treated with methimazole. It is our opinion that methimazole, like other antithyroid drugs, should not be used without careful clinical observation.

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[1] Stanley, M. M., and Astwood, E. B.: 1-Methyl-2-Mercaptoimidazole: An Antithyroid Compound Highly Active in Man, Endocrinology **44**: 588-589 (June) 1949.

[2] Bartels, E. C., and Sjogren, R. W.: 1-Methyl-2-Mercaptoimidazole: New Active Antithyroid Agent, J. Clin. Endocrinol. **11**: 1057-1062 (Oct.) 1951.

[3] Hallman, B. L., and Bondy, P. K.: Experience with Methimazole (Tapazole) in the Treatment of Hyperthyroidism: A Report of 35 Cases, Am. J. Med. **11**: 724-729 (Dec.) 1951.

[4] Beierwaltes, W. H.: 1-Methyl-2-Mercaptoimidazole and 1-Ethyl-2-Mercaptoimidazole: A Clinical Evaluation of Two New Antithyroid Drugs, J. Lab. & Clin. Med. **36**: 861-865 (Dec.) 1950.

[5] Reveno, W. S., and Rosenbaum, H.: Treatment of Hyperthyroidism with 1-Methyl-2-Mercaptoimidazole, J. A. M. A. **143**: 1407-1408 (Aug. 19) 1950.

[6] Taylor, N.; Teitelbaum, M., and Large, A.: Treatment of Thyrotoxicosis with Methimazole (1-Methyl-2-Mercaptoimidazole), Am. J. M. Sc. **222**: 138-141 (Aug.) 1951.

[7] Kendrick, R. L.; Balls, K., and Rose, E.: Treatment of Thyrotoxicosis with 1-Methyl-2-Mercaptoimidazole, A. M. A. Arch. Int. Med. **89**: 368-373 (March) 1952.

[8] McGavack, T. H.: Thyroid, St. Louis, C. V. Mosby Company, 1951.

[9] Bell, G. O., and Mishtowt, G. I.: Use and Abuse of Thiouracil Drugs, Am. J. Med. **10**: 68-76 (Jan.) 1951.

[10] Croke, A. R., and Berry, J. W.: Agranulocytosis Occurring During Methimazole ("Tapazole") Therapy: Report of a Case, J. A. M. A. **148**: 45-47 (Jan. 5) 1952.

SUMMARY AND CONCLUSIONS

1. Forty-five patients with hyperthyroidism were treated with methimazole ("tapazole"), and 54 were treated with propylthiouracil. Results in the two groups are compared.
2. In the group treated with propylthiouracil there was a significant incidence of unsatisfactory response.
3. Methimazole is about 10 times more potent than propylthiouracil, although the dose used was higher than that originally recommended.
4. The time required for control of symptoms with methimazole therapy was about 60% less than that required with propylthiouracil therapy.
5. The size of the goiter and the degree of toxicity are important factors in the amount of antithyroid drug required and the time necessary for control of symptoms.
6. The toxicity due to methimazole approximated that due to propylthiouracil.

1040-1232 W. Michigan St.

[11] Hines, D. C.: Personal communication to the authors.

EXSTROPHY OF BLADDER

OBSERVATIONS FROM EIGHTY CASES

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and
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Exstrophy of the bladder is a rare but tragic anomaly. In 1947 Higgins[1] stated that the incidence of malformation was 1 in every 50,000 births. In his series of 41 patients, 33 were males and 8 were females. In our own material there were 62 males and 18 females. The therapeutic attack on this disease includes these objectives: (1) to cure the incontinence by producing a receptacle that can receive urine and discharge it at frequent intervals at the will of the patient, (2) to remove the sensitive everted bladder mucosa and to repair the abdominal wall defect, and (3) to correct deformities of the genitalia in both male and female by plastic procedures. It is not necessary to attempt to give bony union between the two sides of the pelvis, for over a period of years sufficient fibrous stabilization occurs to give the patient a satisfactory gait.

This communication is a study of the Boston Children's Hospital cases observed during the last 20 years. There have been 80 patients, of whom 54 were treated surgically. Portions of this material have been reported on previous occasions. The entire series is summarized here.

CLINICAL PICTURE

Exstrophy of the bladder is a regional anomaly. The urethra and bladder are open anteriorly, the latter showing as a midline bulging mass in the lower abdomen. Below the umbilicus there is a defect in the abdominal wall, muscles, and fascia through which the bladder protrudes forward. The exstrophied bladder changes in size as the intra-abdominal pressure varies when the child strains or cries. On the lower part of the everted mass of membrane one can see the two small elevated papillomatous projections from which urine comes in intermittent discharges from the ureteral orifices. The membrane tends to become raised up in cauliflower-like heaps around these lateral ends of the trigone. The exposed mucosa is very sensitive, and touching or rubbing any portion of it will make the child wince or cry out with pain and discomfort. This hypersensitivity gradually decreases over the course of several years as the transitional epithelium changes to a squamous form. This transformation tends to begin at the periphery of the bladder, becoming most prominent in the superior and lateral parts. The trigone and tissues immediately surrounding it show little of this change.

In the male there is usually a complete epispadias, with the verumontanum and the ejaculatory ducts widely exposed in the posterior portion of the open urethra. The penis has a flattened shape, is somewhat shortened, and has an upward curve. The scrotum is usually normal but in occasional cases is bifid. In the female there is a midline cleft of the vulva and clitoris. The urethra appears as a shortened groove or sulcus. In some cases the lower third of the vagina is open anteriorly, exposing the cervix. Children thus afflicted are constantly wet by the continuous extravasation of urine. The skin of the lower abdomen, groin, medial parts of the thighs, and buttocks tends to become irritated, macerated, or ulcer-

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Some illustrations have been omitted from The Journal and will be found in the authors' reprints only.

From the Surgical Service of the Children's Hospital and the Department of Surgery of the Harvard Medical School.

[1] Higgins, C. C.: Transplantation of the Ureters into the Rectosigmoid for Exstrophy of the Bladder: Review of 41 Cases, J. Urol. 57: 693, 1947.

ated, depending on the care the child has received. These miserable youngsters have about them a uriniferous odor that becomes more pronounced as they grow older.

There is usually no umbilical depression in the abdominal wall. This flattening of the navel area gives the abdomen a peculiar appearance. While the rectus muscle bellies are widely separated in their lower half, some degree of diastasis recti is usually found between the navel and the ensiform process. The fascia in the midline is widened and thickened above the exstrophied bladder. Lacking a central fusion, the two bodies of the pubic bone are widely separated. This separation has been said to be an invariable characteristic of exstrophy of the bladder, but this is not always true. Higgins[1] described a patient in whom the symphysis was intact and a normal penis was present. Since the wings of the ilia are spread, the hips are wider than normal. The symphysis is greatly widened or attenuated; therefore patients so affected have an unstable pelvis and a waddling gait. After reaching walking age, they also have a rather characteristic posture; that is, they bend forward in an effort to keep their clothes from rubbing against the sensitive bladder mucosa and the irritated skin of their groins and thighs.

Children born with exstrophy of the bladder may have associated anomalies, some of which are incompatible with life. In our series we have found the following malformations:

Inguinal hernia	16
Imperforate anus	6
Undescended testicle	7
Spina bifida	7
Omphalocele	5
Rectovesical fistula	3
Rectoperineal fistula	2
Meningocele and myelomeningocele	3
Double penis	2
Bicornate uterus	1
Syndactylism	1
Supernumerary finger	1
Ectopic kidney	1
Congenital heart disease	1
Umbilical hernia	1
Clubbed foot	1
Preauricular sinus	1
Deformity of left leg with bifid femur and absence of tibia	1

EMBRYOLOGY AND PATHOGENESIS

In 1901 Hart[2] summarized the work of Kolliker, Graf Spee, Lieberkuhn, Keibel, and others. Since then Von Geldern,[3] Bremer,[4] and others have reported their studies on the anomaly of exstrophy of the bladder and epispadias. Bremer feels that in the formation of the urinogenital organs the primitive plate (that is, either the so-called cloacal membrane or the primitive groove) is made up of ectoderm and entoderm and extends from the anus up to and including the lower border of the umbilical cord. Normally, mesodermal masses enter from the sides, meet in the midline between the two layers of the primitive plate, and supply the substance from which the anterior abdominal wall is formed. Bremer and others believe that, when the mesoderm fails to meet in the midline, the abdominal wall will be deficient and the primitive plate will persist as a very thin and weak membrane that splits or cleaves as the embryo grows, leaving the deformity known as exstrophy of the bladder.

Complete epispadias has been explained as a malformation of the genital tubercle that has been brought about by the failure of ingrowth of the mesodermal masses from above downward. Under these circumstances the genital tubercles join inferiorly along the ventral surface but do not join dorsally. This failure to coalesce on the dorsal surface leaves a small, flat, upward-curved penis, with the urethra exposed throughout its length. In those occasional instances of bladder exstrophy accompanied by a communication with the alimentary tract that opens in the exstrophied bladder,[5] the fistula is explained on the basis of either of two aberrations in development. Not only is there exstrophy, produced as described above, but in addition there has been a failure of growth of those mesenchymal masses that normally come in and divide the cloaca into a rectum behind and a urogenital sinus in front. In other words, there is a persistence of the cloacal duct.

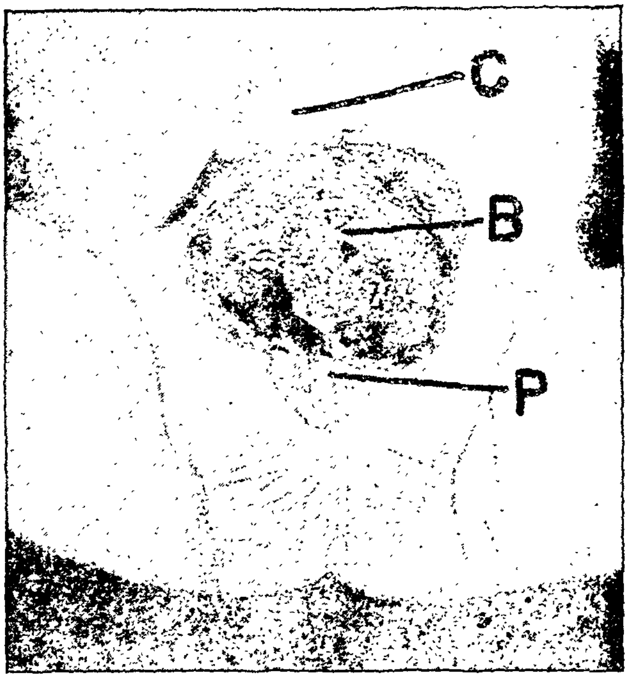

Fig. 1.—Exstrophy of bladder in 1-day-old boy: (*C*) cord stump, (*B*) bladder, (*P*) penis with epispadias.

TREATMENT

Much can be said regarding the historical developments in surgical therapy for exstrophy of the bladder. We shall not mention all the investigators who contributed to our surgical knowledge by proposing procedures that have since been discarded or by devising techniques that have now been accepted as a part of standard treatment but shall present here only the program that we feel is the best approach to the therapy of exstrophied bladder.

Young[6] has reported the cure of urinary *incontinence* by inversion of the exstrophied bladder and repair of the vesical sphincter muscle in several stages. Our attempts at this have been so disappointing that we have abandoned them in favor of transplantation of the ureters to the sigmoid, utilizing the rectosigmoid as a urinary reservoir. This attitude is now almost universally shared by workers in this field.

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[2] Hart, D. B.: A Contribution to the Morphology of the Human Urinogenital Tract, J. Anat. & Physiol. 35:330, 1901.

[3] Von Geldern, C. E.: The Etiology of Exstrophy of the Bladder, Arch. Surg. 8:61 (Jan.) 1924.

[4] Bremer, J. L.: Personal communication to the authors.

[5] Johnston, T. B.: Exstroversion of the Bladder Complicated by the Presence of Intestinal Openings on the Surface of the Exstroverted Area, J. Anat & Physiol. 48:84, 1913.

The age at which ureters should be transplanted to the bowel gives room for discussion and for honest difference of opinion. Higgins [1] and others have favored an early age for performing this operation, believing that the externally draining ureter commonly gives rise to ascending ureteral infection and kidney damage. This is in distinct contrast to our observations, which show that such ascending infection is exceedingly rare. Furthermore, transplanting ureters from the abdominal wall to the bacteria-laden bowel can hardly be regarded as a method of protecting a ureter from infection. While it is technically possible to establish ureterocolic anastomoses in babies, it is our present opinion that it is better to defer such surgery until an age at which the child has learned habits of bowel continence. It will then be easier for him to continue anal continence after the fluid material is turned into the bowel. Furthermore, our performance of anastomosis in older children has now become almost devoid of surgical complication, a statement that cannot always be made by those who perform these anastomoses in babies. For these reasons we defer transplantation until the patient is 3 or 4 years of age. Our results thereby have been so satisfactory that there is little desire to change this policy. With simple measures the child can be kept comfortable until this age for surgery.

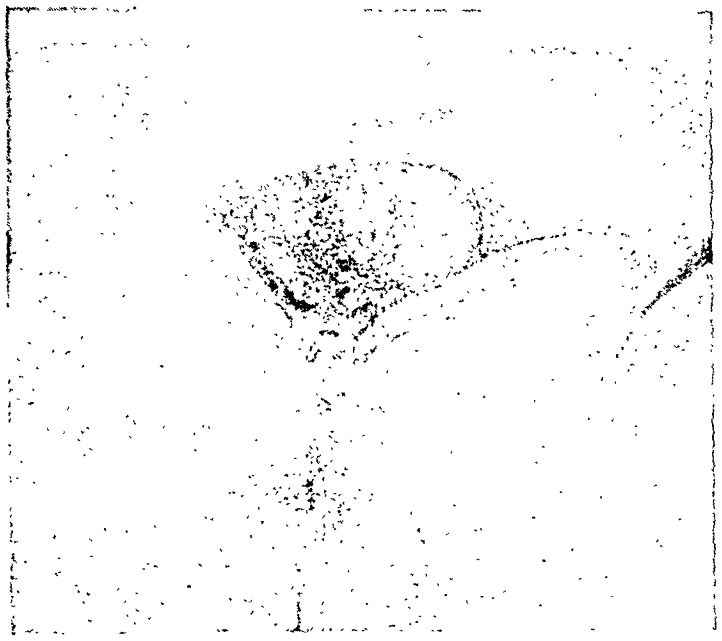

Fig. 2.—Exstrophy of bladder in 2-year-old girl. Note cleft condition of vulva, also bifid clitoris with vaginal orifice in full view. Squamous epithelium is replacing transitional epithelium on some of the exposed bladder surface.

Preoperative Care.—Babies and young children with exstrophy of the bladder can be managed so that they are reasonably comfortable and are not an undue burden on their parents. Frequently the family has been told by some physician that the bladder must be kept covered by a sterile gauze or dressing. This is a serious error, because it imposes a heavy financial burden in the maintenance of sterile coverings that are completely unnecessary. The exposed bladder tends to bleed if rubbed by any rough material such as an ordinary diaper, and it is also very painful if thus irritated. We have found it cheap, easy, and quite satisfactory to use clean (not sterilized) pieces of ordinary bedsheeting to cover the exposed bladder. The mother can keep on hand small bits of this material, 3 or 4 in. square. As one is to be used, it is smeared with petrolatum or boric acid ointment and put directly on the bladder area, and the diaper is then placed over this. (This fine-mesh material is better tolerated than surgical gauze.) When it is necessary to change the diaper, a new piece of the sheeting coated with petrolatum is placed again on the bladder. Little or no effort need be made to clean the bladder.

Surgical Considerations.—Before operation all of these children should be studied by intravenous (or retrograde) pyelography to visualize the ureters and pelves and to ascertain any anomalies that might have surgical significance. For two or three days prior to the transplantation of ureters, the bowel should be prepared by feeding the patient a low-residue diet and also by giving him at least two soapsuds enemas per day. We have little faith in the use of antibiotics or bactericidal drugs for reducing the bacterial flora of the colon, thereby presumably making operations on the colon safer. It is believed that a simple, well-performed anastomosis that does not leak is far more important than any quantity of drugs in reducing the incidence of postoperative regional or peritoneal infection. While it was formerly our custom to transplant the ureters in two stages, during the last 10 years we have usually transplanted both of them at the same operation, and in no instance have we regretted doing this.

Without describing here the details of operative technique of implanting ureters into the colon, it is well to set forth and emphasize certain principles we believe to be fundamental: (1) a transabdominal exposure; (2) simplification of technique; (3) establishment of a long intramural, submuscular implantation of the ureter; (4) projection into the lumen of the bowel of only a very short segment of ureter in order to minimize subsequent scarring and obstruction; (5) adequate anchorage of the ureter in its tunnel, so that it cannot withdraw and give leakage or obstruction; (6) leaving of a small urethral catheter in the ureter during establishment of the anastomosis, so that the tunnel cannot be made too tight; and (7) omission of inlying ureteral catheters after operation, so that the incidence of ureteritis is diminished. Pyelitis and pyelonephritis are seldom caused by ascending infection alone; they usually follow ureteric obstruction. It is therefore highly important to make sure the operation is carried out in such a way that there will be no subsequent delay in the flow of the urine.

Recently there has been considerable discussion regarding the tunnel-like type of anastomosis within the bowel wall versus an end-to-side union that permits an accurate mucosa-to-mucosa suture.[7] Our results with the

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[6] Young, H. H.: Exstrophy of the Bladder: The First Case in Which a Normal Bladder and Urinary Control Have Been Obtained by Plastic Operations, Surg., Gynec. & Obst. 74: 729, 1942.

[7] Cordonnier, L. J.: Ureterosigmoid Anastomosis, J. Urol. 68: 276, 1950.

former method have been so satisfactory, provided it is performed in such a manner that no obstruction results, that we have seen no need to use the mucosa-to-mucosa union in routine cases. We have reserved the latter method for those rare instances in which the ureter is very large and is difficult to place in a colonic tunnel. When used in these circumstances, we have found the method quite satisfactory.

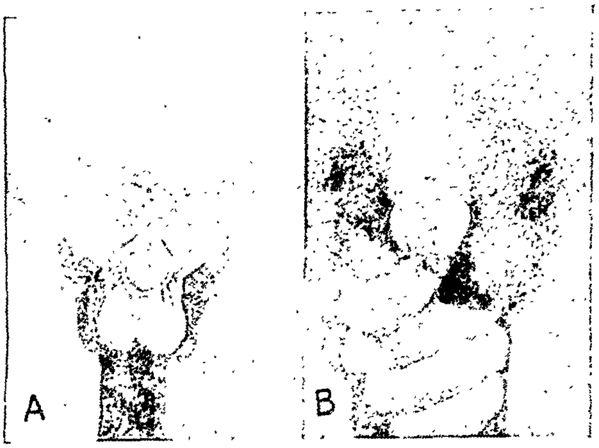

Fig. 5.—*A*, 8-year-old boy with exstrophy of the bladder, following transplantation of ureters into sigmoid, cystectomy, and repair of epispadias. *B*, same patient at age 17, with penis pulled down to show length and satisfactory appearance after plastic repair of epispadias.

While the exstrophied bladder can be removed at the time when ureters are transplanted, we prefer to leave this for a second operation some months later. Although the removal of a bladder is not difficult, in male patients the reconstruction of the penis is a meticulous and time-consuming job. If this is attempted at the time that the ureters are transplanted, there is too great a tendency to make haste and thereby give a poor reconstruction to the penis. We prefer to leave the removal of the bladder and the repair of the penis (or vulva) until a second operation, when there is more leisure to produce the best cosmetic repair of the genitalia. At the removal of the bladder and reconstruction of the penis or vulva, it is not necessary to remove all of the bladder tissue but only to excise the mucosa. The remaining wad of bladder musculature can be pushed inside the abdominal wall defect, and the rectus muscles and fasciae can then be suitably repaired. In the male a portion of the trigone is saved for the establishment of a small receptacle at the base of the urethra, which is then freed along its lateral edges and rolled in to the midline to make a tube (to transport semen). Over this tube the corpora and the portions of the glans are brought together from either side and are joined in a manner to bury the urethra. The skin is closed in the midline along the dorsum of the penis. This reconstruction, suggested more than 50 years ago by Cantwell and more recently emphasized by Young,[6] gives an excellent repair if the reconstruction is performed with delicacy and care. The penis so formed may be short, but it has erectile power and possesses a urethral tube running from the ejaculatory apparatus.

RESULTS OF TREATMENT

During the past 20 years we have observed 80 babies or children with exstrophy of the bladder, 54 of whom have been treated surgically. There have been no deaths, either at operation or at any time subsequent to hospital discharge. In the 54 children treated surgically, there were three cases of double bladders that were exstrophied and received no ureter. These bladders were excised. There were three patients in whom the exstrophied bladder was inverted and an attempt made to carry out the plan of Young's reconstruction. In one of these there was a fair result with respect to urinary continence; in the other two there was serious urinary incontinence, which subsequently led us to transplant the ureters into the rectosigmoid. In the remaining 48 patients there was no attempt to make a primary anatomic reconstruction of the bladder and urethra; the ureters were simply transplanted to the colon.

For the 50 patients with ureterocolonic transplants, 36 were done by two-stage procedures and 14 by one-stage bilateral ureterosigmoidostomies. In the early part of this series there were five instances of peritonitis. The affected children survived, but all have a nonfunctioning kidney on one side (in two, the kidney was subsequently removed). In addition there were six other patients with a nonfunctioning kidney that has caused no symptoms and has not been removed. We believe that most of these complications were due to the technical errors of establishing anastomoses that leaked or were too tight and led to ureteral obstruction. These convictions are strengthened by the fact that, as a result of greater attention to the operative principles above enumerated, there have been in the last 10 years no cases of peritonitis and only one of nonfunctioning kidney, and this has not had to be removed.

In the early part of this series there were many children in whom pyelitis developed postoperatively, making them extremely ill and difficult to treat. In the last decade such complications have become rare, a fact we attribute to the establishment of anastomoses that are free of obstruction and to the great effectiveness of antibiotics in aborting threatened attacks of pyelonephritis.

Rectal tolerance does not always develop at once but can usually be gained before the child is discharged from the hospital. Once tolerance to the presence of urine in the rectum is obtained, these patients can usually hold their urine and feces three to six hours during the day and need to get up only once at night. A child will still occasionally soil himself, however, especially when tired or emotionally upset. Proctitis has occasionally given us some concern in a patient. This is usually caused by acid

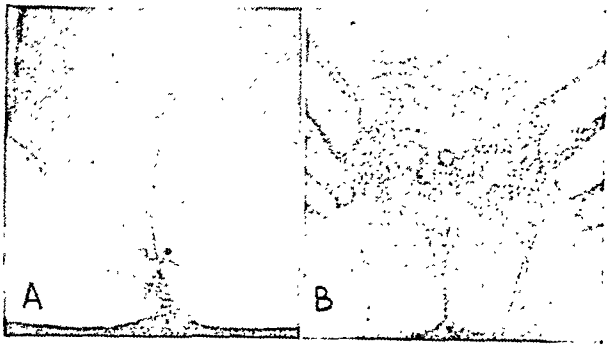

Fig. 6.—*A*, 7-year-old girl after bilateral ureteral transplants and cystectomy. *B*, same patient at age 13.

urine, and on alkalization by oral feeding of sodium bicarbonate the proctitis will generally subside rapidly.

A waddling gait is quite noticeable in the younger children. It is not unlike the gait associated with congenital dislocation of the hips. In older subjects the waddling can be controlled by the patient; in fact many parents have been able to teach their children to walk without any disturbance at all. In general, as these children get older, more stability of the pelvis is obtained, and the waddling is less apparent. No operative correction is necessary.

In our series postoperative pyelography has generally shown some degree of dilatation of the ureters and the calices of the kidneys for some months. It is now known that such dilatation is not necessarily permanent. The kidneys may not stabilize for two or three years, after which time some will return to normal while others will remain slightly dilated. In the last decade we have found that it is rare to find a progressive dilatation of the pelves and ureters. After the first six postoperative months, these patients should have intravenous pyelography two or three times a year for several years, or until stabilization of the urinary tract is observed. This is important in the handling of the case and in directing any additional therapy that might be required.

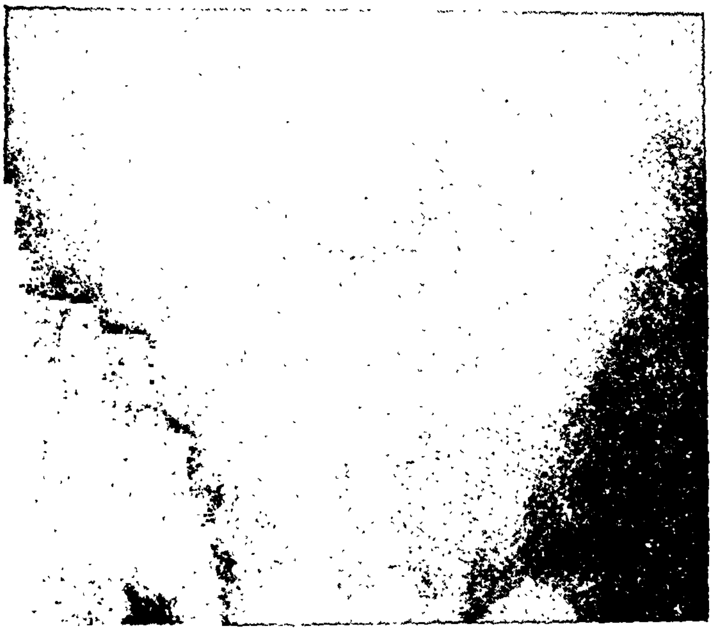

Fig. 9.—Intravenous pyelogram of 13-year-old girl, eight years after bilateral ureterosigmoidostomy. Patient has bilateral bifid renal pelves. There is excellent preservation of architecture of the pelves and calices.

Psychological problems in the female are not of great concern. If the operative steps are successful, the patient may be assured a relatively normal physiological and social life, and it will be possible for her to copulate and reproduce. In the male patient we are confronted with an entirely different disturbance, which is particularly troublesome during school years, when it is difficult for him to conceal the fact that he is different and must sit down to urinate. Parents and teachers who are cooperative and sympathetic can do much to ease this psychological tension. In our plastic repair of the epispadias we try to provide an adequate urethra and to reconstruct the penis so that the appendage is reasonably normal in appearance. After the plastic repair of the epispadias, the male patient should be assured that he can have a normal sexual life.

In 1950 Ferris and Odel [8] first reported blood electrolyte imbalance after the transference of ureters to the sigmoid. In a study of 141 patients who had undergone bilateral ureterosigmoidostomy for various conditions, it was found that various degrees of hyperchloremic acidosis and azotemia had developed in 79% of them. This evidence suggested that the change in electrolyte balance was due to absorption of the urinary solutes across the rectal mucosa from the urine stored in the sigmoid and rectum. The syndrome is manifested by fatigue, weakness, anorexia, loss of weight, salty taste, nausea, vomiting, nitrogen retention, polydipsia, and diarrhea. These symptoms are promptly relieved by correction of the acidosis.

Although Ferris and Odel [8] did not mention hypopotassemia as an accompanying factor, Diefenbach and his associates [9] reported a case with this added feature. In our 50 patients with ureteral transplants, we have recognized this syndrome in only one. It is our impression that such complications are much more likely to occur after ureteral transplantations in adults than in children.

SUMMARY

A report is made of 80 children born with exstrophy of the bladder, of whom 54 have been treated surgically. Some of the preoperative and postoperative problems and their treatment are presented. In the 50 patients treated by ureterosigmoidostomy, there have been no deaths. It was necessary to remove a kidney from two of these patients. It is urged that particular attention be given to providing the best cosmetic repair of the genitalia after the exstrophied urinary bladder has been removed. While there are many distressing features in treating this bladder and genital anomaly by employing the rectosigmoid as a urinary reservoir, the fact remains that the vast majority of the subjects can be given a comfortable existence and can assume a reasonably normal place in society.

300 Longwood Ave. (Dr. Gross).

[8] Ferris, D. O., and Odel, H. M.: Electrolyte Pattern of Blood After Bilateral Ureterosigmoidostomy, J. A. M. A. **142**: 634 (March 4) 1950.

[9] Diefenbach, W. C. L.; Fisk, S. C., and Gilson, S. B.: Hypopotassemia Following Bilateral Ureterosigmoidostomy, New England J. Med. **244**: 326, 1951.

Enlarged Cervical Lymph Node.—In the minds of many clinicians the removal of a cervical lymph node for diagnosis is a safe procedure with no more contraindications than laboratory examinations of the blood, spinal fluid, urine, feces, sternal marrow, etc., as the means of establishing the presence or the absence of a great variety of systemic disorders. In noncancerous diseases the procedures necessary to secure specimens for these various tests, including excision of a lymph node, are in themselves harmless and do not affect in any way the progress of the disease nor do they interfere with subsequent treatment. Should the disease be cancer, however, removal of a lymph node often is a serious and frequently an ultimately fatal handicap to overcome. Perhaps only the surgeon who treats cancer and follows his cases in sufficient numbers to determine end results will fully appreciate the serious consequences to the patient of the injudicious removal of a lymph node for diagnosis.—Hayes Martin, M.D., and Claude Romieu, M.D., The Diagnostic Significance of a "Lump in the Neck," *Postgraduate Medicine,* June, 1952.

CLINICAL NOTES

CHOLELITHIASIS AND PERFORATION OF THE GALLBLADDER IN AN INFANT, WITH RECOVERY

William H. Snyder Jr., M.D.
Lawrence Chaffin, M.D.
and
Leon Oettinger, M.D., Los Angeles

Cholecystitis with cholelithiasis is an unusual finding in infancy, but the condition has been reported several times in the literature of the last 200 years; however, it has always been an autopsy finding, and no case could be found that had been diagnosed during life and treated, with subsequent recovery, in an infant under 1 year of age.

REPORT OF A CASE

A 6-weeks-old female infant was admitted to the Los Angeles Childrens Hospital with a diagnosis of intestinal obstruction. At 2 weeks of age the baby began to be constipated. The scanty stools were described as white, yellow, green, and slimy. At 5 weeks of age, the abdomen began getting larger and became especially noticeable four days prior to admission. Bowel movements were now obtained only by enema. The appetite had been fair, but there had been intermittent vomiting for a week prior to admission. This vomitus contained only curdled milk.

On admission the baby appeared to be well developed, fairly well nourished, and acutely and chronically ill, with a markedly distended tight abdomen. The temperature was 100.6 F, and the blood pressure was 100/60 mm. Hg. Peristalsis was present but not remarkable. There was a suggestion of shifting dulness. The hemoglobin was 62% (colorimetric method), the white blood cell count 7,300, and the urine negative. A tentative diagnosis of abdominal malignancy with ascites was made and further studies were instituted.

Course.—Roentgenograms of the abdomen showed a moderately dilated small bowel with evidence of intraperitoneal fluid. A possible malrotation with chronic volvulus was considered, and a paracentesis was performed with removal of 400 cc. of yellow brown, slightly cloudy fluid. The possibility of a choledochus cyst was seriously considered. A barium enema revealed a normally placed cecum. Intravenous pyelograms were negative. The icteric index was 16, and cholesterol 118 mg. per 100 cc. of blood serum. A second tap allowed removal of 500 cc. of cloudy green fluid. The child took her formula quite well and vomited only occasionally. The temperature rose to 101 F, and on the sixth day after admission, surgery was performed. (Van den Bergh reactions were: direct, 2.3 mg. per 100 cc. of blood serum; indirect, 2.4 mg. per 100 cc.)

Surgery.—Under ether anesthesia, an upper abdominal right rectus incision was made. Blood was administered. Exploration revealed several hundred cubic centimeters of free bile-stained fluid between the coils of intestine. In some areas there were filmy adhesions with some pocketing. In the right upper quadrant was a perforated bile abscess cavity (fig. 2). The liver and loops of intestine had partly walled off this area. The liver itself appeared normal. At the base of the abscess was a perforation of the gallbladder at its junction with the cystic duct. The gallbladder contained one small stone, and there were numerous stones in the common duct. The duct was opened. A tiny probe could not be passed into the duodenum because of stones; some of these were washed out and an attempt made to pass a #8 catheter, but the duct was too small. A section of a #19 polythene® tubing was then inserted, which just fitted the lumen. Under syringe pressure the tiny stones were forced through into the duodenum, and the upper part of the duct was irrigated (fig. 2, inset). The polythene® tubing was inserted in the common duct for drainage. Penrose drains were put in place and the abdomen closed.

Postoperative Course.—Levine stomach tube section was instituted. Blood and fluids were administered intravenously; 300,000 units of penicillin and 150 mg. of terramycin were given daily. The child ran a febrile course for a few days. On the fifth postoperative day the polythene® tube was removed, since it was not draining properly. The Penrose drains were removed on the 10th day. The wound healed nicely with minor infection, and the child made an uneventful recovery. The slight jaundice cleared, and the stools have remained brown and normal. At the time of this report, three months after surgery, the child remains perfectly well.

Fig. 1.—The infant in case reported.

Additional studies showed that prothrombin activity was normal. Blood fragility of the baby, the mother, and the father were within normal limits. A cholecystogram of the mother showed good dye concentration and emptying.

COMMENT

The first reported case of gallstones in an infant was recorded by Lieutaud in 1767.[1] He found calculi in the gallbladder and ducts of a 25-day-old infant at autopsy. Since that time, about 90 cases have been mentioned in

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From the Surgical Service, Los Angeles Childrens Hospital, and the University of Southern California, School of Medicine, Los Angeles.

[1] Lieutaud, J.: Historia anatomico-medica, Paris, 1767, quoted by Potter, A. H.: Biliary Disease in Young Subjects, Surg., Gynec. & Obst. 66: 604-610, 1938.

the world literature,[2] all either found or confirmed at autopsy. Two authors verify their presence even in the fetus.[3] Since Potter's report in 1938, only one additional case can be found in the literature.[4] The fact that no previously reported case has been successfully treated is due, in part at least, to its rarity. Since the presence of gallstones is not suspected in infants, the condition is overlooked in the differential diagnosis.

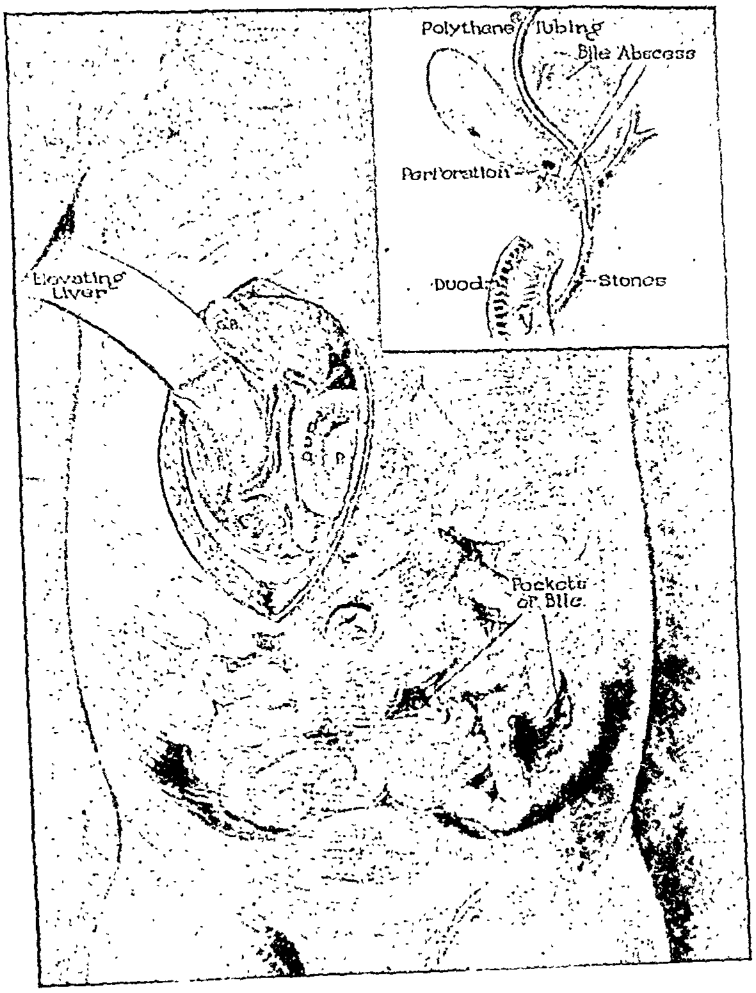

Fig. 2.—Drawing showing perforation of gallbladder at junction with cystic duct, stones in gallbladder and impacted in common duct, and bile abscess with perforation and generalized bile peritonitis.

Why it occurs is a matter of conjecture at the present time. The theories that have been advanced are not borne out by our case. Ladd and Gross [5] have suggested congenital hemolytic anemia as a basis for the formation of stones, but blood fragility tests of the baby, the father, and the mother were all within normal limits. Other conditions believed favorable to the formation of stones, such as biliary stasis, infection, and cholesterosis,[6] were apparently not present in our case. Kellogg [7] says "cholelithiasis in the newborn is due apparently to fetal pathology and is usually fatal."

The condition is known to occur in older children and young adults,[8] contrary to the classical concept of the disease as being one of middle and later life. There have been many reports of successful surgical treatment of gallbladder disease in this young group,[9] enough to emphasize the importance of considering it in a differential diagnosis, and of exploring for it when a laparotomy reveals no other demonstrable disease as a basis for the patient's symptoms. We wish to emphasize that it can also occur in infants and should be ruled out by surgical exploration, if necessary, in any infant with bizarre abdominal signs and symptoms.

There are other points, which this case brings out, that seem worth emphasizing. 1. Even though the true nature of the condition is not recognized preoperatively it can be successfully treated. 2. It provides a clinical illustration of the manifestations of common duct stones in infancy with final perforation of the gallbladder and bile peritonitis. In this case there was at first only constipation, then light colored stools, intermittent vomiting, and, finally, a distended abdomen with slight jaundice and bile-stained fluid, obtained on paracentesis. The course was gradual with increasing manifestations over a period of a month. There is evidence in this patient that bile was present in the free peritoneal cavity for a week before surgery. This seems to indicate that bile peritonitis without infection is well tolerated in the infant and even allows a nearly normal amount of peristalsis for many days. 3. It establishes a method of removal of stones within the infant's common duct by means of tightly fitting polythene® tubing accompanied by syringe pressure.

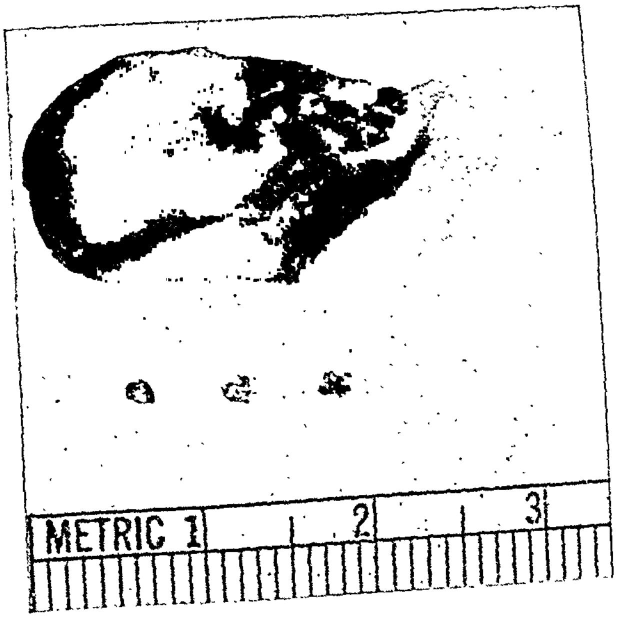

Fig. 3.—Specimens removed at operation.

SUMMARY

Cholelithiasis can occur in the infant and, if gallbladder disease is considered in the differential diagnosis, the condition can be successfully treated surgically. A method is suggested for the technique of removing common duct stones in the infant.

4614 Sunset Blvd. (Dr. Snyder).

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[2] Potter, A. H.: Biliary Disease in Young Subjects, Surg., Gynec. & Obst. 66: 604-610, 1938.

[3] Beale, L. S.: Slight Ailments, London, 1894, quoted by Potter.[2] Mitchell, quoted by Potter.[2]

[4] Spence, G. R.: Cholelithiasis in Newborn Infants, Arch. Pediat. 58: 479-481 (Aug.) 1941.

[5] Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1941, p. 287.

[6] Still, G. F.: Ciliary Calculi in Children, Tr. Path. Soc. London 50: 151, 1899, quoted by Potter.[2]

[7] Kellogg, E. L.: Gall-Bladder Disease in Childhood, Ann. Surg. 77: 587-593, 1923.

[8] Potter, A. H.: Gall-Bladder Disease in Young Subjects, Surg., Gynec. & Obst. 46: 795-808, 1928.

[9] Heringman, E. C., and Aiken, D. W.: Cholecystitis and Cholelithiasis in the Young, Am. J. Surg. 74: 27-32, 1947.

COMBINED INTERNAL AND EXTERNAL HERNIA WITH INCARCERATION OF BOWEL

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and

F. Eugene Roach, M.D., Lakewood, Ohio

The following case is being reported for two reasons. 1. Incarceration of the bowel caused by a congenital defect in the appendicial mesentery has not been previously reported; the possibility of the existence of such a defect is not mentioned either in the textbooks on anatomy and embryology or in the articles written on mesenteric defects. 2. The simultaneous occurrence of an internal and an external hernia is so rare that it should be recorded.

In his original lectures on the anatomy of the intestinal canal and peritoneum in man, Sir Frederick Treves,[1] in 1885, described the occurrence of defects in the mesentery and stated that although they may occur anywhere in the intestinal mesentery, most of them are found in the mesentery of the terminal ileum. Cutler,[2] in 1925, collected data on 28 cases of intestinal obstruction caused by mesenteric defects, all of which occurred in the terminal ileum. Dolton and Robison,[3] in separate, recent articles, reviewed the literature on mesenteric defects. Iason [4] discussed various types of hernia in children in an article in his book devoted to hernia. In neither these reports nor in standard textbooks of anatomy is the occurrence of a defect in the mesentery of the appendix mentioned.

REPORT OF A CASE

The patient was a 1-month-old boy who had been delivered and had developed normally until two days before admission. Two testicles had been palpated in the scrotum, and an inguinal swelling had not been noted previously. For two days before admission, the baby appeared to have intermittent abdominal cramps, which the mother attributed to "colic." Fourteen hours before admission, the baby passed a hard, constipated stool, and an irreducible mass appeared in the right side of the scrotum. On examination, the baby did not appear acutely ill, so the attending physician decided to observe the infant temporarily. Twelve hours later, the mass was still irreducible, and the baby appeared listless. He had vomited twice during the night. His temperature was 98 F and his pulse was 110 per minute. Immediate surgery was advised and performed.

While the patient was under ether anesthesia, the inguinal canal was exposed through the usual right inguinal incision. The fascia of the external oblique was split in the direction of its fibers and the cremasteric fascia opened. The mass still could not be reduced. Because of inflammatory reaction, the wall of the hernial sac was identified with difficulty and opened. A long loop of terminal ileum was taken from the scrotum. The medial arm of this loop was dark red, and there were small areas of necrosis in the fat of the mesentery. In the upper portion of the sac, the appendix was found lying transversely and *pointing laterally.* The loop of ileum had herniated through a hole near the base of the mesentery of the appendix and, strangely enough, from the lateral to the medial side of the mesentery. The edges of the hole were smooth. It was necessary to sever the blood supply to the appendix to release the bowel. With the administration of hot packs, the color of the bowel returned to normal, and peristalsis became evident. There were no defects in the mesentery of this loop. The bowel was replaced in the abdomen and the appendix removed without inverting the stump. The hernial sac was dissected free, the base closed with a purse-string suture, and the excess sac excised. It was then found that a cystic mass was still present in the scrotum. The mass was brought out to the wound and found to be a hydrocele. A partial removal of the hydrocele sac was done and the testis replaced in the scrotum. The cremasteric fascia was closed and the cord dropped into place. The external oblique fascia was closed, leaving sufficient room at the lower angle for exit of the cord. The subcutaneous fascia and skin were closed with interrupted sutures. Cotton sutures, no. 40 and no. 60, were used throughout the operation. The postoperative course was uneventful, and the patient left the hospital on the seventh postoperative day.

Microscopic examination of the appendix showed extensive extravasation of erythrocytes, with slight to moderate necrosis. This involved particularly the mucosa and submucosa. In other sections the mucosa showed only moderate neutrophilic infiltration. The muscularsis and serosa in all sections showed slight to moderate infiltration with neutrophils as well as smaller numbers of eosinophils and lymphocytes. In addition there was considerable edema. The final pathological diagnosis was hemorrhagic necrosis and acute inflammation of the appendix.

SUMMARY

A case of a simultaneous internal and external hernia with incarceration of bowel through a congenital defect in the mesentery of the appendix is reported. As far as can be determined, this is the first instance of its kind to be reported.

14805 Detroit Ave. (Dr. Thiessen).

APPARATUS FOR WEIGHING BED PATIENTS

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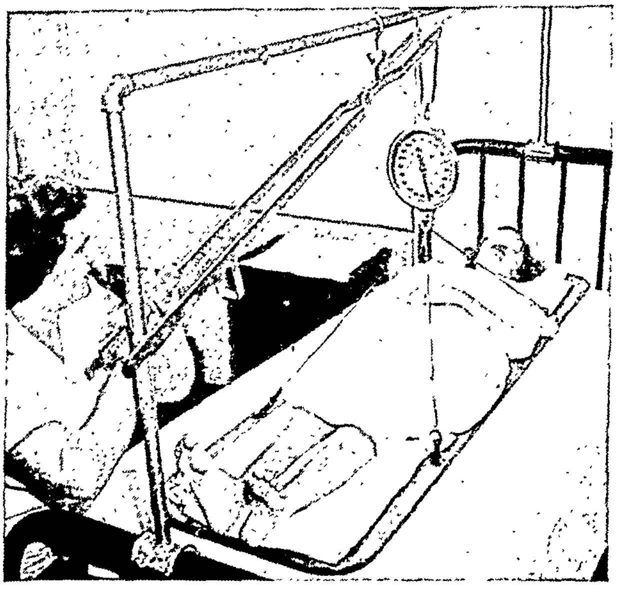
In several departments of a general hospital it is necessary to weigh patients who are confined to bed. The difficulty of doing this by methods usually available has been felt especially in the medical service in cases in which frequent weighing is necessary to determine water retention or loss as well as changes in nutrition. The ap-



Patient being weighed, showing operation of apparatus described.

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1. Treves, F.: Lectures on the Anatomy of the Intestinal Canal and Peritoneum in Man, Brit. M. J. 1: 470-474 (March) 1885.
2. Cutler, G. D.: Mesenteric Defects as a Cause of Intestinal Obstruction, Boston M. & S. J. 192: 305-311 (Feb. 12) 1925.
3. Dolton, E. G.: Mesenteric Defects, Brit. J. Surg. 31: 275-277 (Jan.) 1944. Robison, J. S.: Intestinal Obstruction: Consideration of an Infrequent-Type of Mechanical Obstruction of the Small Intestines Resulting from Herniation of the Bowel Through an Aperture in the Mesentery, J. Indiana M. A. 35: 295-303 (June) 1942.
4. Iason, A. H.: Hernia in Infancy and Childhood, Am. J. Surg. 68: 287-296 (June) 1945.

From the Department of Medicine, Hospital for Women and Children.

paratus illustrated in the figure was designed to fill this need. It was built in the maintenance shop of a hospital and required only materials readily available.

In use, the scales and lifting lever are hung over the patient's bed from any type of overhead frame. The lifting board, a 2 by 6 ft. sheet of ¾ in. plywood, is then slid under the patient, and the four lifting wires are attached to the corners of the board by snap hooks. The attendant raises the weighing apparatus by pulling down on the long lever arm, and when the patient is suspended an inch or two above the bed, reads the weight from the dial. The dial of the scale is adjusted to read zero when loaded with the board and lifting wires so that the patient's net weight is read directly. This apparatus has been found to be inexpensive, easily constructed, and accurate and safe in operation.

450 Sutter St. (8).

EXAMINATION OF HUMAN APPENDIXES FOR HISTOPLASMA CAPSULATUM

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Madelyn P. Miller, B.S.
and
Emil Kotcher, Sc.D., Louisville

The reports of Raftery[1] and Raftery, Trafas, and McClure[2] indicate a high incidence, 12.3% in children 16 years and younger and 5% in an unselected series, of organisms identified morphologically by them as Histoplasma capsulatum in patients' appendixes. These reports stimulated us to analyze our own material critically, since we had not noted H. capsulatum in our routine histological examinations of either appendixes or mesenteric lymph nodes. It was of special interest to us since we were dealing with patients in a presumably endemic area where disseminated cases are being recognized with regularity and the diagnosis substantiated by both histological and cultural methods without apparent difficulty. At the same time there is a high incidence, about 60%, of positive reactions to the histoplasmin skin test (Armstrong,[3] Christie and Peterson,[4] and Palmer[5]) in subjects who have no clinical evidence of the disease.

MATERIALS AND METHODS

Routine surgical specimens from a public and a private general hospital and a children's hospital were submitted to our laboratory in a fresh state, immediately after removal. One hundred of these were examined by direct tissue smear prepared with Leishman's stain and cultured on Sabouraud's dextrose agar to which had been added penicillin and streptomycin (20 units and 40 units per cubic centimeter, respectively) in order to inhibit bacterial growth. These techniques have proved satisfactory for the demonstration of H. capsulatum in experimentally infected white mice.[6] Cultures were kept for one month before being discarded as negative. Routine histological sections from this same group were stained with hematoxylin and eosin. Twenty-four were acutely inflamed, and 76 showed no active inflammation. None revealed H. capsulatum by any of these techniques. (During this period an appendix from a patient with previously proved generalized histoplasmosis was obtained at autopsy and revealed the organism by culture, tissue smear, and section.)

In addition to the above series, 1,307 appendixes (512 of them with acute inflammation and 795 of them without active inflammation) were sectioned and stained with hematoxylin and eosin in the usual manner. The lymphoid tissue was then carefully searched for organisms. H. capsulatum could not be found in any appendix of this series. Small chromatin masses were occasionally found in the reticulum cells. These masses were interpreted as being phagocytized nuclear fragments. None of the fragments had structure suggestive of Histoplasma.

COMMENT

In the work reported by Raftery[1] and Raftery, Trafas, and McClure[2] the identification of H. capsulatum was made on a morphological basis on examination of tissue sections. In only one instance did Raftery[1] substantiate the morphological identification by culture in artificial mediums to show the characteristic tuberculate chlamydospores of H. capsulatum. In the second report,[2] although 2,135 surgical specimens were examined, complete study (microscopic examination of sectioned tissue, culture on artificial mediums, and animal inoculation) was performed in only 21 "current" cases. In only one of these cases were organisms found that resembled Histoplasma morphologically in sectioned tissue. Material from this appendix reproduced the disease in mice, although this apparently was not confirmed by cultural isolation of the organism. Material from the appendix was cultured at room temperature, but only a sterile mycelium was produced, which did not show the characteristic tuberculate chlamydospores. Similar findings were reported from a single case of mesenteric adenitis.

We have no explanation for our failure to duplicate the findings of Raftery[1] and Raftery, Trafas, and McClure.[2] We believe our criteria of histoplasmosis are more rigid, since they involve not only the finding of structures morphologically similar to H. capsulatum in sectioned tissues but also cultural isolation of the organism from such tissues. Conant and co-workers[7] have pointed out the desirability of such confirmatory procedures.

There is, of course, the possibility that subclinical histoplasmosis may exist in various forms in different geographic localities. Perhaps in this area (Kentucky and Southern Indiana) the healed or dormant pulmonary form is the only common one. We have not seen any data, however, to suggest this possibility.

From the Departments of Pathology and Microbiology, University of Louisville, School of Medicine.

This investigation was supported by the Medical Research Fund of the Commonwealth of Kentucky.

[1] Raftery, A.: Subclinical Histoplasmosis: Gastrointestinal Histoplasmosis of Children, J. A. M. A. **145**: 216-220 (Jan. 27) 1951.

[2] Raftery, A.; Trafas, P. C., and McClure, R. D.: Histoplasmosis: A Common Cause of Appendicitis and Mesenteric Adenitis, Ann. Surg. **132**: 720-728 (Oct.) 1950.

[3] Armstrong, J. W.: Histoplasmosis: Study of Reactors to Histoplasmin, Am. J. Pub. Health **39**: 1136-1140 (Sept.) 1949.

[4] Christie, A., and Peterson, J. C.: Histoplasmin Sensitivity, J. Pediat. **29**: 417-432 (Oct.) 1946.

[5] Palmer, C. E.: Geographic Differences in Sensitivity to Histoplasmin Among Student Nurses, Pub. Health Rep. **61**: 475-487 (April 5) 1946. Histoplasma Capsulatum from Tissues of Experimentally Infected Mice, J. Bact. **62**: 613-620 (Nov.) 1951.

[6] Kotcher, E.; Robinson, J. W., and Miller, M. P.: The Isolation of

[7] Conant, N. F., and others: Manual of Clinical Mycology, Philadelphia, W. B. Saunders Company, 1944.

SUMMARY

One hundred appendixes (24 acutely inflamed and 76 "normal") were examined for Histoplasma capsulatum by culture on Sabouraud's dextrose agar, by tissue smears prepared with Leishman's stain and by histological sections stained with hematoxylin and eosin and found to be negative for this organism. An additional 1,307 appendixes (512 acutely inflamed and 795 "normal") were examined for structures morphologically similar to H. capsulatum by hematoxylin and eosin histological sections and found negative for this organism. One appendix obtained at autopsy from a previously proved case of disseminated, generalized histoplasmosis was found positive for H. capsulatum by culture, by stained tissue smear, and by hematoxylin and eosin histological sections.

101 W. Chestnut St. (2) (Dr. Christopherson).

PERIARTERITIS NODOSA

REPORT OF CASE WITH CHARACTERISTIC URINARY SEDIMENT

Leon R. Cole, M.D., Minneapolis

A type of urinary sediment believed to be characteristic of periarteritis nodosa and lupus erythematosus disseminatus was described by Krupp[1] in 1943. Only one report, that of Miale,[2] has appeared in the literature to confirm this observation. It is well recognized, of course, that hematuria is frequently present in periarteritis nodosa.

Krupp reported the presence in a single urine specimen of red blood cells, red cell casts, oval fat bodies, fatty and waxy casts, and, frequently, broad casts. There was also an abnormal amount of protein present. These findings were demonstrated in 14 of 21 cases of periarteritis nodosa and lupus erythematosus studied. The changes in the urinary sediment were found to vary with the state of the patient, being more extreme during clinical exacerbation and tending to disappear during remission. According to Krupp, red cell casts are the result of glomerulitis, and in acute glomerulonephritis they are the characteristic finding in the urinary sediment. Oval fat bodies and fatty and waxy casts reflect tubular degeneration and are the characteristic sediment of the nephrotic syndrome. Broad casts originate in the lower portions of collecting tubules, in which the flow of urine is slowed because the nephrons have had marked impairment of function. Broad casts are seen in renal failure, and in glomerulonephritis they are characteristic of the chronic stage. Addis[3] never observed the simultaneous presence of the above-mentioned types of urinary sediment in glomerulonephritis, though any two of the three types may be found at the same time. Hence Krupp considered this picture to be distinctive.

Krupp felt that he could correlate this urinary sediment qualitatively with the condition of the kidneys. Arkin[4] described four stages of activity of the lesions of periarteritis nodosa, ranging from acute inflammation to healing, and noted that all four may be present at the same time. Possibly the urinary sediment reflects this variable nature of the lesions.

The following case is reported in confirmation of Krupp's observation. The patient's clinical course was suggestive of periarteritis nodosa. Histological confirmation was lacking, however, as two muscle biopsies examined during life were reported as negative. The urinary sediment provided further evidence for the diagnosis.

REPORT OF A CASE

A. S., a 46-year-old woman, was admitted to University of Minnesota Hospital on Nov. 29, 1951, with a history of sore throat and cervical adenitis one month previously. Throat gargles were the only therapy, and symptoms disappeared in one week. Two weeks before admission puffiness of the face, feverishness, and occasional chilliness developed and persisted; for the last few days she had had dyspnea, orthopnea, and ankle edema.

Physical examination revealed an orthopneic woman with some periorbital and ankle edema. A few small tender lymph nodes were palpable at the angles of the mandibles. Ocular fundi were normal. Tonsils were enlarged. There was a right pleural effusion. The heart was slightly enlarged, and the blood pressure was 180/100 mm. Hg.

Urinalysis showed a specific gravity of 1.010, albumin of 2+, numerous red and white blood cells, red cell casts, oval fat bodies, fatty and waxy casts, and broad casts. Blood hemoglobin was 9.2 gm.; leukocyte count was 7,150 with 66% neutrophils, 31% lymphocytes, 1% monocytes, 1% eosinophils, and 1% basophils. Carbon dioxide combining power was 23 mEq., chlorides 114 mEq., and total serum protein 5.7 gm. per 100 gm. with 2.2 gm. albumin and 3.5 gm. globulin. Throat culture showed alpha and beta streptococci. Phenolsulfonphthalein excretion was 31% in 120 minutes. Quantitative 24 hour urinary protein was 13.5 gm. Chest film revealed slight cardiac enlargement, pulmonary congestion, and pleural effusion at the right base.

The patient was febrile during her entire hospital course. Her initial complaint was dyspnea, relieved somewhat by a thoracentesis. The leukocyte count was 14,650 on Dec. 4 and between 25,000 and 40,000 thereafter. Urinary output was good for the first 11 days, but the blood urea nitrogen level remained elevated. On Dec. 9 mild ascites and clinical signs of peritonitis developed. She received penicillin and aureomycin, but the occurrence of a generalized red macular skin rash caused the penicillin to be discontinued. Diagnostic paracentesis of the abdomen produced only a few milliliters of cloudy fluid, which contained many leukocytes with a differential count of 91% neutrophils, 7% mononuclears, and 2% eosinophils. The fluid was sterile. The evidence of peritonitis subsided somewhat. On Dec. 15 there was an onset of slight nuchal rigidity and flaccid weakness of most muscle groups of the right extremities. She was severely azotemic on Dec. 19 with a blood urea nitrogen level of 118 mg. per 100 cc. Treatment with the artificial kidney improved azotemia and acidosis; however, she was lethargic and had no clinical improvement. The blood urea nitrogen level rose again rapidly. She died from pulmonary edema on Dec. 26.

The autopsy was carried out by members of the department of pathology, University of Minnesota Hospital. There was cardiac hypertrophy with congestion of the lungs and liver. The right kidney weighed 270 gm. and the left 310 gm. Both kidneys were quite large, pale, and swollen, with numerous small subcapsular hemorrhages. The arterioles and medium sized arteries presented a similar over-all picture in most of the organs examined. These changes were most pronounced and variable in

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From the Department of Medicine, University of Minnesota Medical School.

[1] Krupp, M. A.: Urinary Sediment in Visceral Angiitis (Periarteritis Nodosa, Lupus Erythematosus, Libman-Sacks "Disease"): Quantitative Studies, Arch. Int. Med. **71**: 54-61 (Jan.) 1943.

[2] Miale, J. B.: Characteristic Urinary Findings in Visceral Angiitis (Periarteritis Nodosa, Lupus Erythematosus), Am. J. Clin. Path. **17**: 820-822 (Oct.) 1947.

[3] Addis, T.: Glomerular Nephritis: Diagnosis and Treatment, New York, The Macmillan Company, 1948, p. 156.

[4] Arkin, A.: Clinical and Pathological Study of Periarteritis Nodosa: Report of 5 Cases, One Histologically Healed, Am. J. Path. **6**: 401-426 (July) 1930.

the kidneys and were typical of periarteritis nodosa, mainly in the acute stage. Some of the glomerular capillaries showed similar changes. Some similar vascular changes were observed in the pulmonary septums. The brain was normal.

COMMENT

The antemortem diagnosis of periarteritis nodosa has been made in only a small proportion of the cases reported. Spiegel,[5] in 1936, noted that in only 12% of the cases in the literature had the antemortem diagnosis been made. This percentage has not increased, since Ward,[6] in a review of recent literature, found the diagnosis to have been made ante mortem in about 12% of instances. Millikan [7] states that periarteritis nodosa is disclosed by routine pathological examination in only 20 to 25% of biopsy specimens of patients ultimately proved to have the disease.

Involvement of the kidneys is frequent in periarteritis nodosa, as Baker [8] reported renal involvement in 80% of cases. All of Spiegel's 15 patients had renal lesions. Harris [9] reported involvement of the kidneys in 87% of 87 cases and the occurrence of uremia in 13%. Uremia developing in periarteritis nodosa, often described in the past, has been emphasized recently.[10] It would appear from the present case, considered in relation to the reports of Krupp and Miale, that the peculiar urinary sediment often encountered in this disease, although probably not pathognomonic, may be of considerable aid in diagnosis.

SUMMARY

A case of periarteritis nodosa is presented in which there was a type of urinary sediment described by Krupp[1] as being characteristic.

4319 Minnetonka Blvd.

5. Spiegel, R.: Clinical Aspects, Periarteritis Nodosa, Arch. Int. Med. **58**: 993-1040 (Dec.) 1936.

6. Ward, R.: Periarteritis Nodosa, in Brenneman, J.: Practice of Pediatrics, Hagerstown, Md., W. F. Prior Co., Inc., Publisher, 1948, vol. 3, chap. 13, p. 98.

7. Millikan, C. H.: Clinical Diagnosis of Periarteritis Nodosa, Dis. Nerv. System, **12**: 131-138 (May) 1951.

8. Baker, L. A.: Periarteritis Nodosa, with Report of 2 Cases, Ann. Int. Med. **17**: 223-235 (Aug.) 1942.

9. Harris, W. A.; Lynch, G. W., and O'Hare, J. P.: Periarteritis Nodosa, Arch. Int. Med. **63**: 1163-1182 (June) 1939.

10. Wold, L. E., and Barker, N. W.: Periarteritis Nodosa (Essential Polyarteritis): Clinical Data on 30 Cases Proved at Necropsy, Minnesota Med. **32**: 715-719 (July) 1949. Wainwright, J., and Davson, J.: The Renal Appearances in the Microscopic Form of Periarteritis Nodosa, J. Path. & Bact. **62**: 189-196 (April) 1950.

Preventive Medicine.—The most important concept of this century in the field of medical care is that preventive medicine is an integral and essential part of the everyday practice of medicine. It is not a separate body of knowledge to be understood only by a skilled technician who has been highly trained in this field. There is no "specialty" of preventive medicine as there is a specialty of surgery, radiology, or pediatrics. There is, of course, a specialty of public health, which is a well-organized body of knowledge and requires a high degree of skill. But preventive medicine must be interrelated with diagnosis, treatment, and rehabilitation. . . . The great physicians of all times have understood and have followed the principle that the practice of medicine is not simply an exercise in diagnostic acumen, nor a test of therapeutic skill, but requires a complete understanding of an individual as a person . . . who lives in an environment that has a profound influence . . . upon his health. They have also . . . followed the principle that he is entitled to the benefits of preventive medicine as an essential part of his medical care.—W. G. Smillie, M.D., Preventive Medicine and Public Health, ed. 2, New York, the Macmillan Company, 1952, p. 4.

ARTIFICIAL ELBOW FLEXION

George J. Boines, M.D., Wilmington, Del.

A mechanical device has been developed for artificial elbow flexion. It consists of a brace for the complete upper extremity, including the shoulder and the hand. The shoulder support is kept in place by means of a strap around the chest and under the opposite axilla. The hand mechanism, which appears fixed in the photograph, has been made movable at the wrist, so that the hand can be fixed in the pronated position or in the mid position by adjusting a screw at the wrist portion of the brace. The patient is able to flex the elbow at any desired position by swinging the arm forward. The elbow joint has several stops in flexion, with a release lever on the outside of the forearm. The hand may be turned into pronation or mid position by releasing screw catch at the wrist.

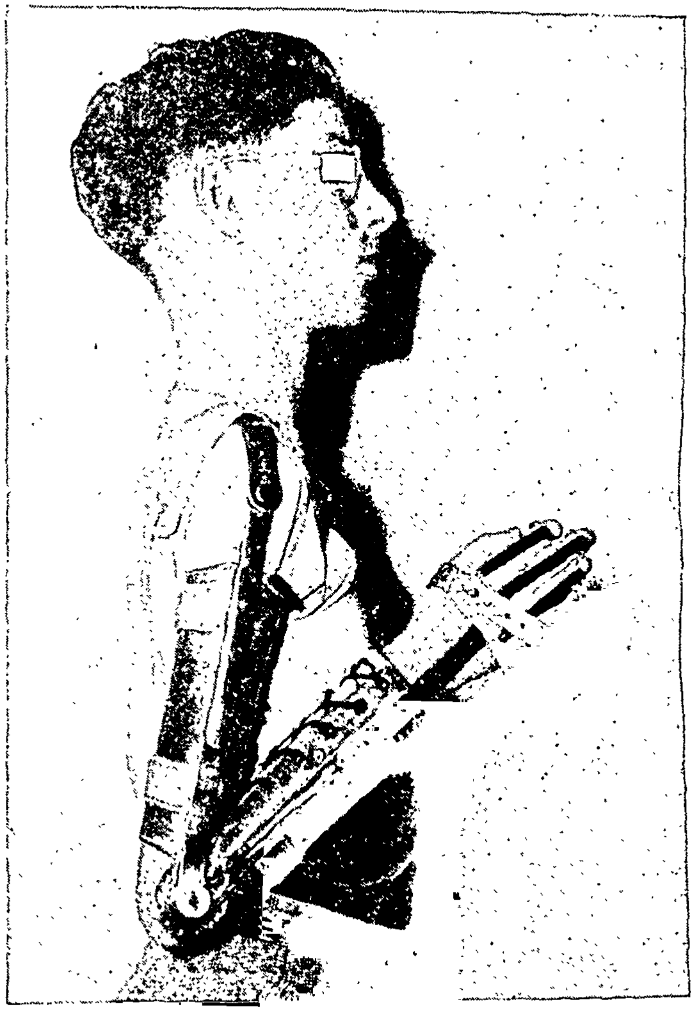

Fig. 2.—The right upper extremity is flail, with the exception of a trace of flexion power of the third and fourth fingers. The humerus is partly out of the socket. There is good power in the upper trapezius, rhomboids, supraspinatus, serratus anterior, latissimus dorsi, and levator scapulae. There is full range of motion of all joints.

The functional benefits of this device are as follows: 1. The flail extremity does not hang loose against the body. 2. Further subluxation of the head of the humerus is prevented. 3. When the patient is dressed and wearing a glove or has his hand in his trouser pocket, the disability is not apparent. 4. The patient is able to make

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Several of the illustrations have been omitted due to lack of space; they will appear in the author's reprints.

The brace was developed through the cooperation of the General Limb and Brace Company, 1916 Market St., Wilmington, Del.

Attending chief of communicable diseases, including poliomyelitis, Doris Memorial Unit of Wilmington General Hospital and St. Francis Hospital.

practical use of the arm adduction power against his body and thus hold objects. He cannot do this without the brace. 5. In school and at home when working on a flat top desk, he is able to hold paper and turn pages of a book by using a pushing motion in which power is transmitted from the shoulder and scapula muscles. He could not do this without the brace. 6. The patient is able to push and move objects with the hand by the transmission of power through the brace from his shoulder muscles.

Improvements desired include automatic methods of releasing the elbow joint latch and of flexing the forearm. The device is being modified to permit artificial flexion and extension (by means of a spring) at the elbow for the ambulatory patient with a flail upper extremity.

413 N. Broom St.

COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION

REPORT OF THE COUNCIL

The Council on Physical Medicine and Rehabilitation has authorized publication of the following report.

Ralph E. De Forest, M.D., *Secretary.*

ABSTRACT OF THE MINUTES OF THE ANNUAL MEETING

The Council on Physical Medicine and Rehabilitation held its Annual Meeting at the American Medical Association Headquarters in Chicago, on Dec. 14 and 15, 1951. The following Council members were present: Drs. Frank H. Krusen, Frank R. Ober, Morris A. Bowie, Anthony C. Cipollaro, Otto Glasser, Dean M. Lierle, George M. Piersol, Derrick Vail, Shields Warren, Arthur L. Watkins, and Walter J. Zeiter.

Dr. Krusen was reelected Chairman and Dr. Ober was reelected Vice-Chairman. In the field of education, it was reported that 72 questionnaires entitled "Teaching Program in Physical Medicine" were mailed to the deans of medical schools and 70 replies were received. As a result of this, a report was prepared entitled "Teaching Physical Medicine and Rehabilitation in the Medical Schools of the United States." It was planned to publish the article in The Journal. The activities of the Council's Advisory Committee on Education in relation to the development of the essentials for training schools for ancillary personnel in the field of physical medicine and rehabilitation was reviewed. It was voted that the Advisory Committee continue its work in considering a set of standards for such schools. The question of adequate training for those persons who do electrolysis was discussed. It was voted that the Secretary send a questionnaire to various schools of physical therapy and departments of dermatology in order to determine whether they would be interested in teaching persons to remove superfluous hair, thereby assuring training under the guidance of qualified medical personnel.

The activities of the Joint Committee on Rehabilitation formed by representatives of the Council on Physical Medicine and Rehabilitation and representatives of the Council on Industrial Health were discussed. The potential hazard inherent in the use of x-ray apparatus for the fitting of shoes was reviewed, as well as the activities of the Joint Committee in reviewing legislation regarding physical medicine and rehabilitation.

In a report regarding the dangers of radioactive isotopes, it was pointed out that the Atomic Energy Commission has been teaching persons how to handle them and that so far serious hazards have been avoided.

The activities of the Council and its Advisory Committee on Audiometers and Hearing Aids were reviewed. The need for reconsidering a standard procedure for evaluating the percentage loss of hearing and the problem of noise and health were considered.

The Council's cooperation with the Armed Forces, National Research Council, The American National Red Cross, and others in developing a new method of applying manual artificial respiration, the back pressure-arm lift method, it was reported resulted in the adoption of this method of manual artificial respiration by the previously mentioned organizations, as well as by this Council.

APPARATUS ACCEPTED

The following additional products have been accepted as conforming to the rules of the Council on Physical Medicine and Rehabilitation of the American Medical Association for inclusion in Apparatus Accepted. A copy of the rules on which the Council bases its action will be sent on application.

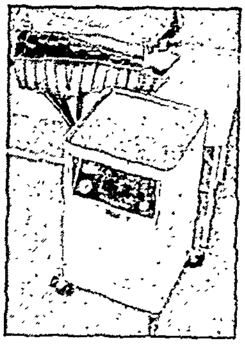
Ralph E. De Forest, M.D., *Secretary.*

Vaso-Pneumatic

Poor & Logan Manufacturing Company, 7319 Varna Ave., North Hollywood, Cal.

The Vaso-Pneumatic is an apparatus designed for use in the treatment of peripheral vascular disease. It consists essentially of two parts: one, a series of cuffs to be applied to the affected extremity of the patient; the other, a control cabinet which governs the distribution of pressure to the cuffs. Powered by an electric motor, the apparatus within the cabinet inflates and deflates the cuffs seriatim in such a way as to mimic a peristaltic wave passing in either the distal or the proximal direction.

Unpacked, the control cabinet measures 84 (height) by 48 by 48 cm. (33 by 18 by 18 in.) and weighs 59 kg. (130 lb.). Packed for shipment it measures 94 by 58 by 58 cm. (37 by 23 by 23 in.). A cradle for housing the rubber cuffs and supporting the patient's extremity is included in a separate carton which measures 15 by 23 by 71 cm. (6 by 13 by 28 in.). The combined shipping weights are 84 kg. (185 lb.).



Vaso-Pneumatic

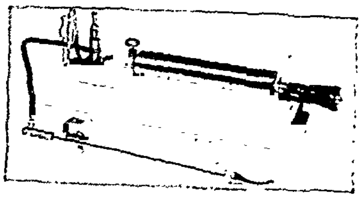
The motor requires a 60-cycle alternating current at 115 volts and draws 300 watts.

From acceptable sources the Council secured evidence which indicated that this apparatus was well designed and was useful in the treatment of certain types of peripheral vascular disease.

Gynogauge (Weisman)

Goodman-Kleiner Co., Inc., 5 E. 17th St., New York 3.

The Gynogauge (Weisman) is a modification of two forms of apparatus, the Gynographs, Models 1 and 2, previously accepted by the Council. It is a device for injecting carbon dioxide at controlled pressure into the uterine tubes. It comes in a carrying case that weighs, with contents, 2.8 kg. (6.2 lb.) and measures 40 (length) by 14.5 by 11.7 cm. (15¾ by 5¾ by 4⅞ in.).



Gynogauge (Weisman)

The Council obtained evidence which indicated that the apparatus was well made and performed satisfactorily under standard office conditions.

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A NEW LONG-ACTING ESTROGEN

The discovery that the synthetic compound diethylstilbestrol possessed high estrogenic activity when administered orally prompted a search for related compounds with a more prolonged activity. In 1942, Robson and his associates [1] reported that the triphenyl ethylene derivative diethoxy-triphenyl bromoethylene (*αα*-di [-*p*-ethoxyphenyl] *β*-phenyl bromoethylene), or D. B. E., showed a markedly prolonged estrogenic action in mice subjected to ovariectomy when given in doses somewhat greater than those producing a threshold response. The English investigators showed this prolonged effect was due to the fact that, on introduction into the body, D. B. E. in contrast to the estrogenic substances then available was not immediately destroyed or excreted in the urine but was stored in a number of tissues, particularly the fat, and was only gradually eliminated from the body.[2] It was further shown that this compound was a proestrogen, a term introduced by Emmens to describe compounds that do not act directly on the vagina but must be absorbed into the body and metabolized to a true estrogen before inducing their effects.[3] Preliminary clinical trials indicated that menopausal symptoms could be controlled with weekly maintenance doses of the drug following an initial large priming dose. Since results with carcinoma of the prostate were less encouraging, it was suggested that either the dosage employed was too low or the body was unable to convert proestrogen to estrogen at a rate sufficient to provide an effective concentration for controlling carcinoma of the prostate.[4]

In this country, Thompson and Werner have studied another triphenyl ethylene derivative, tri-*p*-anisylchloroethylene, or TACE.[5] They have shown that this compound, like D. B. E., is initially stored in body fat and is slowly liberated and metabolized, probably largely in the liver, to a much more potent estrogen than the parent substance. In addition they noted certain differences between the effect elicited by TACE and by the estrogen hexestrol when given to mice in doses of equal biologic potency, as judged by uterine and vaginal changes. Thus TACE, unlike hexestrol, did not cause enlargement of the pituitary gland. Furthermore, TACE caused only a slight increase, while hexestrol caused a marked increase, in the weight of the adrenals, and TACE resulted in less ovarian atrophy than hexestrol. While these findings suggest fundamental differences in activity between the two estrogens, further work will be required before this interesting possibility is established, for, as the authors acknowledge, it is difficult to define strictly comparable physiological doses between two compounds with such widely different durations of action. Preliminary clinical studies indicate that TACE is as effective as diethylstilbestrol in the palliative treatment of carcinoma of the prostate.[6] It has been suggested that adrenal hypertrophy may be one of the factors limiting the value of estrogen treatment of prostatic carcinoma. If this is indeed the case and if it can be proved that TACE controls prostatic carcinoma without affecting the adrenals, this drug might offer advantages over estrogens now in use.

REGISTRATION OF MEDICAL GRADUATES

The National Advisory Committee to the Selective Service System has issued an urgent appeal to all 1951 and 1952 graduates of medical schools who have not already done so to immediately register with their local Selective Service System boards under the provision of Public Law 779. Previous registration under the general selective service law, the committee warns, does not exempt male physicians under the age of 50 from registration under Public Law 779, nor does the fact that a physician is deferred as a regular registrant. The only male physicians who are not liable for registration under Public Law 779 are those who are currently members of reserve components of the Army, Navy, and Air Force. Previous military service does not exempt physicians from such registration unless they are currently members of reserve components of the armed forces.

Preliminary study of the 1952-1953 survey questionnaire of interns, residents, and fellows in hospitals, being conducted by the Health Resources Advisory Committee of the Office of Defense Mobilization, indicates that there is a considerable number of interns, residents, and fellows who are not members of reserve components who have not registered under Public Law 779.

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[1] Robson, J. M., and Schönberg, A.: A New Synthetic Oestrogen with Prolonged Action When Given Orally, Nature **150**: 22, 1942.

[2] Robson, J. M., and Ansari, M. Y.: The Fate of D. B. E. (*αα* Di-[-*p*-Ethoxyphenyl] *β*-Phenyl Bromo-Ethylene) in the Body, J. Pharmacol. & Exper. Therap. **79**: 340, 1943.

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[5] Thompson, C. R., and Werner, H. W.: A Comparison of Two Synthetic Estrogens, Tri-*p*-Anisyl Chlorethylene and Hexestrol, Federation Proc. **4**: 137, 1945. Thompson, C. R., and Werner, H. W.: Studies on the Fate of Tri-*p*-Anisyl Chlorethylene and Hexestrol, Federation Proc. **5**: 208, 1946. Thompson, C. R., and Werner, H. W.: Studies on Estrogen Tri-*p*-Anisylchloroethylene, Proc Soc. Exper. Biol. & Med. **77**: 494, 1951.

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The National Advisory Committee is also asking its state advisory committees to check with state boards of medical examiners to obtain the lists of all physicians granted licenses within the past two years. These will then be checked against Selective Service Public Law 779 registration lists to discover those physicians who are not registered in accordance with the law.

In issuing its statement, the National Advisory Committee called attention to the fact that failure to register as a special registrant makes such delinquents subject to investigation by the Federal Bureau of Investigation and liable to prosecution by the Department of Justice. It also pointed out that it believed that failure to register on the part of many 1951 and 1952 graduates has resulted from lack of understanding that they were required to register under both the general Selective Service System law and Public Law 779. Such failure the committee stated, however, is a specific violation of the Selective Service law.

HOUSE FLY RESISTANCE TO CHEMICALS

House fly resistance to DDT was first recognized in Europe shortly after the conclusion of World War II. Reports of house fly resistance to DDT in the United States were quite prevalent by 1947; by 1948 an alarming degree of DDT resistance was observed in California, and similar conditions were described by numerous investigators at scattered locations throughout the southern, eastern, and central United States. According to Decker and Bruce,[1] DDT and related materials such as methoxychlor are currently of little or no practical value in fly control in Illinois. With approximately 50% of the farms in the state harboring flies that are resistant to lindane and dieldrin, there is reason to believe that most if not all of the chlorinated hydrocarbon insecticides will have lost their usefulness within a year or two. In some sections of the United States where chlorinated hydrocarbons have been employed more extensively than in Illinois, conditions may be even worse.

In a previous communication,[2] the authors demonstrated that the initial processes of segregation and selection leading to establishment of high levels of resistance proceed slowly at first, but that, when resistance is once established, its intensification is rapid and proceeds to equilibrium with the toxicant found in the house fly's environment.

It has also been shown that contamination of breeding mediums contributes to development of DDT resistance under conditions prevailing in the field. DDT resistance appeared first and was intensified most rapidly on farms where sanitation was poor and where spraying operations resulted in serious contamination of manure and similar breeding mediums. Acquisition of tolerance for one chemical may predispose a strain of house flies to rapid development of resistance to some other chemical. In acquiring a high degree of resistance to DDT, flies also acquire a fair degree of resistance to methoxychlor but only a slight degree of resistance to lindane, chlordane, and dieldrin. These products apparently are closely related, and house flies resistant to any one of the three have a fair degree of resistance to the other two.

It seems possible, if not probable, that tolerance for insecticides may be extremely variable and may be induced by the independent or joint action of many factors. In some cases resistance is due to the house fly's ability to metabolize almost immediately or at least to decompose DDT into harmless components.[3] At the same time, there are some highly resistant strains in which DDT is not immediately decomposed and in which sizable amounts of the substance can be found in the blood and body tissues of living insects. In still other strains it appears that resistance or tolerance may be due largely to varied morphological characters or variations in habits rather than to chemical factors. It is therefore reasonable to expect that house fly strains of dissimilar origin produced under quite distinct environmental conditions will differ in their response to varied laboratory conditions.

Research on house fly resistance to chemicals is under way at many investigative centers, and, while the genetic aspects of the problem and the exact nature of resistance have not as yet been clearly defined, considerable progress is being made. There is reason to hope that better understanding of the variables encountered in resistance investigations will further the progress of research on house fly resistance to chemicals.

SALARIES OF OFFICE EMPLOYEES OF SOME PHYSICIANS FREED FROM CONTROLS

A recent amendment to the Defense Production Act apparently frees most physicians from the restrictions previously imposed on them as employers by wage and salary controls in the manner and amount they can compensate their office employees. The amendment in effect exempts from wage and salary controls "wages, salaries, or other compensation of persons employed in small-business enterprises," which the amendment defines as "any enterprise in which a total of eight or less persons are employed." Thus, physicians who employ eight or less persons in connection with the practice of medicine are freed from the confusing wage and salary controls and may now compensate and increase the compensation of their employees as they deem advisable.

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[1] Decker, G. C., and Bruce, W. N.: House Fly Resistance to Chemicals, Am. J. Trop. Med. & Hyg. 1: 395 (May) 1952.

[2] Bruce, W. N., and Decker, G. C.: House Fly Tolerance for Insecticides, Soap and San. Chem. 26: 122, 1950.

[3] Sternburg, J.; Kearns, C. W., and Bruce, W. N.: Absorption of DDT by Resistant and Susceptible Houseflies, J. Econom. Entomol. 43: 214, 1950.

THE PRESIDENT'S PAGE

A MONTHLY MESSAGE

Last month I stated that many persons regard the American Medical Association as a pressure organization devoted to the interests of industry and management. There are two reasons for this impression. One is that during the last four years the Association has had to conduct a campaign to protect the freedom of medicine. This has been highlighted to the exclusion, from the minds of the public, of the Association's scientific activities. The other reason, in my opinion, is that we have been remiss in not keeping the people informed of all we are doing to raise the health level of the public. This remissness on our part existed long before we engaged in any campaign. Now that the intensity of the campaign against socialized medicine is about at an end, and with the recent reorganization of our Public Relations Department, I believe neither of these reasons will any longer be a factor.

Perhaps one of the best ways to demonstrate the amount of work the Association devotes to the furthering of the scientific aspects of medicine will be to show how the Association spends its income. Figures are rather dry, but they do tell the story. I shall use round figures only, so as to render them less confusing.

The total income of the Association from dues (less the amount allocated to subscriptions to THE JOURNAL), investments, and miscellaneous sources was approximately $1,958,000 for 1951. The income from subscriptions to and advertising in our scientific publications was roughly $5,781,000. From this latter figure, however, must be deducted the costs of production, including printing. These costs were about $3,613,000, leaving a total income on publications of $2,168,000. This, added to the dues and investment income of $1,958,000, gives us a working income of $4,126,000.

The American Medical Association Headquarters occupies a nine story building covering about two-thirds of a city block. It houses nearly 900 full-time employees. For what may be termed administrative purposes, the costs are roughly $227,000. Added to this there is a sum of about $208,000 for such items as annuities and depreciation. The Association maintains biographical records of everyone in medicine from the time he enters medical school until he dies. From this record is compiled the Amedican Medical Directory. Membership records are also maintained. The Association has to a certain extent lent financial support to the Student American Medical Association. The total of these activities amounts to another $158,000. When all of these are deducted from available income, approximately $3,533,000 is left for other activities.

On straight scientific activities, such as those of the Councils on Medical Education and Hospitals, Pharmacy and Chemistry, Foods and Nutrition, Physical Medicine and Rehabilitation, Industrial Health, Rural Health, Health Education, the Bureau of Investigation (which deals with frauds and charlatanism), the Committee on Cosmetics, the Commission on Chronic Illness, the Survey on Medical Education, the Library, the laboratories, the Scientific Sessions (including the Scientific Exhibits), and extra costs of publication of pamphlets and books, the Association spends roughly $1,300,000. Closely associated with the purely scientific activities are the work of the Council on Medical Service and the Bureau of Medical Economic Research. They work constantly in the socioeconomic fields, such as hospital and medical care insurance, care of veterans, care of the indigent, labor plans, and distribution of physicians. The total cost of these activities is $280,700. The Council on National Emergency Service, which is concerned with problems of mobilization and cooperates with federal agencies, spent nearly $40,000. A half-million dollars was given to the American Medical Education Foundation as a contribution to help support our medical schools. Hence, on scientific and semiscientific activities the Association spent about $2,121,000.

The Public Relations Department, including the Press Bureau, spent approximately $209,000; the Washington Office, $180,500; and $51,500 was spent by the Bureau of Legal Medicine and Legislation, which analyzes and reports on all enacted federal and state legislation and court decisions affecting medicine.

We have accounted, therefore, for $3,154,000. This leaves us approximately $1,000,000 to account for. Nearly half ($443,000) was added to capital. The balance, slightly over $500,000, was allotted to the National Education Campaign, which is being brought to a close this year. The amount of $2,121,000, spent on scientific and related activities, does not represent the real total, as much of the money spent by the Public Relations Department and the Press Bureau was devoted to disseminating scientific information.

Members of the Association sometimes ask how their dues are spent. Of the $25.00 paid by each individual member, $15.00 is allocated to cover the cost of his subscription to THE JOURNAL. That leaves $10.00 for other purposes. It was formerly possible to run the Association on subscriptions, advertising, income from investments and Fellowship dues. Fellowship dues no longer exist, as the Fellowship category has been abolished, although $228,000 was received from that source in 1951. If, however, we take the total amount available in 1951 from advertising, subscriptions, investment income, and income from miscellaneous sources, we shall find that there was available only $2,403,000. That would have been insufficient to cover the costs of the scientific and semiscientific activities, administration, and the membership-biographical services, which totaled over $2,840,000. Merely on these activities alone we should have had a deficit of nearly a half million dollars.

Where, then, can we cut down on our expenses? For each of the past two years the Association has contributed $500,000 to the American Medical Education Foundation. It has also absorbed all the expense of administration of the Foundation, which in 1951 amounted to over $14,000. This has been done so that every dollar contributed by any physician would go to the medical schools. Shall we discontinue this and have our medical schools become dependent on federal aid with all of its dangers? Not that the half million dollars the Association contributes will in itself prevent it, but it is a help in the general campaign for private support of medical education.

The National Education Campaign, which in 1951 expended $529,000, will be brought to a close this year. Out of these funds was spent the cost of the Presidential inaugural broadcast (about $30,000), at the Annual Meeting in 1951. This broadcast has been termed one of the best, if not the best of the public relations activities of the Association.

If we allocate the $25.00 paid by the individual member in 1951, we shall find that it was distributed as follows: $15.00 went to cover the cost of his subscription to THE JOURNAL; $5.14 to the scientific and semiscientific activities; $1.06 to administration; $0.39 to biographical and membership records; $1.07 to public relations, the Washington Office, and the Bureau of Legal Medicine; $1.07 to the capital of the Association; and $1.27 to the National Education Campaign. In 1953 the amount allotted to the National Education Campaign will probably be divided among the other activities in a relative proportion.

That, then, is what becomes of a member's dues. Can anyone submit a legitimate complaint against such a distribution? The whole environment of medical practice in this country today, high-standard medical education, high standards in therapeutic agents and apparatus, high quality of hospitals, protection against fraudulent remedies and charlatanism, high standards of medical care, and the right to practice in a free state are due to your Association. In addition you receive the outstanding medical publication in the world. Can any member think of any way in which to spend $25.00 and obtain greater return for his money?

LOUIS H. BAUER, M.D., Hempstead, N. Y.

ORGANIZATION SECTION

ASSOCIATION EXHIBITS

Health exhibits for fairs and expositions and other public gatherings have been scheduled for numerous showings during the next two months by the Bureau of Exhibits. These exhibits are shown with the cooperation of the various state or county medical societies. Medical exhibits for medical societies and other scientific groups have also been reserved for several occasions. The schedule is as follows:

Aug. 5 to 8,	"Home Accidents," Rural Health Conference, Little Rock, Ark.
Aug. 6 to 9,	"Diseases Transmitted from Animals to Man," Trumbull County Fair, Warren, Ohio.
Aug. 8 to 17,	"Animals in Research," "Voluntary Health Insurance," and "Mechanical Quackery," Illinois State Fair, Springfield, Ill.
Aug. 10 to 15,	"School Health," Miami County Fair, Bradford, Ohio
Aug. 11 to 15,	"The Failing Heart," Pocahontas County Fair, Marlinton, W. Va.
Aug. 13 to 16,	"You and Your Body," Clark County Fair, Springfield, Ohio
Aug. 16 to 24,	"Testing the Drinking Driver," Missouri State Fair, Sedalia, Mo.
Aug. 17 to 22,	"You and Your A. M. A.," American Pharmaceutical Association Meeting, Philadelphia
Aug. 18 to 24,	"Home Accidents," Sioux Falls, S. D.
Aug. 21 to Sept. 1,	"Testing the Drinking Driver," Iowa State Fair, Des Moines, Iowa
Aug. 25 to 30,	"You and Your Body," Dutchess County Fair, Rhinebeck, N. Y.
Sept. 3 to 6,	"The Failing Heart," Highland County Fair, Hillsboro, Ohio
Sept. 6 to 12,	"Home Accidents" and "Appendicitis," Kansas Free Fair, Topeka, Kan.
Sept. 7 to 9,	"You and Your A. M. A.," New Hampshire State Medical Society Meeting, Bretton Woods, N. H.
Sept. 8 to 9,	"Testing the Drinking Driver," Indianapolis Police Department, Indianapolis
Sept. 8 to 11,	"Testing the Drinking Driver," Institute of Traffic Engineers, Chicago
Sept. 9 to 13,	"Animals in Research," National Chemical Exposition, Chicago
Sept. 14 to 21,	"Mechanical Quackery," Massachusetts Medical Society, West Springfield, Mass.
Sept. 15 to 18,	"Medical Education," American Hospital Association Meeting, Philadelphia
Sept. 15 to 19,	"Home Accidents and Appendicitis," Kansas State Fair, Hutchinson, Kan.
Sept. 16 to 19,	"You and Your Body," Washington County Fair, Fayettesville, Ark.
Sept. 16 to 27,	"Testing the Drinking Driver," Virginia State Fair, Richmond, Va.
Sept. 19 to 28,	"You Can Reduce," Atlantic Rural Exposition, Richmond, Va.
Sept. 21 to 25,	"Testing the Drinking Driver," International Association of Police Chiefs, Los Angeles
Sept. 22 to 26,	"Medical Education," American College of Surgeons Meeting, New York
Sept. 28 to Oct. 1,	"Mechanical Quackery," Virginia Medical Association, Richmond

IF YOU CHANGE YOUR ADDRESS

Your copy of THE JOURNAL is addressed to you several days in advance of the publication date; so if you change your address, please notify us at least three weeks in advance.

Send your old address together with the new, preferably clipping the name and address from your last copy of THE JOURNAL.

Copies mailed to your old address will not be forwarded by the Post Office, unless you provide the Post Office with the forwarding fee.

Send your change of address to:

American Medical Association
THE JOURNAL Subscription Department
535 N. Dearborn St., Chicago 10.

USE OF GAMMA GLOBULIN IN PREVENTION OF PARALYTIC POLIOMYELITIS

The following statement has been issued by the chairman of the Sub-Committee on Blood of the Health Resources Advisory Committee of the Office of Defense Mobilization:

Whether gamma globulin will be effective in the prevention of paralytic poliomyelitis is not now known. On the basis of animal experiments and preliminary study on humans, it is possible that globulin will have value in human poliomyelitis, but serious questions remain to be answered before such a hope can be substantiated. Nevertheless, public dissemination of information on the status and objectives of current studies, which have been incompletely presented or misunderstood, has created a serious demand for gamma globulin that cannot be met.

Virtually the entire output at current production rates is required to meet the demand for prevention or modification of the course of measles and infectious hepatitis. Under the circumstances, it is obvious that the existing limited supply and current production of gamma globulin should be reserved for use in the diseases in which its efficacy has been established.

SURVEY OF RESEARCH PROJECTS

The A. M. A.'s Committee on Research will send out questionnaires early in September to determine what medical research projects currently are in progress in the United States. The survey has a threefold purpose: to establish an up-to-date file of medical research projects; to evaluate the premise that certain fields of medical research are suffering from lack of financial support; and to consider the actual contribution of individual scientists in terms of free time and personal expenditure of funds. A random sample of 15,000 physicians from all parts of the country and selected personnel from medical schools, public health services, and pharmaceutical firms will be asked to participate in the survey.

COMMISSION ON ACCREDITATION OF HOSPITALS OPENS OFFICE

Director Edwin L. Crosby, M.D., former superintendent of Johns Hopkins Hospital, Baltimore, will open the new Joint Commission on Accreditation of Hospitals office Sept. 1 at 660 Rush Street, Chicago. The commission, with representatives from the American Hospital Association, the American College of Surgeons, the American College of Physicians, the Canadian Medical Association, and the American Medical Association, will assume responsibility for the hospital standardization program formerly carried out by the American College of Surgeons. The commission's program will get under way early this fall.

THE JACOBI FUND

The Jacobi Fund of the Section on Pediatrics received $1,696 from 282 contributors, according to the report made at the June meeting in Chicago by Hugh L. Dwyer, M.D., Custodian. Net income from investments was $188.76. Expense and distribution to essayists was $731.80. Trust value May 30, 1952, was $6,890.04.

MEDICAL NEWS

ARIZONA

Infant Health Campaign.—To stimulate the organization of community health councils and committees for promotion of health clinics, the *Arizona Farmer* in Phoenix and the First National Bank of Arizona are sponsoring a statewide rural infant health campaign. One $100 and two $75 awards will be made to three rural organizations that make major contributions to the cause of infant health in Arizona between now and May 31, 1953. One of the $75 awards will go to an Indian organization in Arizona, and these organizations also will be eligible for either of the other two awards.

CALIFORNIA

Suicides in Los Angeles.—Los Angeles had a higher average suicide rate for the 10 year period 1941-1950 than the State of California (9% higher) and the United States as a whole (67% higher). The southern states have the lowest rates. Of 354 suicides in 1950 for Los Angeles residents, 265 (75%) were men.

Dr. Lewis to Study Health Conditions in Iran.—In response to a request from Iran for help in putting into effect its legislation for the protection of the health of workers, the World Health Organization and the International Labor Organization have jointly appointed Dr. Leon Lewis of Berkeley to survey health conditions in factories in Iran and to advise the ministry of health on how they can be improved. Dr. Lewis was formerly head of the division of industrial hygiene in the School of Public Health, University of California, Berkeley. In Iran he will study conditions among workers in mines, handicrafts shops, and carpet, match, and tea factories.

DISTRICT OF COLUMBIA

The Gibson Award.—At its annual banquet in the Williamsburg Room of the Mayflower Hotel, Washington, D. C., May 17, the Washington Medical and Surgical Society presented the Frank E. Gibson Award and a cash prize of $25 to Dr. James H. Scully of Washington, D. C., who had given the outstanding scientific paper before the society during the past year. The subject of his paper was "The Treatment of Metastatic Malignancy from Breast Cancer."

Dr. Beattie Accepts Chicago Appointments.—Dr. Edward J. Beattie Jr., of Washington, D. C., has been appointed clinical assistant professor of surgery (Rush) on the faculty of the University of Illinois College of Medicine, Chicago, and will also serve on the medical staff of Presbyterian Hospital in Chicago. Dr. Beattie was an instructor in surgery at Harvard Medical School, Boston, for two years before going to London, England, as a Moseley Traveling fellow to study medicine and cardiology. On his return, he became affiliated with George Washington University in Washington, D. C., where for the past five years he has been assistant professor of surgery and director of the surgical research laboratories. He also was consultant in thoracic surgery for five Washington, D. C., hospitals and was a Markle scholar.

ILLINOIS

Hospital Celebrates Golden Anniversary.—The St. Joseph Hospital in Elgin will celebrate its 50th anniversary on Sept. 24. Sister M. Melitta is administrator of the hospital, which has a bed capacity of 150 for adults and children and 30 bassinettes. There will be open house from 2 to 8:30 p. m.

Health Officer Appointed.—Dr. Albert L. Hildinger of Omaha has been appointed medical director for the Jo Daviess County Health Department, with offices at Galena. Dr. Hildinger has had seven years of experience in public health work in Nebraska, serving as health officer for the Sarpy-Otoe-Nemoha Tri-County Health Department and for the Scottsbluff County Health Department. Previously he was in private practice in Omaha for 14 years.

Chicago

Northwestern University Retirements.—On Sept. 1 three physicians will retire from the staff of Northwestern University Medical School. Dr. Karl A. Meyer, professor of surgery since 1945, has been affiliated with Northwestern since 1925. Dr. Arthur R. Metz, associate professor of surgery, has been on its staff since 1942. Dr. Frederick W. Merrifield has been professor of oral surgery in the dental school and assistant professor of surgery in the medical school since 1940.

Reception Honoring Dr. Bernhard Zondek.—The Chicago chapter of the American Jewish Physicians' Committee will hold a dinner and reception at the Standard Club Sept. 2 in honor of Dr. Bernhard Zondek, professor of gynecology and obstetrics at the Hebrew University Medical School, Jerusalem, Israel. Members of the sponsoring committee include Drs. Max Thorek, Nicholas I. Fox, Sidney O. Levinson, Heinrich Necheles, Lester E. Frankenthal Jr., Jacob S. Golden, Hymen I. Sapoznik, Sidney A. Portis, Salamon Boros, Joseph T. Bolotin, and Maurice S. Mazel. Dr. Morris Fishbein will be the toastmaster.

KENTUCKY

Psychiatric Leader Honored.—Dr. S. Spafford Ackerly, head, department. of psychiatry, University of Louisville School of Medicine, and medical director of the Louisville Mental Hygiene Clinic, was honored at a party given by former students, psychiatrists, social workers, and others interested in mental hygiene. He was presented with a silver tray inscribed "for twenty years of unselfishly dedicated service to his friends, profession, students, university, city, and state." He was also given an outboard motor.

MICHIGAN

State Medical Meeting in Detroit.—The annual session of the Michigan State Medical Society will be held at the Sheraton-Cadillac Hotel, Detroit, Sept. 24-26, under the presidency of Dr. Otto O. Beck of Birmingham. The Andrew P. Biddle Lecture will be delivered by Paul de Kruif, Ph.D., Holland, at 9:15 Wednesday evening, as a feature of Officers' Night, when the meeting will be open to the public. Other guest speakers include: Dr. Gaylord W. Anderson, Minneapolis; Drs. Claude S. Beck, Spencer Braden, and George Crile, Cleveland; Dr. John J. Bonica, Tacoma, Wash.; Dr. Ormond S. Culp, Rochester, Minn.; Dr. Daniel C. Darrow, New Haven, Conn.; Drs. Edwin J. De Costa, Peter C. Kronfeld, Roland P. MacKay, Philip Thorek, and Leonard F. Weber, Chicago; Dr. Garfield G. Duncan, Philadelphia; Dr. Carl W. Eberbach, Milwaukee; Drs. Dwight E. Harken, Duncan E. Reid, Samuel F. Marshall, and Claude E. Welch, Boston; Dr. Milton I. Levine, New York; Drs. Earl R. Miller and David A. Rytand, San Francisco; Dr. Emil Novak, Baltimore; Dr. Ben H. Senturia, St. Louis; and Dr. Evan W. Thomas, Albany, N. Y. Three discussion conferences or quiz periods will be held Wednesday, Thursday, and Friday in the Grand Ballroom from 12 noon to 1 p. m. All guest speakers of the day will be present on the platform.

Thursday evening, State Society Night, will afford entertainment in the Grand Ballroom. On Wednesday the Michigan branch of the Academy of Pediatrics will have a dinner and business meeting at 6:30 p. m. in the English Room; the Michigan State Medical Society Section on Ophthalmology and the Detroit Ophthalmological Society will meet for dinner at 7 p. m. in the Pan American Room, and the Detroit branch of the American Urological Association will have a dinner at 7:30 p. m. in Suite 500, preceded by a reception at 6:30 p. m. in the suite assigned to

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Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

Dr. William Bromme, Detroit. "Upper Peninsula Day" will be celebrated with cocktails and dinner in parlors G, H, and I at 6 p. m.

On Thursday the Michigan Allergy Society will meet for dinner at 7:15 p. m., after which there will be a meeting at 8:30 p. m., addressed by Dr. Samuel M. Feinberg, Chicago, professor of medicine, Northwestern University Medical School. The Wayne University Alumni Association will hold its banquet at 6:30 p. m. in the English Room. The Michigan Regional Committee on Trauma, American College of Surgeons, will hold a meeting after its luncheon at 12 noon in the Pan American Room. The Michigan chapter of the American College of Chest Physicians will assemble for dinner at 7 p. m. in the Pan American Room. The directors of the Michigan Academy of General Practice will meet for dinner at 6 p. m. in Parlor F, and the Michigan chapter of the Arthritis and Rheumatism Foundation will hold a joint dinner meeting with the Michigan Rheumatism Society at 7 p. m. in the Founders' Room. The Michigan Pathological Society will hold a meeting from 2 to 4:30 p. m. in the Michigan Room of the Statler Hotel and will also have a dinner meeting in the same room at 6:30 p. m. This society will conduct a slide seminar on dermatological lesions, at which Dr. Elson B. Helwig of the Armed Forces Institute will act as moderator. On Friday the Michigan Society of Neurology and Psychiatry will hold a reception at 5:30 p. m. before its 7 o'clock dinner in the Pan American Room.

The Woman's Auxiliary to the Michigan State Medical Society will meet Sept. 22-25 at the Fort Shelby Hotel, Detroit, and the Michigan State Medical Assistants Society will hold its meeting Sept. 24-25 at the Tuller Hotel in Detroit.

MISSOURI

General Practitioners Study Club.—The following programs for 1952 have been arranged by the General Practitioners Study Club of Greater St. Louis:

Sept. 17, Roy V. Boedeker, Management of Abortion.
Oct. 15, Robert W. Bartlett, Differential Diagnosis and Treatment of Cancer of the Colon and Rectum.
Nov. 19, Maurice J. Lonsway, Newer Concepts in Pediatrics of Interest to General Practitioners.
Dec. 17, Garold V. Stryker, Common Skin Diseases.

Dr. Moore Heads Geographic Pathologists.—Dr. Robert A. Moore, dean, Washington University School of Medicine, St. Louis, and Edward Mallinckrodt professor of pathology, was elected president of the International Society of Geographic Pathology at its recent meeting in Liége, Belgium. The society is devoted to the study of geographic distribution of disease and discusses in detail one disease at each meeting. Epidemic jaundice was the topic at the Liége meeting; the next meeting in Washington, D. C., in September, 1954, will be concerned with cancer.

Dr. Moore also participated in a conference on lung cancer at the University of Louvain, Belgium, and, at the invitation of the United States Army, spent several days in Frankfurt, Germany, as consultant on pathology.

MONTANA

State Medical Meeting at Missoula.—The Montana Medical Association will hold its annual meeting at the Hotel Florence in Missoula, Sept. 18-21, under the presidency of Dr. Francis L. McPhail of Great Falls. Guest speakers will include Drs. Edward D. Allen and Lester R. Dragstedt, Chicago; Laurentius O. Underdahl, Rochester, Minn.; Roger W. Barnes, Los Angeles; John B. Grow, Kenneth D. A. Allen, and John C. Long, Denver; and Kenneth S. Landauer, New York. On Thursday and Friday at 12:15 p. m. there will be three panel discussions. Drs. Barnes and Edward Allen will be guest speakers at the panels on obstetrics, gynecology, and urology. These panels will be sponsored and conducted by the Montana Obstetrical and Gynecological Society. Drs. Dragstedt and Grow are guests for the panels in surgery, which will be sponsored and conducted by the Montana Chapter of the American College of Surgeons. The panels on internal medicine and radiology sponsored by the Montana-Wyoming Section of the American College of Physicians, will have as guest speakers Drs. Underdahl and Kenneth Allen.

Guest speakers at the banquet will be Dr. Louis H. Bauer, Hempstead, N. Y., President of the American Medical Association, who will discuss "Medicine, the Public and Citizenship," and O. Meredith Wilson, Ph.D., dean of the University College of the University of Utah, Salt Lake City, who will speak on "Western Regional Cooperation in Higher Education."

The Western Montana Medical Society will be host to members and guests of the Montana Medical Association at a fish fry Friday, 6 p. m., at the Missoula Country Club.

The woman's auxiliary will hold its annual meeting in Missoula at the Hotel Florence Sept. 18-19. Other organizations that will meet during the annual session include the Montana Urological Association, the Montana Obstetrical and Gynecological Society, and the Montana Academy of Oto-Ophthalmology. The Montana Physicians' Service will meet Sunday morning.

NEW YORK

Cancer Talks at Albany.—Albany Medical College announces that as part of the cancer teaching program Dr. Virginia K. Frantz of Columbia University College of Physicians and Surgeons, New York, will talk on "Carcinoma of the Thyroid" Sept. 26 from 12 to 1 p. m. Dr. J. Englebert Dunphy, associate professor of surgery, Harvard Medical School, Boston, will talk on "Observations on the Natural Course of Cancer" Oct. 24 from 12 to 1 p. m. These lectures at the college are open to the medical public.

Lectures on Occupational Medicine.—The faculty of medicine, Columbia University, announces a series of lectures on occupational medicine on Saturday mornings (6 to 10 a. m.) in Amphitheater A, College of Physicians and Surgeons, 630 W. 168th St., New York 32. Members of the medical and allied professions are invited. There will be no tuition fee.

The schedule is as follows:

Sept. 20, History of Occupational Medicine.
Sept. 27, Scope, Objectives and Practice of Occupational Medicine.
Oct. 4, Industrial Toxicology—General Principles.
Oct. 11, Toxic Metals I.
Oct. 18, Toxic Metals II.
Oct. 25, Toxic Solvents I.
Nov. 1, Toxic Solvents II.
Nov. 8, Toxic Dusts.
Nov. 15, Toxic Gases.
Nov. 22, Occupational Dermatoses and Infections.
Nov. 29, Ventilation, Lighting, Noise.
Dec. 6, Elementary Nuclear Physics; Radiation Hazards.
Dec. 13, Workmen's Compensation.

The order in which the topics are presented may be slightly modified depending on the availability of lecturers.

New York City

Hospital News.—Dr. Henry W. Kolbe, formerly medical superintendent of Harlem Hospital, has been appointed a general medical superintendent of the city department of hospitals, with over-all supervision of the 12 municipal hospitals in the boroughs of Brooklyn, Bronx, and Queens. Dr. Bernard B. Nadell, formerly medical superintendent of Lincoln Hospital, has replaced Dr. Kolbe as medical superintendent of Harlem Hospital, and Dr. Sander V. Smith, former deputy medical superintendent of Bellevue Hospital, has succeeded Dr. Nadell as medical superintendent of Lincoln Hospital.

Personal.—Dr. Paul F. Russell, representative in Italy of the Rockefeller Foundation, Division of Medicine and Public Health, was a guest lecturer during July at a World Health Organization Malaria Training Course in Lagos, Nigeria. Dr. Russell's address after Sept. 1 will be Rockefeller Foundation, 20 Rue de La Baume, Paris 8^{e}, France.——Dr. Frank R. Ferlaino, assistant clinical professor of industrial medicine, University Post-Graduate Medical School of the New York Bellevue Medical Center, has been appointed counselor of the New York Metropolitan District by the Industrial Medical Association.

PENNSYLVANIA

School Health Improvement.—A research program designed to improve the health of school children has been established at Pennsylvania State College with a grant of $35,000 from the Health Information Foundation, New York. According to Milton S. Eisenhower, LL.D., president of the college, and Adm. William H. P. Blandy, USN (Ret.), president of the foundation, methods will be sought of impressing parents with the importance of providing treatment for defects reported in school health examinations. In Pennsylvania, an examination is given each school child every other year, and notice of the observed defects is sent to the home with a request that parents take the child to a private doctor or dentist for treatment and report to the school when this is done. House-to-house studies in four communities revealed that, of 763 school children examined, 363 were found in need of treatment, but only 166 had received treatment. Various agencies of the state government as well as private organizations in the fields of health, welfare, and education will work with the research personnel. After information now available is assembled and the programs are observed, a symposium on the problem will be held.

Philadelphia

Gonorrhea Cases Increase.—Dr. James P. Dixon, health commissioner, reports a 13% increase in gonorrhea during the first half of the current year, a reversal of the downward trend for this disease begun in 1949. Gonorrhea topped the list of reportable diseases in Philadelphia during 1951, with 7,743 cases reported. Figures on the reporting of gonorrhea for continental United States show a decrease since Jan. 1. According to Dr. Dixon, special federal funds will be available within the next month to finance a project aimed at finding and treating Philadelphians infected with gonorrhea. One phase of the new project will be psychiatric examination of patients infected more than one time.

TEXAS

Another Twenty-Four Million Volt Betatron.—The M. D. Anderson Hospital for Cancer Research, Houston, will become the second hospital in the United States to have a 24 million volt betatron for treatment of patients and cancer research. The University of Illinois Research Hospital has a similar machine. Purchase of the machine for Anderson is made possible by grants totaling $121,000 from the U. S. Public Health Service's National Cancer Institute. Additional support for the second and third years of operation of the project has been committed depending on availability of public health funds at that time. Dr. Gilbert H. Fletcher, radiotherapist, and Peter Wooten, physicist, will be in charge of the research project involving the betatron.

UTAH

Annual State Medical Meeting.—The Utah State Medical Association will hold its annual meeting Sept. 4-6 in the Union Building of the University of Utah, Salt Lake City, under the presidency of Dr. Lewis W. Oaks of Provo. Out-of-state speakers will include:

Richard Ford, Boston, System and Technique for Investigation of Violent and Unexplained Death.
Charles P. Bailey, Philadelphia, Surgery of Mitral Valvular Disease.
Edgar M. Holmes, Boston, Diagnosis and Treatment of Facial Fractures.
Robert J. Bloor, Rochester, N. Y., X-Ray Diagnosis of Neoplasms of the Gastrointestinal Tract.
Walter G. Maddock, Chicago, Observations Leading to a Better Understanding of Gastrointestinal Distention.
William C. Keettel, Iowa City, Indication and Contraindication for Induction of Labor.
Roy H. Turner, New Orleans, 1. On Eating Fat. 2. Some Generalizations on Diagnosis of Liver Disease.

Luncheon meetings on Thursday and Friday will include round-table discussions. Movies will be shown at noon on Saturday and also at 12:45 p. m. Thursday and Friday. At 4 p. m. Friday there will be a meeting of the Utah chapter of the American Academy of General Practice, and at 7 p. m. the annual banquet will be held in the Hotel Utah.

Members of the medical corps of the U. S. Armed Forces in uniform are invited to attend the scientific sessions without registration fee. Other physicians, resident in Utah, who are not members of the Utah State Medical Association will be charged a registration fee equal to the current state dues.

WASHINGTON

State Medical Meeting in Seattle.—The Washington State Medical Association will hold its annual convention in Seattle at the Olympic Hotel, Sept. 13-17, under the presidency of Dr. Reuben A. Benson, Bremerton. The presidential address at 11:30 Tuesday will be followed by a talk by Dr. Ernest B. Howard, Chicago, Assistant Secretary of the American Medical Association. Guest speakers and their first presentations at the scientific session are as follows:

John H. Dingle, Cleveland, Viruses and Virus Diseases.
Dwight E. Harken, Boston, Responsibility of the Physician in Recognizing Surgical Mitral Heart Disease.
Edwin Jack Wylie, San Francisco, Surgical Revascularization in Arteriosclerotic Thrombosis.

The public relations luncheon at 12:30 Wednesday will be addressed by Dr. Lewis A. Alesen, Los Angeles, president-elect of the California Medical Association.

The section on ophthalmology and otolaryngology, which will meet on Wednesday, will have a symposium on "Retrolental Fibroplasia" at 9:40 a. m. and a panel discussion on "Lesions of the Mouth" at 11 o'clock.

The forum on fundamental problems Wednesday, 2:40-5 p. m., will include discussions on mitral commissurotomy, experimental pulmonary edema, candida (monilia) following treatment with antibiotics, fertility and sterility, mechanisms of muscle pain, psychophysiological aspects of colic, and diseases of the blood.

The nonscientific program includes a "no-host" family dinner on Sunday, during which the general practitioner's and past presidents' awards will be made; the salmon fishing derby, golf tournament, and sportsmen's banquet will take place on Monday. The banquet Tuesday will be followed by a dance. On Wednesday at 6:30 p. m. the president's reception will be held. The woman's auxiliary will hold its annual meeting at the Olympic Hotel Sept. 14-17.

WISCONSIN

Meeting of Ophthalmologists and Otolaryngologists.—The Central Wisconsin-Upper Michigan Society of Ophthalmology and Otolaryngology will meet at Maxwelton Braes, Bailey's Harbor, Door County, Sept. 6-7. On Saturday afternoon Dr. Noah D. Fabricant of Chicago will discuss "Current Trends in Medication of the Ear, Nose, and Throat," and Dr. William F. Hughes Jr., Chicago, will speak on "Repair of Lid Injuries, Including Entropion and Ectropion."

Cocktail hour at 6 p. m. precedes the dinner at which Dr. Fabricant will present "Amusing Quotations for Doctors and Patients." Sunday morning Dr. Hughes will read a paper on "Present Day Practice Regarding Orbital Implants After Enucleation" and present a movie on "The Technique of Implantation of Buried Polyvinyl Implants."

The meeting will conclude with luncheon at 12:30 p. m.

GENERAL

National Board Changes Address.—After Sept. 1 the National Board of Medical Examiners will be located at 133 S. 36th St., Philadelphia 4; telephone number, EVergreen 6-6704.

Malaria Reported.—Of 434 cases of malaria reported to the U. S. Public Health Service for the week ended Aug. 2, 62 occurred in civilians. Twenty of the 22 cases in California were reported in Veterans' Administration hospitals; in 12 of these the infection was of the tertian type, and in 9 the disease had been acquired in Korea. The patient in one civilian case was a Mexican national. Other states reporting five or more civilian cases of malaria were Oklahoma (11), Iowa (6), and Texas (5).

Death Certificates.—A booklet called "Medical Certification of Cause of Death," prepared by the World Health Organization, provides instructions for physicians on use of the medical certification of the cause of death. Since this country has adopted the international system of classification of causes of death, the booklet should prove useful in all vital statistics offices, in training programs in nosology, in assisting young physicians, and in programs of querying by mail.

Research in Arthritis.—The Arthritis and Rheumatism Foundation is offering research fellowships on both the predoctoral and postdoctoral levels in the basic sciences related to arthritis. Fellowships will run for one year with prospect of renewal. Predoctoral fellowships will range from $1,500 to $3,000 per annum, depending on the family responsibilities of the fellow, and postdoctoral fellowships from $3,000 to $6,000 on the same basis. Deadline for applications is Nov. 1, 1952. Applications will be reviewed and awards made by Feb. 15, 1953. For information address the Medical Director, The Arthritis and Rheumatism Foundation, 23 W. 45th St., New York 36.

Poliomyelitis in Other Countries.—The U. S. Public Health Service reports that the incidence of poliomyelitis has increased in Canada, principally in the four western provinces. Up to Aug. 4, 104 cases with five deaths were reported in the province of Alberta, and in the city of Calgary, 30 cases had occurred. The largest previous total (20) was reported in 1930. For the two weeks ended July 26, 26 cases were reported in Cuba, 21 of which were in the Province of Habana. About 80% of the cases reported are of the paralytic type. In the German Federal Republic, 492 cases were reported for the week ended July 26, as compared with 326 for the previous week. Switzerland reported more cases during the four weeks ended July 26 than during similar periods of 1950 and 1951.

Life Insurance Medical Research Fellowships.—Applications for awards available July 1, 1953, will be received by the Life Insurance Medical Research Fund as follows: 1. Applications for postdoctoral research fellowships will be accepted until Oct. 31, 1952. Preference is given to those who wish to work on cardiovascular function and disease or related fundamental problems. Stipends vary from $3,300 to $4,500. 2. Applications for grants to institutions in aid of research on cardiovascular problems will be accepted until Nov. 15, 1952. Support is available for physiological, biochemical, and other basic research on cardiovascular problems as well as for clinical investigation in this field. Information may be obtained from the Scientific Director, Life Insurance Medical Research Fund, 2 E. 103rd St., New York 29.

Anesthetists Meet in Virginia.—The Congress of the International Anesthesia Research Society will be held at Virginia Beach, Sept. 22-25, under the presidency of Dr. George J. Thomas, Pittsburgh. The following presentations will be accompanied by lantern slide demonstrations:

Robert M. Smith, Boston, Prevention of Tracheitis Following Endotracheal Intubation on Pediatric Anesthesia.
Francis F. Foldes, Pittsburgh, Role of Cholinesterase in Anesthesiology.
David M. Little Jr. and L. Jennings Hampton, New Haven, Conn., Diacetylcholine (Succinylcholine): A Controllable Muscle Relaxant.
Clarence L. Hebert, Staten Island, N. Y., Role of Anesthesiologist in Patient Care During the Immediate Postanesthesia-Postoperative Period.
Joseph Owens Hayes, Buffalo, Translaryngeal Anesthesia.
D. Dwight Grove and George E. Covintree, Philadelphia, Precautions with Gases Used by the Anesthesiologist Exclusive of Fire and Explosion Hazards.
Dexter R. Branch, Cambridge, Mass., Ether Anesthesia—A True Gas Vaporization Technique.

The sessions will close with a panel discussion on "Controlled Hypotension as an Adjunct to Surgery."

American Roentgen Ray Society.—The annual meeting of the American Roentgen Ray Society will be held at the Shamrock Hotel, Houston, Texas, Sept. 23-26, under the presidency of Dr. Charles L. Martin, Dallas, Texas, who will be installed at the meeting Tuesday morning. During the sessions 34 papers will be presented. Tuesday evening Dr. Edward B. D. Neuhauser of Boston will give the Caldwell Lecture on "Growth Differentiation and Disease." On Wednesday evening Dr. Clyde A. Stevenson of Temple, Texas, will serve as moderator for a film reading session. Collaborators will be Drs. Paul C. Hodges, Chicago, Fred J. Hodges, Ann Arbor, Mich., Merrill C. Sosman, Boston, and C. Allen Good, Rochester, Minn.

A golf tournament will be held Monday at the River Oaks Country Club. Arrangements for deep sea and other fishing trips may be made through a committee headed by Dr. Palmer Wigby, 506 Hermann Professional Bldg., Houston. Instruction courses will meet at 8:30 a. m. each day during the convention.

Prevalence of Poliomyelitis.—According to the National Office of Vital Statistics, the following number of reported cases of poliomyelitis occurred in the United States, its territories, and possessions in the weeks ended as indicated:

Area	Aug. 9, 1952	Aug. 2, 1952	Aug. 11, 1951
New England States			
Maine	5	2	5
New Hampshire	8	4	5
[illegible]	1	..	1
[illegible]	45	19	26
[illegible]	2	3	..
[illegible]	19	16	25
Middle Atlantic States			
New York	129	89	105
New Jersey	38	38	10
Pennsylvania	75	47	25
East North Central States			
Ohio	180	128	73
Indiana	40	41	16
Illinois	212	140	129
Michigan	225	159	110
Wisconsin	98	67	57
West North Central States			
Minnesota	162	113	19
Iowa	192	244	33
Missouri	37	14	26
North Dakota	7	7	2
South Dakota	13	8	3
Nebraska	126	96	25
Kansas	71	83	30
South Atlantic States			
Delaware	9	4	..
Maryland	5	2	7
District of Columbia	11	8	3
Virginia	67	46	18
West Virginia	57	58	12
North Carolina	21	15	16
South Carolina	2	6	7
Georgia	10	16	38
Florida	23	20	20
East South Central States			
[illegible]	94	94	17
[illegible]	37	27	51
[illegible]	14	18	71
[illegible]	37	24	28
West South Central States			
Arkansas	18	13	17
Louisiana	17	51	36
Oklahoma	53	59	49
Texas	200	276	97
Mountain States			
Montana	11	10	1
Idaho	20	11	5
Wyoming	5	9	3
Colorado	27	26	112
New Mexico	36	30	3
Arizona	22	13	20
Utah	4	..	34
Nevada	4	6	2
Pacific States			
Washington	53	50	19
Oregon	12	9	16
California	95	86	89
Territories and Possessions			
Alaska	1	1	1
Hawaii	6	5	3
Puerto Rico	1	..	..
Total	2,657	2,311	1,520

FOREIGN

International Congress on Hearing Technique.—The first International Meeting on Hearing Technique will be held at Teatro dell'Opera, Casino Municipale in Sanremo, Italy, Sept. 21-28. The subjects to be discussed include street and factory noises, acoustic insulation in buildings, apparatus for testing simulation of deafness, artificial hearing apparatus, apparatus for teaching deaf people and backward children, and institutions to combat noises and deafness. Every participant will be enabled to hear all lectures and communications in his own language by means of headphones of the simultaneous translation apparatus. For information, contact Centro Audiotecnico Internazionale, C. P. 207, Sanremo, Italy.

All-India Obstetric and Gynaecological Congress.—The Seventh All-India Obstetric and Gynaecological Congress will be held Dec. 15-17 in Calcutta. The chief subjects for discussion will be "Cephalopelvic Disproportion" and "Nonmalignant Lesions of the Cervix." Obstetric and gynecologic societies of India and abroad, as well as teaching institutions and practitioners in the profession, are invited to take part in the conference. Those who desire to present papers on either of the main subjects are requested to communicate the title to the secretary's office of the Bengal Obstetric and Gynaecological Society, 91B, Chittaranjan Ave., C. M. C. House, Calcutta 12, by Oct. 15 and to submit a typed copy of the paper with abstracts by the end of October.

EXAMINATIONS AND LICENSURE

BOARDS OF MEDICAL EXAMINERS

Alabama: *Examination.* Montgomery, June 23-25. Sec., Dr. D. G. Gill, 537 Dexter Ave., Montgomery.

Alaska:* *Examination.* Juneau. On application in other towns where there are board members. *Reciprocity.* On application. Sec., Dr. W. M. Whitehead, Box 140, Juneau.

Arkansas:* *Examination.* Little Rock, Nov. 6-7. Sec., Dr. Joe Verser, Harrisburg.

California: *Written.* Sacramento, Oct. 20-23. Application must be received at least two weeks prior to date of examination. *Oral for Reciprocity Applicants.* San Francisco, Nov. 8. Applicants are requested not to arrange to come to an oral examination until they receive notice from the Credentials Committee. *Oral and Clinical for Foreign Medical School Graduates.* San Francisco, Nov. 9. Sec., Dr. Frederick N. Scatena, 1020 N St., Sacramento.

Colorado:* *Reciprocity.* Denver, Oct. 14. Exec. Sec., Mrs. B. H. Hudgens, 831 Republic Bldg., Denver.

District of Columbia:* *Reciprocity.* Washington, Sept. 8. Sec., Dr. Daniel L. Seckinger, 4130 E. Municipal Bldg., Washington.

Florida:* *Examination.* Jacksonville, Nov. 23-25. Sec., Dr. Homer L. Pearson, 701 Dupont Bldg., Miami 32.

Georgia: *Reciprocity.* Atlanta, Oct. 14-15. Sec., Mr. R. C. Coleman, 111 State Capitol, Atlanta 3.

Guam: The Commission on Licensure will meet whenever a candidate appears or submits his credentials. Ex. Sec., Dr. Austin W. Matthis, Agana.

Illinois: *Examination.* Chicago, Sept. 23-25. Supt. of Regis., Mr. Charles F. Kervin, Capitol Bldg., Springfield.

Kansas: Topeka, Dec. 10-11. Sec., Dr. O. W. Davidson, 864 New Brotherhood Bldg., Kansas City.

Maryland: *Examination.* Baltimore, Dec. 9-12. Sec., Dr. Lewis P. Gundry, 1215 Cathedral St., Baltimore 1.

Michigan:* *Examination.* Lansing, Oct. 8-10. Sec., Dr. J. Earl McIntyre, 202-4 Hollister Bldg., Lansing.

Minnesota:* Minneapolis, Oct. 21-23. Sec., Dr. J. F. Du Bois, 230 Lowry Medical Arts Bldg., St. Paul 2.

Mississippi: *Reciprocity.* Jackson, December. Asst. Sec., Dr. R. N. Whitfield, State Board of Health, Jackson 113.

Montana: Helena, Oct. 6-8. Sec., Dr. S. A. Cooney, 214 Power Block, Helena.

Nebraska:* *Examination.* Omaha, June 1953. Director, Mr. Husted K. Watson, Room 1009 State Capitol Bldg., Lincoln 9.

New Hampshire: Concord, Sept. 10. Sec., Dr. John S. Wheeler, 107 State House, Concord.

New Jersey: *Examination.* Trenton, Oct. 21-24. Sec., Dr. E. S. Hallinger, 28 W. State St., Trenton.

North Carolina: *Reciprocity.* Durham, Oct. 13. Sec., Dr. Joseph J. Combs, 419 Professional Bldg., Raleigh.

Ohio: *Reciprocity.* Columbus, Oct. 7. Sec., Dr. H. M. Platter, 21 W. Broad St., Columbus.

South Carolina: *Reciprocity.* Columbia, Sept. 3. Sec., Mr. N. B. Heyward, 1329 Blanding St., Columbia.

Tennessee:* *Examination.* Memphis, Oct. 1-2. Sec., Dr. H. W. Qualls, 1635 Exchange Bldg., Memphis.

Virginia: *Examination.* Richmond, Dec. 11-13. *Reciprocity.* Richmond, Dec. 10. Sec., Dr. K. D. Graves, 631 First St., S.W., Roanoke, Va.

West Virginia: Charleston, Oct. 13-14. Sec., Dr. N. H. Dyer, State Office Bldg #3, Charleston 5.

Wyoming: Cheyenne, Oct. 6. Sec., Dr. Franklin D. Yoder, State Office Bldg., Cheyenne.

Virgin Islands: *Examination.* St. Thomas, Nov. 12-13. Sec., Dr. Earle M. Rice, St. Thomas.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

Alaska: *Examination.* On application. Juneau or other towns in Territory as decided by Board. *Reciprocity.* On application. Sec., Dr. C. Earl Albrecht, Box 1931, Juneau.

Arizona: *Examination.* Tucson, Sept. 16. Sec., Mr. Herbert D. Rhodes, Room 423, Liberal Arts Bldg., University of Arizona, Tucson.

Arkansas: *Examination.* Little Rock, Oct. 1-2. Sec., Mr. Louis E. Gebauer, 1002 Donaghey Bldg., Little Rock.

Colorado: *Examination.* Denver, Sept. 10-11. Sec., Dr. Esther B. Starks, 1459 Ogden St., Denver 18.

Connecticut: New Haven, Oct. 11. Address: State Board of Healing Arts, 110 Whitney Ave., New Haven 10.

District of Columbia: *Examination.* Washington, Oct. 20-21. Sec., Dr. Daniel L. Seckinger, 4130 E. Municipal Bldg., Washington.

Florida: *Examination.* Gainesville, Oct. 25. Sec., Mr. M. W. Emmel, Box 340, University of Florida, Gainesville.

Michigan: *Examination.* Detroit and Ann Arbor, Oct. 10-11. Sec., Mrs. Anne Baker, 423 W. Michigan Ave., Lansing.

Minnesota: *Examination.* Minneapolis, Oct. 7-8. Sec., Mr. Raymond N Bieter, 105 Millard Hall, University of Minnesota, Minneapolis.

Nebraska: *Examination.* Omaha, Oct. 7-8. Director, Bureau of Examining Boards, Mr. Husted K. Watson, 1009 State Capitol Bldg., Lincoln.

New Mexico: *Examination.* Santa Fe, Sept. 21. Sec., Mrs. Marguerite Cantrell, Box 1592, Santa Fe.

Oklahoma: *Examination.* Oklahoma City, Sept. 13. Sec., Dr. Clinton Gallaher, 813 Braniff Bldg., Oklahoma City.

Oregon: *Examination.* Portland, Sept. 6 and Dec. 6. Sec., Dr. Charles D. Byrne, State Board of Higher Education, University of Oregon, Eugene.

South Dakota: *Examination.* Vermillion, Dec. 5-6. Sec., Dr. Gregg M. Evans, 310 E. 15th St., Yankton.

Tennessee: *Examination.* Memphis, Sept. 24-25. Sec., Dr. O. W. Hyman, 874 Union Ave., Memphis 3.

Texas: *Examination.* Austin, October. Sec., Brother Raphael Wilson, 407 Perry-Brooks Bldg., Austin.

Wisconsin: *Examination.* Madison, Sept. 13, Milwaukee, Dec. 6. Sec., Mr. W. H. Barber, 621 Ransom St., Ripon.

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* Basic Science Certificate required.

MEETINGS

American Medical Association Public Relations Institute, Edgewater Beach Hotel, Chicago, Sept. 4-5. Mr. Leo E. Brown, 535 N. Dearborn St., Chicago 10, Director.

American Academy for Cerebral Palsy, Durham, N. C., Oct. 2-4. Dr. Meyer A. Perlstein, 4743 North Drake Ave., Chicago 25, Secretary.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 12-17. Dr. W. L. Benedict, 100 First Avenue Bldg., Rochester, Minn., Executive Secretary.

American Academy of Pediatrics, Palmer House, Chicago, Oct. 20-23. Dr. E. H. Christopherson, 610 Church St., Evanston, Ill., Secretary.

American Association of Blood Banks, Hotel Schroeder, Milwaukee, Oct. 9-11. Miss Marjorie Saunders, 3500 Gaston Avenue, Dallas 1, Texas, Secretary.

American Association of Medical Record Librarians, Shoreham Hotel, Washington, D. C., Oct. 13-17. Miss Doris Gleason, 510 N. Dearborn St., Chicago 10, Executive Secretary.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, The Homestead, Hot Springs, Va., Sept. 4-6. Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Texas, Secretary.

American Clinical and Climatological Association, Elmira, N. Y., Oct. 16-18. Dr. Marshall N. Fulton, 124 Waterman St., Providence 6, R. I., Secretary.

American College of Surgeons, Waldorf-Astoria, New York, Sept. 22-26. Dr. Michael L. Mason, 40 East Erie St., Chicago 11, Secretary.

American Fracture Association, Hotel Sherman, Chicago, Oct. 6-9. Dr. H. W. Wellmerling, 219 N. Main St., Bloomington, Ill., Secretary.

American Hospital Association, Philadelphia, Sept. 15-18. Mr. George Bugbee, 18 East Division St., Chicago 10, Executive Director.

American Medical Writers' Association, Jefferson Hotel, St. Louis, Oct. 1. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

American Physiological Society, New Orleans, Sept. 4-6. Dr. E. F. Adolph, University of Rochester School of Medicine and Dentistry, Rochester, N. Y., Secretary.

American Public Health Association, Public Auditorium, Cleveland, Oct. 20-24. Dr. Reginald M. Atwater, 1790 Broadway, New York 19, Executive Secretary.

American Roentgen Ray Society, Shamrock Hotel, Houston, Texas, Sept. 23-26. Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Secretary.

American Society of Clinical Pathologists, Chicago, Oct. 13-17. Dr. Clyde G. Culbertson, 1040 W. Michigan St., Indianapolis, Secretary.

American Society of Maxillofacial Surgeons, Hotel del Coronado, San Diego, Calif., Sept. 10-13. Dr. Casper M. Epsteen, 25 East Washington St., Chicago 2, Secretary.

Annual Gulf Coast Regional Conference on Industrial Health, Rice Hotel, Houston, Tex., Sept. 24-27. Dr. W. H. Hamrick, P. O. Box 2371, Houston, Tex., Chairman.

Association of Life Insurance Medical Directors of America, Los Angeles, Oct. 21-23. Dr. Henry B. Kirkland, P. O. Box 594, Newark 1, N. J., Secretary.

Biological Photographic Association, Hotel New Yorker, New York, Sept. 10-12. Mr. Lloyd E. Varden, Pavelle Color, Inc., 533 W. 57th St., New York 19, Secretary.

College of American Pathologists, Drake Hotel, Chicago, Oct. 13-17. Dr. M. G. Westmoreland, 203 N. Wabash Ave., Chicago 1, Executive Secretary.

Colorado State Medical Society, Stanley Hotel, Estes Park, Sept. 9-12. Mr. Harvey T. Sethman, 835 Republic Bldg., Denver 2, Executive Secretary.

Constantinian Society, Lake Placid Club, Lake Placid, N. Y., Oct. 1-4. Dr. C. F. Shook, P. O. Box 1035-36, Toledo 1, Ohio, Secretary.

Delaware, Medical Society of, Rehoboth, Sept. 8-10. Dr. Andrew M. Gehret, 822 North American Bldg., Wilmington, Secretary.

District of Columbia, Medical Society of the, Hotel Statler, Washington, Sept. 29-Oct. 1. Mr. Theodore Wiprud, 1718 M St. N.W., Washington, Secretary.

Great Falls Medical-Surgical Conference, Great Falls, Mont., Oct. 24-25. Dr. William E. Sullens, Box 1066, Great Falls, Mont., Chairman.

Gulf Coast Clinical Society, San Carlos Hotel, Pensacola, Fla., Oct. 16-17. Dr. Dale E. York, 21½ E. Wright St., Pensacola, Fla., Secretary.

Kansas City Southwest Clinical Society, Kansas City, Mo., Oct. 6-9. Dr. Galen M. Tice, 630 Shukert Bldg., Kansas City, Kansas, Secretary.

Kentucky State Medical Association, Brown Hotel, Louisville, Oct. 7-9. Dr. Bruce Underwood, 620 S. Third St., Louisville 2, Secretary.

Michigan State Medical Society, Book-Cadillac Hotel, Detroit, Sept. 24-26. Dr. L. Fernald Foster, 606 Townsend St., Lansing 15, Secretary.

Mississippi Valley Medical Society, Jefferson Hotel, St. Louis, Oct. 1-3. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

Montana Medical Association, Florence Hotel, Missoula, Sept. 18-21. Mr. L. R. Hegland, 104 North Broadway, Billings, Executive Secretary.

National Gastroenterological Association, Hotel Statler, New York, Oct. 20-22. Dr. Sigurd W. Johnsen, 1819 Broadway, New York 23, Secretary.

Neurosurgical Society of America, The Cloister, Sea Island, Ga., Sept. 18-20. Dr. C. D. Hawkes, 22 North Manassas St., Memphis 5, Tenn., Secretary.

New Hampshire Medical Society, Mt. Washington Hotel, Bretton Woods, Sept. 7-9. Dr. Deering G. Smith, 44 Chester St., Nashua, Secretary.

North Pacific Pediatric Society, Challenger Inn, Sun Valley, Idaho, Sept. 19-20. Dr. S. Gorham Babson, 1107 S.W. 13th Ave., Portland 5, Ore., Secretary.

North Pacific Society of Internal Medicine, Sun Valley, Idaho, Sept. 19-20. Dr. Robert L. King, 1115 Terry Ave., Seattle 1, Wash., Secretary.

North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, Texas, Sept. 17. Dr. E. Aubrey Cox, Hamilton Bldg., Wichita Falls, Texas, Chairman.

Oregon State Medical Society, Portland, Oct. 8-11. Dr. Robert F. Miller, 833 S.W. 11th Ave., Portland 5, Secretary.

Pennsylvania, Medical Society of the State of, Bellevue-Stratford Hotel, Philadelphia, Sept. 28-Oct. 2. Dr. Walter F. Donaldson, 500 Penn Ave., Pittsburgh 22, Secretary.

Southwestern Surgical Congress, Baker Hotel, Dallas, Texas, Oct. 20-22. Miss June Marchant, 1227 Classen St., Oklahoma City, Executive Secretary.

Tri-State Medical Society of Texas, Louisiana and Arkansas, Texarkana, Texas, Oct. 2-3. Dr. Albert M. Hand, 619 Main St., Texarkana, Texas, Secretary.

U. S. Chapter, International College of Surgeons, Conrad Hilton Hotel, Chicago, Sept. 2-5. Dr. Arnold S. Jackson, 1516 Lake Shore Drive, Chicago, Executive Secretary.

Utah State Medical Association, Salt Lake City, Sept. 4-6. Dr. T. C. Weggeland, 42 S. Fifth East St., Salt Lake City, Secretary.

Vermont State Medical Society, Mt. Washington Hotel, Bretton Woods, N. H., Sept. 7-9. Dr. James P. Hammond, 128 Merchants Row, Rutland, Secretary.

Virginia, Medical Society of, Jefferson Hotel, Richmond, Sept. 28-Oct. 1. Mr. Robert L. Howard, 1105 West Franklin St., Richmond, Executive Secretary.

Washington State Medical Association, Olympic Hotel, Seattle, Sept. 13-17. Dr. Bruce Zimmerman, 338 White-Henry-Stuart Bldg., Seattle, Secretary.

Wisconsin, State Medical Society of, Hotel Schroeder, Milwaukee, Oct. 6-8. Mr. Charles H. Crownhart, 704 E. Gorham St., Madison, Secretary.

INTERNATIONAL

Aero Medical Association Interim Meeting, Paris, France, Sept. 26-28. Dr. Armand Robert, 2 Rue Marbeuf, Paris 8e, France, Secretary.

American Congress of Industrial Medicine, Rio de Janeiro, Brazil, Sept. 20-28. Prof. Dr. Jose Pedro Reggi, Calle Arenales No. 981 Buenos Aires, Argentina, Director General.

Congress of International Anesthesia Research Society, Virginia Beach, Virginia, U. S. A., Sept. 22-25.

European Congress of Cardiology. University of London, Bloomsbury, W.C.1, England, Sept. 10-12. Dr. K. Shirley Smith, 35 Wimpole St., London, W.1, England, Secretary.

European Society of Cardiovascular Surgery, Strasbourg, France, Oct. 3-4. Sir James Learmonth, Department of Surgery, University New Buildings, Edinburgh 8, Scotland.

Interamerican Cardiological Congress, Buenos Aires, Argentina, Aug. 31-Sept. 7. Dr. Blas Moia, Larrea 1132, Buenos Aires, Argentina, Secretary-General.

Inter-American Congress of Public Health, Havana, Cuba. Sept. 26-Oct. 1. Pan-American Sanitary Bureau, 1501 New Hampshire Ave. N.W., Washington 6, D. C.

Inter-American Congress of Radiology, Mexico City, Mexico, Nov. 2-8. Dr. Guido Torres Martinez, Marsella No. 11, Mexico, D.F., Secretary-General.

International Congress of Anthropology and Ethnology, Vienna, Austria, Sept. 1-8. For information write: Secretaries, Royal Anthropological Institute, 52 Upper Bedford Place, London, W.C.1, England.

International Congress of Haematology, Mar del Plata, Argentina, Sept. 21-26. Dr. Carlos Reussi, Anchorena 1710, Buenos Aires, Argentina, Secretary.

International Congress of the History of Medicine, Nice-Cannes, France, and Monaco, Sept. 8-14. Dr. F. A. Sondervorst, 124 Avenue des Allies, Louvain, Belgium, Secretary-General.

International Congress on Hydatid Disease, Santiago, Chile, Nov. 21-24. Organizing Committee, P. O. Box 9183, Santiago, Chile.

International Congress of Internal Medicine, Friends House, London, N.W.1, Eng. Sept. 15-18. Sir Harold Boldero, 12 Pall Mall East, London, S.W.1, England, Secretary.

International Congress for Medical Librarians, London, England, July 20-25, 1953. Mr. W. R. LeFanu, % London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C.1, England, Chairman.

International Congress of Medical Press, Venice, Italy, Oct. 4-5. Dr. Bruno de Biasio, S. Marco 4646, Venice, Italy, Secretary.

International Congress on Medical Records, London, England, Sept. 7-12. Miss Gwen Perkins, 120 C Street S.E., Washington, D. C., Chairman.

International Congress of Military Medicine and Pharmacy, Montevideo, Uruguay, March 1-7, 1953. Dirreccion General del Servico de Sanitad Militar, 8 de Octubre y Mariano Moreno, Montevideo, Uruguay, Secretariat of the Congress.

International Congress on Neuropathology, Rome, Italy, Sept. 8-13. Dr. Armando Ferraro, 722 W. 168th St., New York, N. Y., U. S. A., Secretary General.

International Congress of Otorhinolaryngology, Amsterdam, Netherlands, June 8-15, 1953. Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam, Netherlands, Secretary.

International Congress of Radiology, Copenhagen, Denmark, July 19-25, 1953. Professor Flemming Norgaard, 10 Oster Voldgade, Copenhagen K., Denmark, Secretary General.

International Congress of Therapeutics, Madrid, Spain, Oct. 16-18. Professor Armijo, Instituto de Farmacología, Cuidad Universitaria, Madrid, Spain, Secretary.

International Congress of Urology, The Waldorf-Astoria, New York, N. Y., U. S. A., Sept. 15-18. Dr. John A. Taylor, 2 East 54th St., New York, N. Y., U. S. A., Secretary-General.

International Hospital Congress, London, England, May 25-30, 1953. Capt. J. E. Stone, 10 Old Jewry, London, EC2, England, Hon. Secretary.

International Study Conference on Child Welfare, Bombay, India, Dec. 5-12. For information write: All India Save the Children Committee, 5 Carmichael Road, Bombay, India.

Medical Women's International Association, Vichy, France, Sept. 12-16. Dr. Margaret M. Wurts, 27 East Wellesley Road, Montclair, N. J., U. S. A., National Secretary.

Neuroradiological Symposium, Stockholm, Sweden, Sept. 17-20. Docent Ake Lindbom, Symposium Neuroradiologicum, Serafimerlasarettet, Stockholm K., Sweden, Secretary.

Pan American Congress of the Medical Press, Buenos Aires, Argentine, July 12-16, 1953. Secretaria del Congress, 763 Uriburu, Buenos Aires, Argentine.

World Congress of Medicine, Montecatini, Italy, Oct. 16-18. Secretariat: Corso Trieste, 65, Rome, Italy.

World Medical Association, Athens, Greece, Oct. 12-16. Dr. Louis H. Bauer, 2 East 103d St., New York 29, N. Y., Secretary General.

DEATHS

Sutton, Richard Lightburn ⊕ McAllen, Texas; professor emeritus of dermatology at the University of Kansas School of Medicine in Kansas City, Kan.; lieutenant (jg) U. S. Navy, retired; born in Rockport, Mo., July 9, 1878; University Medical College of Kansas City, Mo., 1901; George Washington University School of Medicine, Washington, D. C., 1904; veteran of the Spanish-American War; entered the U. S. Navy Feb. 17, 1903, and was retired June 30, 1905 for incapacity resulting from an incident of the Service; at one time practiced in Kansas City, Mo., where he was affiliated with the Kansas City General, Bell Memorial, and St. Luke's hospitals; Associate Fellow and in 1913-1914 Chairman of the Section on Dermatology of the American Medical Association; past president of the Jackson County (Mo.) Medical Society; member of the American Dermatological Association, Missouri State Medical Association, and French Geographic Society; life fellow of the Royal Geographical Society of Great Britain; fellow of the Royal Society of Edinburgh; corresponding member of the Swedish Dermatological Society; specialist certified by the American Board of Dermatology and Syphilology; trustee of the Kansas City Museum and member of the board of directors of the Salvation Army in Kansas City, Mo.; special representative with the department of natural history, University of Missouri, African Expedition in 1923-1924 and expeditions for the same department to Indo-China and India, 1925-1926; was head of an African-Asiatic expedition in 1929-1930, Arctic expedition, north of Spitsbergen, as special representative, department of natural history, University of Kansas in 1932; conducted several expeditions to New Zealand and Australia, investigating habits of swordfish, and to Peru in 1941; in 1939 was with East Arctic Patrol, Northwest Territories, to Ellesmere Island, Somerset Island, and Baffinland; received the degree of LL.D. from the University of Missouri in 1922 and the honorary degree of Sc.D. from Washburn College, Topeka, in 1925; author of "African Holiday" 1924, "Tiger Trails in Southern Asia" 1926, "The Long Trek, Around the World With Camera and Rifle" 1930, "Arctic Safari: With Camera and Rifle in the Land of the Midnight Sun" 1932; co-author with Dr. Richard L. Sutton Jr., "Diseases of the Skin" (10th edition) 1939, "An Introduction to Dermatology" (4th edition) 1941, "Synopsis of Diseases of the Skin" 1942, and "Handbook of Diseases of the Skin" 1949, part two, 1950; author of "Silver Kings of Aransas Pass and Other Stories" 1937, section on "The Mycoses in Tice's System of Medicine" 1919, rewritten with Dr. R. L. Sutton Jr., in 1940, "Collected Verse" 1946, and also numerous scientific articles in American and foreign journals; died in Baylor University Hospital, Dallas, May 18, aged 73.

Slaughter, Donald Horace ⊕ Vermillion, S. D.; born in Shelby, Iowa, July 13, 1905; State University of Iowa College of Medicine, Iowa City, 1929; since 1946 dean and professor of pharmacology at University of South Dakota School of Medical Sciences; acting dean, Southwestern Medical College of the Southwestern Medical Foundation, Dallas, Texas, 1943-1944, when he became dean of students, professor of pharmacology, and chairman of the department of pharmacology and physiology, serving until 1946; professor of pharmacology and physiology and chairman of the department at the University of Vermont College of Medicine in Burlington, 1942-1943; formerly associate professor of pharmacology at Baylor University College of Medicine, Dallas, Texas; instructor in pharmacology at his alma mater from 1929 to 1931 and associate from 1932 to 1937; in 1942 received the Certificate of Merit from the Scientific Exhibit of the American Medical Association for his original investigation on a quantitative pain threshold machine; fellow of the American Association for the Advancement of Science; member of the American Society for Pharmacology and Experimental Therapeutics, Society of Experimental Medicine and Biology, serving as secretary of its Southwest Section, American Association of University Professors, New York Academy of Science, American Chemical Society, Omega Beta Pi, Alpha Kappa Kappa, Alpha Omega Alpha, and Sigma Xi; died June 6, aged 46.

Eaton, H. Douglas ⊕ Los Angeles; born in New York March 14, 1886; Columbia University College of Physicians and Surgeons, New York, 1911; formerly associate clinical professor of medicine (neurology), University of Southern California School of Medicine; specialist certified by the American Board of Psychiatry and Neurology; member of the Central Neuropsychiatric Association; examiner for the commission of psychiatry of Los Angeles County; a consultant for the Los Angeles police pension board, and the Pacific Electric Railway; consulting psychiatrist to Occidental College; during World War II served as consulting psychiatrist to the Selective Service Board, and held a commission in the U. S. Public Health Service; member of the staff of the Good Samaritan Hospital, and one of the founder members of the Good Hope Clinic; for many years an active member of the staff of the Children's Hospital, having been elected chief of the neurological service at the time of its inception; died May 24, aged 66, of coronary occlusion.

Hawkins, Arthur Hanson ⊕ Cumberland, Md.; born in La Plata, Md., Dec. 27, 1868; College of Physicians and Surgeons, Baltimore, 1895; an Associate Fellow of the American Medical Association; past president of the Allegany-Garrett Counties Medical Society and the Medical and Chirurgical Faculty of Maryland; fellow of the American College of Surgeons; member of the Southern Medical Association; during World Wars I and II served as chairman and surgeon of the Selective Service Medical Advisory Board; consulting surgeon, Miners Hospital, Frostburg, and Hazel McGilvery Hospital in Meyersdale, Pa.; affiliated with Memorial Hospital, where he was chairman of the Army examining board, and where he died June 9, aged 83, of hypertension, cardiovascular renal disease, and uremia.

Miller, Richard Henry ⊕ Boston; born in 1884; Harvard Medical School, Boston, 1910; member of the House of Delegates of the American Medical Association from 1935 to 1938 and from 1941 to 1944; at one time on the faculty of his alma mater; member of the founders group of the American Board of Surgery; member of the American Surgical Association and the New England Surgical Society; fellow of the American College of Surgeons; served during World War I; consulting surgeon, Massachusetts General and Boston Lying-In hospitals in Boston, Henry Heywood Memorial Hospital in Gardner, Leominster Hospital in Leominster, Mass., and Memorial Hospital in Pawtucket, R. I.; died June 3, aged 67.

Heyward, Nathan Barnwell ⊕ Columbia, S. C.; born in Columbia, S. C., Dec. 27, 1886; Columbia University College of Physicians and Surgeons, New York, 1911; examiner in bacteriology and pathology, state board of medical examiners, of which he was a member for many years and in recent years secretary; formerly secretary of the South Carolina Medical Association; past president of the Columbia Medical Society; an associate of the American College of Physicians; member of the consulting staff, Providence Hospital; member of the visiting staff, Columbia Hospital and South Carolina Baptist Hospital, where he died June 3, aged 65, of coronary thrombosis.

Heim, Hugh Wilson ⊕ Nanticoke, Pa.; born in Schuylkill Haven, Pa., Aug. 20, 1911; Jefferson Medical College of Philadelphia, 1935; specialist certified by the American Board of Surgery; fellow of the American College of Surgeons; served during World War II; consulting surgeon, Good Samaritan Hospital, Pottsville, Retreat State Hospital in Retreat, and Wyoming Valley Homeopathic Hospital in Wilkes-Barre; surgeon, Nesbitt Memorial Hospital in Kingston, and Wilkes-Barre General Hospital in Wilkes-Barre; surgeon in chief and chief of staff, Nanticoke State Hospital; died July 1, aged 41, of essential hypertension.

⊕ Indicates Member of the American Medical Association.

Englehart, Charles Clayton ⊕ Houghton, Mich.; Temple Univerversity School of Medicine, Philadelphia, 1931; affiliated with St. Joseph's Hospital in Hancock; died recently, aged 52, of coronary thrombosis.

Handmacher, Benjamin Berolf, Ambridge, Pa.; University of Wooster Medical Department, Cleveland, 1906; died in Sewickley recently, aged 69, of carcinoma of the stomach.

Hassett, Myron Frederick ⊕ St. Paul; University of Minnesota Medical School, Minneapolis, 1932; died in Riverview Memorial Hospital April 23, aged 46, of dissecting aneurysm and hypertension.

Heberer, Joseph M. ⊕ Evansville, Ind.; Louisville (Ky.) Medical College, 1900; died April 2, aged 73, of cerebral hemorrhage and hypertensive cardiovascular disease.

Schafer, Robert ⊕ Chicago; University of Illinois College of Medicine, Chicago, 1925; served in the European theatre during World War II; on the staff of South Chicago Community Hospital, where he died May 1, aged 53, of acute glomerulonephritis.

Schanze, Raymond Andrew, Homerville, Ga.; University of Louisville (Ky.) School of Medicine, 1933; died in University Hospital, Augusta, May 1, aged 43, of heart disease.

Shailer, Sumner, Clearwater, Fla.; University of Pennsylvania Department of Medicine, Philadelphia, 1899; died recently, aged 77, of arteriosclerotic heart disease.

Sheldon, Benjamin Burt, New York; New York Homeopathic Medical College and Hospital, New York, 1900; died in Apopka, Fla., recently, aged 84, of bronchopneumonia and arteriosclerosis.

Simon, John Sr. ⊕ Englewood, Colo.; Denver and Gross College of Medicine, 1907; served as mayor; affiliated with Mercy Hospital and Porter Sanitarium and Hospital in Denver; died May 2, aged 76, of acute coronary thrombosis.

Skrabucha, William Albert ⊕ Lowell, Mass.; Middlesex University School of Medicine, Waltham, 1935; affiliated with St. Joseph's Hospital; accidentally drowned in Lakewood April 30, aged 41.

Sloan, Ambrose Victor ⊕ Wilkes-Barre, Pa.; McGill University Faculty of Medicine, Montreal, Canada, 1924; served with the Canadian Army in France and Belgium during World War I; on the staff of the Mercy Hospital; died May 7, aged 56, of coronary occlusion.

Smith, Racy Hawkins, Lincolnton, Ga.; University of Georgia Medical Department, Augusta, 1925; affiliated with Washington (Ga.) General Hospital; died May 4, aged 53, in an automobile accident.

Snelson, Andrew J., Checotah, Okla. (licensed in Oklahoma by years of practice); affiliated with Oklahoma Baptist Hospital in Muskogee; died May 4, aged 90, of myocardial degeneration.

Somers, Pearl Ellsworth ⊕ Tullahoma, Tenn.; Rush Medical College, Chicago, 1897; member of the Iowa State Medical Society; past president of the Poweshiek County (Ia.) Medical Society; died April 4, aged 81, of Parkinson's disease and pneumonia.

Somme, Joseph, New York (licensed in New York, year unknown); died April 28, aged 85, of arteriosclerotic heart disease.

Steinmetz, Bruno, New York; Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin, Prussia, Germany, 1920; died April 9, aged 57, of cancer.

Stewart, James A., Portal, Ga.; University of Georgia Medical Department, Augusta, 1906; died recently, aged 71, of uremia, hypertension, and arteriosclerosis.

Stripp, Albert Edward, Billings, Mont.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1904; formerly mayor of Laurel; served as city health officer; died May 7, aged 75.

Taddiken, Paul Gerald ⊕ Ogdensburg, N. Y.; Columbia University College of Physicians and Surgeons, New York, 1895; served on the staff of St. Lawrence State Hospital; died May 2, aged 78, of uremia, chronic nephritis, and hypertrophy of the prostate.

Templeton, Charles A., Glendora, Calif.; Rush Medical College, Chicago, 1895; died recently, aged 83, of cerebral thrombosis.

Thomas, Ernest, Quinton, Okla.; University of Arkansas School of Medicine, Little Rock, 1911; served on the staffs of the McAlester General and St. Mary's hospitals; died May 8, aged 69, of massive cerebral hemorrhage.

Thorne, John Mairs ⊕ Pittsburgh; Jefferson Medical College of Philadelphia, 1886; associate professor emeritus of medicine at University of Pittsburgh School of Medicine; specialist certified by the American Board of Internal Medicine; past president of the Allegheny County Medical Society; fellow of the American College of Physicians; consulting physician, Presbyterian and Woman's hospital; died May 5, aged 87.

Tilt, LeRoy Wortendyke Jr. ⊕ Shelby, N. C.; University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, 1943; served during World War II; affiliated with Shelby Hospital; died in Kings Mountain May 12, aged 33, of Hodgkin's disease.

Tinkess, Arthur Lewis ⊕ LaFargeville, N. Y.; Queen's University Faculty of Medicine, Kingston, Ontaria, Canada, 1898; served during World War I; died in Greenwich, Conn., April 6, aged 80, of arteriosclerosis.

Van Winkle, Charlotte C., Taunton, Mass.; Johns Hopkins University School of Medicine, Baltimore, 1921; affiliated with Taunton State Hospital, where she died May 9, aged 59, of adenocarcinoma.

Wales, Reginald C., Lincoln, Ill.; the Hahnemann Medical College and Hospital, Chicago, 1908; died in St. Petersburg, Fla., recently, aged 69, of carcinoma of the bladder.

Walker, Thomas Dick, Los Angeles; University of Tennessee Medical Department, Nashville, 1893; also an ordained Baptist clergyman; died April 13, aged 80, of intestinal hemorrhage and arteriosclerosis.

Whitehead, Vernon Erick, Blountsville, Ala.; University of Alabama School of Medicine, 1915; at one time on the faculty of his alma mater; died April 11, aged 61, of myocardial infarction and pulmonary emphysema.

Wickliffe, John Wakefield, West Union, S. C.; College of Physicians and Surgeons, Baltimore, 1888; University of the City of New York Medical Department, 1889; medical examiner for selective service during World Wars I and II; died April 23, aged 86, of cardiovascular renal disease.

Williams, Robert Bridgeforth, Huntsville, Ala.; Vanderbilt University School of Medicine, Nashville, Tenn., 1912; died in Tuscaloosa April 28, aged 67, of cerebral hemorrhage.

Williamson, Edwin Oliver ⊕ Gurley, Ala.; Chattanooga (Tenn.) Medical College, 1898; died recently, aged 79, of cerebral hemorrhage and arteriosclerosis.

Wollman, Cecilie Halberstadt, Philadelphia; Woman's Medical College of Pennsylvania, Philadelphia, 1917; physician for the board of education; died May 7, aged 57, of coronary occlusion.

Woods, William Edward ⊕ Highland Park, Mich.; University of Toronto Faculty of Medicine, Toronto, Canada, 1920; affiliated with Florence Crittenton Hospital, Detroit, and Highland Park General Hospital, where he died April 23, aged 58, of intestinal obstruction.

Woodward, Erle B., Monrovia, Calif.; University of Michigan Homeopathic Medical School, Ann Arbor, 1899; died May 7, aged 76.

Wyland, Asa O. ⊕ Underwood, Iowa; State University of Iowa College of Medicine, Iowa City, 1889; died in Council Bluffs, April 12, aged 85, of intestinal obstruction.

Yarow, Fannie Hutoryansky, New York; New York Homeopathic Medical College and Hospital, New York, 1922; a physician with the Russian Army during World War I; died May 8, aged 65, of a cerebral accident.

Young, Harvey Wade, Red Bank, N. J.; New York University Medical College, New York, 1897; died in Riverview Hospital May 31, aged 79, of carcinomatosis.

GOVERNMENT SERVICES

SELECTIVE SERVICE SYSTEM

Lists for Inductions.—The National Advisory Committee to the Selective Service System has issued "Information Bulletin," vol. 3, no. 12, which reads as follows:

1. Selective Service lists for induction.—The National Advisory Committee has been sending to the state chairmen lists of special registrants who have been issued orders for induction. We are informed by the Selective Service System that subsequent to the receipt of these lists some chairmen are declaring men so listed as essential and requesting that they be deferred.

After an order for induction has been issued, a local board may not reopen the classification or consider a case anew except by direction from the state director. Neither can a local board postpone an induction except when there is an extreme emergency over which the registrant has had no control. Therefore, the only recourse at this time is to make representation to the state director and explain to him the changed circumstances that necessitate the declaration of essentiality.

When the registrant fills an essential position where replacement is not available every effort should be exerted to keep the local board fully advised at all times concerning essentiality of priority 1 physicians and priority 1 and 2 dentists; otherwise the local board will retain the special registrant in a class available for military service and hold him available to fill a call.

If a special registrant has been ordered for induction and the state director of Selective Service does not grant a request from a chairman to direct the local board to reopen and consider anew a classification and the chairman feels that it is imperative that the special registrant not enter the military service, representation should not be made to the armed force to which the registrant has been allocated for commission with request for delay in orders to active duty. Instead, a full report of the case should be made immediately to the National Advisory Committee for its consideration and such action as it deems appropriate with the National Headquarters of the Selective Service System.

2. Advice on priority 3 classifications.—In some state offices this has presented itself as a considerable work load. For the present, until further ground rules can be laid down, the youngest in the group should be considered first, working up through the ages as far as is feasible with personnel available and the necessary considerations to be given to cases of priority 1 and 2 reservists and registrants or other correspondence requiring immediate attention.

Essential in Community Practice.—Item one of "Information Bulletin," vol. 3, no. 8, of the National Advisory Committee to the Selective Service System, released July 8, reads as follows:

> The Illinois State Advisory Committee is using a plan which the National Advisory Committee desires all State Advisory Committees to use in connection with declaring physicians and dentists essential to community practice where it is considered to be necessary to do so. Such men are declared essential for six months with the understanding that the Chamber of Commerce, mayor or whatever organization has supported the essentiality must show evidence in writing at the end of six months as to their efforts to obtain someone to practice in that community; otherwise, the deferment will not be renewed. Such evidence would be in running ads in medical journals, contacting placement agencies, etc.

NAVY

Courses for Reserve Officers in Malariology and Insect Control.—Two week training courses for Volunteer Naval Reserve Medical, Medical Service, and Hospital Corps officers in malariology and insect control will convene at the Naval Air Station, Jacksonville, Fla., on the first and third Wednesdays of October, November, and December. The instruction will provide information on techniques to be employed in insect control and practical field experience, which is not readily available to these officers in their civilian occupation. Inactive Volunteer Reserve Medical, Medical Service, and Hospital Corps officers are encouraged to take advantage of the opportunity to attend these courses on active training duty orders in a pay status. Quotas have been allotted to the commandants of the first, third, fourth, sixth, and ninth Naval Districts and the Potomac River Naval Command. Eligible officers, who so desire, should submit requests to the commandant of their home naval district. Meals and sleeping quarters will be available at the Bachelor Officers' Quarters for those officers desiring such accommodations.

Personal.—The Surgeon General, Rear Adm. Lamont Pugh, and his executive assistant, Lieut. Commander Roy T. Brooks, left Washington, Aug. 1, 1952, for an extended tour of Naval medical facilities in Alaska, the Far East, and the European-Middle East areas. Last year Admiral Pugh visited Naval medical activities in the Pacific Ocean area, Japan, and Korea; and earlier this year he visited medical activities in the Caribbean area.——Rear Adm. Frederick C. Greaves, Medical Inspector General, and his executive assistant, Lieut. Commander Edwin W. White, left Washington, Aug. 10, on an 18 day inspection trip to the Pacific Northwest and Alaska.

VETERANS ADMINISTRATION

New GI Education and Training Bill.—Veterans with service anywhere in the world since the start of the Korean conflict, June 27, 1950, may now apply for up to 36 months' education and training under the new Korean GI Bill, Veterans Administration has announced. While in training, these veterans may receive an allowance from the government to cover part of their expenses. Application forms are available at all VA regional offices. Photostatic or certified copies of all discharges from the armed forces since June 27, 1950, must accompany the completed application. VA cannot pay educational allowances for training for any period that begins prior to Aug. 20, 1952.

VA advises veterans to take great care in planning and outlining the program of education and training in the second section of the form, since VA will not approve more than one change of program under any circumstances. Veterans should make sure that the school or business establishment is approved by the appropriate state agency for training under the Korean GI Bill (Public Law 550, 82nd Congress). The bill prohibits VA from approving a program of education or training leading to an educational, professional, or vocational objective for a veteran who is already qualified for such objective. It also prohibits the approval of avocational and recreational programs.

PUBLIC HEALTH SERVICE

Regular Corps Examination for Bacteriologists.—A competitive examination for appointment of bacteriologists to the Regular Corps of the United States Public Health Service will be held on Nov. 4-6 at a number of points throughout the United States. Applications must be received no later than Sept. 23, 1952. Appointments will be made in the grades of Assistant Scientist and Assistant Sanitarian (equivalent to Navy rank of Lieutenant, junior grade) and Senior Assistant Scientist and Senior Assistant Sanitarian (equivalent to Lieutenant). Appointments are permanent and provide opportunities for qualified bacteriologists to pursue their profession as a life career in research and public health activities. Candidates must be United States citizens and at least 21 years of age. Application forms may be obtained from the Chief, Division of Commissioned Officers, Public Health Service, Washington 25, D. C.

FOREIGN LETTERS

BRAZIL

New Use for BCG.—Drs. Luiz Batista and Norberto Belliboni presented a paper based on observations of a case of lupus erythematosus of fixed type, which had been developing for two years and recently showed progression of the lesions. They observed that there was tuberculin hypersensitivity on dilution of 1:1,000 (0.1 mg.), in consequence of which they submitted the patient to oral treatment with BCG. Before this therapy they had administered vitamin C and sodium salicylate to the patient for a short period, without any improvement of skin lesions. For desensitization purposes they used oral BCG in a dosage of 0.2 gm. per week. After a total dose of 1.2 gm. appreciable improvements were affected in the dermatological features, and after a total of 1.8 gm. the lesions had disappeared nearly completely, without signs of intolerance by the patient. After reviewing the literature on the oral use of BCG, Drs. Batista and Belliboni stressed the desensitizing and immunity strengthening action of this vaccine, suggesting this kind of treatment in all cases of dermatoses hypersensitive to tuberculin. They cited their experience in this field with erythema induratum, disseminatus, and discoid, and scrofuloderma, papulonecrotic tuberculids, Darier-Roussy's sarcoids, and erythema pernio.

Surgical Diseases of Esophagus and Cardia.—Dr. Fernando Paulino and his co-workers recently published their experiences in the treatment of diseases of the esophagus and cardia. They presented 66 cases with the following distribution: cicatricial stenosis 26, carcinoma 18, cardiospasm 9, diverticula 3, congenital stenosis 2, acute esophagitis 2, peptic ulcer 2, fibrosis of the cardia 2, and congenital atresia and stenosis from undetermined cause, 1 each. Regarding preoperative care, the authors emphasized the importance of the correction of blood volume and anemia, as well as the necessity of a liquid hypercaloric and hyperproteic diet and the use of local and general antibodies. After the operation, during the first days, intravenous feeding is in order, soon afterwards combined with oral nourishment, to give the patient the liquids, salts, proteins, calories, and vitamins that he needs in accordance with the body weight. Anesthesia by ether-oxygen is administered with intratracheal tube with cuff. With the experience acquired through over 150 thoracotomies performed by the same surgical team, the authors base the amount of blood to be replaced on the estimated amount of blood lost, according to hematocrit determinations. The quantity of blood necessary during an esophagectomy does not go beyond 1,500 cc. in the average case. Two cases of congenital deformities treated successfully through dilatation and one case of atresia in which a termino-terminal anastomosis of the esophagus was performed were shown. The authors pointed out the necessity for making maternity and pediatric clinics conscious of the possibilities in the treatment of congenital deformities of the esophagus, which should always be suspected when a newborn infant has excessive mucus in its throat, breathes poorly, or has attacks of cyanosis. Cicatricial stenosis and cardiospasm that do not present difficulties are generally treated through dilatation by different methods. Surgery is indicated only exceptionally in cases of cicatricial stenosis and in about 20% of the cases of cardiospasm. Cardioplasty was performed in five cases with good results. Three patients with diverticula were operated on, two of them had the pharyngoesophageal variety, and a one stage diverticulectomy was performed. In one supradiaphragmatic diverticulum a right transthoracic diverticulectomy was done. In all cases the results were good. The left transthoracic approach was used for esophagectomies, high or low according to the situation of the tumor. The anastomosis is done with interrupted sutures. Clamps are never used. Patients are fed orally from the second postoperative day, and from the sixth day a bland diet is given. The authors stressed the necessity for an early diagnosis of cancer of the esophagus, in order to improve the results obtained by surgery.

LUXEMBURG

Interallied Committee of Medical Reserve Officers.—The Sixth Study Session of the Interallied Committee of Medical Reserve Officers, which includes the medical reserve officers of the Atlantic Pact countries, was held in Luxemburg under the presidency of Dr. Kremer of the Netherlands. The committee is composed of representatives of the federative unions of reserve physicians of the various countries, and in the course of the opening session Major Felton, M.D., Chief of the Health Service of the Army of Luxemburg, announced the formation of an association of reserve physicians of Luxemburg.

One of the aims of the committee is to clarify major questions in the treatment of wartime emergencies. The session took up the question of the treatment of burns. Dr. Kummer, Netherlands delegate, outlined the following scheme for the initial treatment. A burn is sterile and must not be infected. Clothing should not be removed from the burned person at the place where the injury occurred, because proper dressing of the injury is then impossible. A sterile dressing should be placed over open wounds, without causing pressure from the dressing. If the injury exceeds 15% of the body surface, the patient should be given antishock treatment, consisting of plasma, sodium chloride solutions and sodium citrate solutions by mouth, an antitetanus injection, morphine, and treatment with penicillin.

At a first-aid station antishock treatment should be given according to the seriousness of the case. If possible, the patient should be evacuated rapidly while penicillin therapy is continued; the urine should be examined. If the patient is kept at the first-aid station, results of the antishock treatment should be awaited before beginning local treatment. The patient should be undressed, placed on sterile compresses, and treated for relief of pain with morphine or light anesthesia with ether. Surgical débridement of the wound and ablation of the necrosed tissues should be carried out; dressing with sterile compresses, toes and fingers separated, should be applied. Immobilizing plaster casts permitting freedom of movement for toes and fingers are necessary. Grafts should not be applied at the first-aid station. Hydration should be maintained by drinks in adequate quantities; in the ensuing period of from 2 to 10 days, the patient should be evacuated to a specialized hospital, sending along a detailed report of the treatment given. Colonel Vandermeiren, M.D., of the Belgian Army, dealt with the problem in the capacity of a military medical expert and a sanitary tactician. He emphasized the importance of the standardization of the first aid to be given and the necessity of evacuating the patient to a sanitary establishment where he can be kept, because rest plays a large part in the efficacy of therapeutic measures. He insisted on the value of systematic organization of the examination and treatment.

Dr. Koch (Amsterdam) showed a series of slides illustrating the operative techniques to be used in correcting facial cicatrices. Professor Menegaux (Paris) reviewed the various complications that may be observed in the course of cicatrization, such as hypoproteinemia and death, and discussed the treatment of cicatrices, dwelling in particular on certain points of technique concerning the hand and fingers. Functional re-education of the burned patients should be undertaken as soon as possible.

Colonel Tugan (Turkey) advocated the slow injection of concentrated plasma in the treatment of hypoproteinemia. Professor Cinerei (Italy) emphasized the economy that can be secured in the use of plasma by replacing it in part with alkaline solutions by mouth and especially by the use of modified horse plasma. He pointed out the useful effects of subcutaneous oxygen therapy and the use of arterial injections of penicillin-novocaine in burns of the members as well as the ease of compress dressing with gelatin sponges.

Following these expositions, General Glorieux, M.D., Belgian Army, summarized all the present information in the form of a syllabus, which was adopted by the committee and can be used as an instruction manual for all physicians.

The items in these letters are contributed by regular correspondents in the various foreign countries.

PARIS

International Youth Center.—Under the direction of Prof. R. Debré, member of the National Academy of Medicine, the activities of the International Youth Center are progressing. During 1952 two exhibitions intended for public education took place: "The First Year of Life" and "The Hazards of Tuberculosis and the Child." Social investigations on various questions concerning the child's welfare are undertaken under the name of "Mégève Colloquies." Other investigations on the lack of maternal care are simultaneously pursued in Paris and London. An important documentation center and library with microfilms and photocopies has been organized at the Longchamp Center. The center publishes a monthly journal, *Le Courrier,* and is preparing a quarterly review, *Etude néo-natale.*

The scientific works performed by the center's pilot station are pursued in the fields of pertussis and BCG vaccination. On Feb. 18, 1952, a conference of the study group for antituberculous vaccination was held at the Longchamp Castle. It was attended by several French and foreign personages, among whom were H. J. Ustvett (Oslo), C. Palmer, T. H. Madsen, and Miss L. Edwards (all from Copenhagen), and D. Thomson and J. Fanconi (Switzerland). Since 1950, the pilot station has undertaken a series of laboratory and clinical examinations of the technique of BCG vaccination. Interest was shown in the Trambusti test, performed with a needle with automatic penetration as a unique method for mass vaccinations. The method of intradermal injection of dead bacilli gave very severe local reactions at the pilot ation, which led them to discard it. It is believed that a scarifica n through a drop of BCG killed at 100 C would avoid this inconvenience. The systematic radiological examinations of children vaccinated at the Lille Pasteur Institute permitted observation in children of hilar-pulmonary images. A device is under study that would make it possible to obtain allergy of sufficient degree and duration, together with a minimum of ganglial lymph node complications. The problem is to find the precise doses to be given according to age as well as the proper time and dose for revaccinations. In the course of the discussion, C. Palmer reported that the observations at Copenhagen indicate that a very noticeable reduction of the dose of vaccine does not modify appreciably the rate of the postvaccinal allergy. He also stressed the effect that exposition to light exerts on the vaccine by diminishing the number of living germs it contains, a possible cause of discordance between the various results obtained. Professor Sarrouy (Algeria), J. Gaud (Morocco), and J. Goujou (Tunisia) stated that for two years the number of vaccinated subjects was 1,007,000 for Morocco, 264,000 for Tunisia, and 295,000 for Algeria. A practical question, which has not yet been resolved, is the necessity of associating smallpox vaccination with BCG.

Scientific Program.—The pilot station will continue to deal with the following questions: (1) systematic follow-up of subjects previously vaccinated with BCG, (2) the relation between the natural disposition to develop a strong allergy (which appears to be partly hereditary) and the resistance or sensitivity to tuberculous immunity existing in certain families, a question that will be studied in Paris and certain large towns of North Africa, (3) the reactions of mediastinal glands occurring in vaccinated persons who will submit to monthly systematic radiological controls from the third to the eighth month following vaccination, and (4) comparative study of BCG vaccine of various origins, dry vaccines and vaccines originating from cultures maintained in bile containing medium.

The program, in the process of being carried out, was presented by M. Tubiana: (1) to study the reaction of organisms as regards BCG and BK, both tagged by radioactive isotopes, (2) to find out in guinea pigs whether the resistance to superinfection only persists as long as allergy exists, (3) to study the effects exerted by injections of BCG in rabbits treated with corticotropin (ACTH) or cortisone, (4) to search for the presence of tuberculous antibodies by the reaction of hemagglutination, and (5) to try to sensitize healthy animals by the injection of tuberculin fixed on human red blood cells.

To coordinate scientific work, it was decided that organizations such as the Medical Research Council (of Great Britain) and the pilot station will come to an agreement on the repartition of the subjects to be studied.

SWEDEN

Birth Rate and Abortion Problems.—As recently pointed out by Prof. Sven Brisman, there are few countries in the world, if any, with a more widely fluctuating birth rate than Sweden's. The remarkably low birth rate in the 1930's led to a series of remedial measures by the government. There was a low record in 1935, with only 86,000 live births, and for the next few years Sweden's birth rate was the lowest in its history. World War II suddenly sent the birth rate up, and in 1945 there were as many as 135,000 live births. Since this year there has been a fall of the birth rate, and in 1951 the number of live births was only 110,000.

This remarkable fall in the Swedish birth rate gives added importance to the high legal abortion rate. In 1950 in Stockholm there were 11,587 live births, and in the same year and city 1,790 legal abortions were induced. More than 30% of the 6,000 legal abortions induced in 1950 in the whole of the country were in Stockholm. These legal abortions are being scrutinized, and reference has already been made in THE JOURNAL (**147:**1775 [Dec. 29] 1951) to an investigation into the psychological reactions of the women for whom abortions have been induced. A more recent investigation has now been undertaken by a psychiatrist, Dr. Martin Ekblad, who has had personal conversations with women for whom abortion had been induced at the Sabbatsberg Hospital. They were questioned directly after the operation while still in the hospital and also after a considerable interval. One-third of the women for whom legal abortion was undertaken became pregnant again within a short time, 20% of them within 12 months of the abortion. Hence, Dr. Ekblad concluded that it is impossible to solve the abortion problem before the problem of teaching the prevention of conception has been successfully solved. Regrets and a sense of guilt were expressed by 21% of the women he questioned, whereas 79% expressed satisfaction with, and gratitude for, the operation they had undergone.

Vanishing Tuberculosis Mortality Under the Age of 50.—The counties of Uppsala and Södermanland have lately given statistical information about the age distribution of persons who die of tuberculosis. Prof. Erik Hedvall and Dr. Olle Hillerdal noted that practically all the tuberculosis deaths in 1951 in the town of Uppsala occurred in persons over the age of 50. Thereupon they investigated the age distribution of all the tuberculosis deaths in the county of Uppsala between 1943 and 1951. In 1943 there were 48 tuberculosis deaths in persons under the age of 50, 15 such deaths in those between the ages of 50 and 70, and 5 such deaths in those over the age of 70. In 1951 there were only 3 tuberculosis deaths in persons under the age of 50, 8 such deaths in those between the ages of 50 and 70, and 11 such deaths in those over 70. In other words, the fall in the tuberculosis mortality in the period under review depended almost entirely on the age group 0-50 years. Professor Hedvall and Dr. Hillerdal discuss five alternative or complementary explanations for this state of affairs; it may be due to the ever extending BCG vaccination, diminished infection, modern chemotherapeutic and antibiotic treatment, advances in chest surgery, or the change in the age distribution of the population with a relative increase in the number of elderly persons dying of tuberculosis. With regard to the third point, it should be noted that extensive use was not made of modern chemotherapeutic and antibiotic treatment for tuberculosis till 1948. This treatment has undoubtedly helped to reduce the tuberculosis mortality, but its fall was already demonstrable in 1947. With regard to the fourth point, instructive light is thrown on it by Dr. P. Rudström, who quoted statistics published by the Swedish National Association Against Tuberculosis. They show that in 1945 the number of patients with pulmonary tuberculosis operated on in Sweden was 719. In 1950 the corresponding figure was 1,616. In a survey of 300 patients operated on in a surgical hospital in Uppsala during the period 1948-1951, Dr. Rudström has pointed out that there were only 8 fatalities among them. In an earlier period, 1929-1932, the operation mortality for this hospital was as high as 23%. Since then it has fallen steadily, and at the present time patients as well as physicians in Sweden are often keen to extend the indications for operative treatment.

TURKEY

Isonicotinic Acid Hydrazid Therapy in Tuberculosis of the Lung.—Phthisiologist Dr. Tevfik Ismail Gökçe, medical director of the 530 bed Istanbul-Heybeliada Tuberculosis Sanatorium, presented a report to the Istanbul Tuberculosis Society concerning the results obtained in 10 patients who were given "nydrazid" (Squibb & Sons) therapy, in 12 patients given "nicotibin" (Lepetit Laboratory), and in 9 patients given "rimifon" therapy (Hoffmann-La Roche Inc.) during last March.

The selected patients had extensive active tuberculosis with a profusion of Koch bacilli. The majority of the patients had caverns in both lungs and were febrile. They failed to respond to streptomycin and other antibiotics. Of the 31 patients 20 were women and 11 were men. Twenty-five patients were 18 to 30 years old, and six patients were 30 to 60 years old. In 25 patients both lungs were affected, and the majority had exudative forms, in 6 patients one lung was affected, 17 patients had caverns in both lungs, and 10 patients had caverns in one lung. In six patients pneumothorax had been of no avail. Ten patients had pneumoperitonitis, of whom two patients had hydropneumothorax in one lung, one patient in both lungs, and one had pneumothorax in one lung and extrapleural pyopneumothorax in the other lung; one patient had pyopneumothorax and an external fistula as the result of pneumonectomy; one patient had tuberculous enteritis; and one patient had tuberculous laryngitis as a complication.

Of 31 patients 28 had previously been given streptomycin therapy; of these, 19 patients were given streptomycin and *p*-aminosalicylic acid therapy and 1 patient was given streptomycin, *p*-aminosalicylic acid, and amithiozone. Seven patients were given 10 to 30 gm. of streptomycin, nine patients 31 to 60 gm., six patients 61 to 90 gm., and six patients 90 to 150 gm.; streptomycin resistance developed in eight of these patients.

Before the new therapy was begun, all patients were given a thorough check-up, the sputum was reexamined, blood counts, formula, and sedimentation rate redetermined, urinalyses made, kidney and liver function investigated, and new roentgenograms taken. These investigations were repeated every 10 days during the therapy and at its termination. The patients were given a 50 mg. tablet of isonicotinic acid hydrazid four times daily and a 100 mg. tablet twice daily, an average of 3 to 5 mg. per kilogram of body weight. At the initiation of the therapy 1 of the 31 patients was subfebrile and 24 patients were febrile; of these 11 patients had been so for 12 months, 6 patients for 3 months, and the other patients for 4 to 6 weeks. In 19 of the 24 patients the temperature subsided, and in 5 patients it continued. In 27 patients coughing decreased, and in 4 patients it remained unchanged; in 24 patients the amount of sputum decreased, and it remained unchanged in 7 patients. At the initiation of the therapy, the sputum of the majority of the patients revealed a profusion of tuberculosis bacilli examined by direct method. After a month of therapy, the sputum of 27 of 29 patients had negative results by direct method; the result was positive in 18 patients by homogenization method and negative in 9 patients. These nine patients, or 31%, also had negative reactions by culture method.

The general conditions of the patients were markedly improved. In patients who had previously steadily lost weight, the appetite improved and was followed by an immediate gain in weight; two patients developed a ravenous appetite. Of 31 patients, 3 lost weight during the therapy and 28 gained weight. Of the latter, 4 patients gained 200 gm. to 1 kg.; 8, 1 to 2 kg.; 9, 2 to 3 kg.; 5, 3 to 4 kg.; 2, 4 to 5 kg.; and 1, 7 kg. 700 gm. during the one month of therapy. The average gain was 2.5 kg. per patient. Roentgenographic evidence showed no change in 24 patients. There was improvement of variable degrees in seven patients, as evidenced by regression of exudative processes, reduction in the size of caverns, and the absence of cavities by direct radiography. These improvements occurred in patients with new fresh lesions. In one patient the radiological symptoms increased, and new nodules and lesions developed. An exudate developed in the patient with pneumothorax. In the patient with tuberculous enteritis as a complication the diarrhea subsided. In the patient with tuberculous laryngitis as a complication of 14 months standing the infiltration in the arytenoid region and in the left plica vocalis subsided after a week's therapy, difficulty in swallowing disappeared, the voice began to regain its natural tone, and laryngitis had completely disappeared at the end of the month.

Except in one patient, undesired and toxic reactions indicating discontinuation of the therapy did not occur. Reflexes increased in five patients, two of whom had vertigo; diarrhea developed in three patients and eosinophilia in two. One patient had a rise in blood pressure, and one had a decrease. In one patient the sense of hearing was diminished; one patient had skin eruptions and itching, and one patient had vertigo. All these side-effects subsided after a few days. In the patient who had an external fistula as a result of pneumonectomy and also had amyloid degeneration the albumin in the urine increased from 6 gm. per liter to 9 gm. and the edema increased; the therapy was discontinued.

Terramycin Therapy in Trachoma.—In *Eye Clinic,* an Istanbul periodical, ophthalmologist Dr. Izzet Bilger has published his observations on the effectiveness of terramycin on trachoma on 700 patients in the Adana Trachoma Hospital and Dispensary. Terramycin hydrochloride was administered per os, 250 mg. in capsules, 0.1% and 0.5% ointment, and 0.5% solution of eye wash. Of 38 patients with acute trachoma 27 patients recovered after 3 to 6 weeks and 11 patients after 5 to 10 days. Twenty-seven patients who were given terramycin orally, ointment, and eye wash recovered after 20 days, but slight gastrointestinal irritation developed. Patients with chronic trachoma needed from two to three months therapy for complete recovery, and to prevent a relapse the therapy was continued for another month. A combination of chemotherapy and surgical intervention in chronic and complicated cases accelerated recovery.

From a special school for children with trachoma, 640 children who had chronic trachoma were selected and divided into three groups. The patients in group one were given terramycin only, in group two terramycin and chemotherapy, and in group three terramycin, chemotherapy, and surgical treatment. In almost all patients bleeding and suppuration decreased in from one to two weeks, and after from two to six weeks 20% of the patients had recovered. After 12 weeks 80% recovered, but therapy was continued for another month after recovery. In seven patients the symptoms increased at first; two of these patients improved as treatment was continued, but in the other five patients there was no improvement even after 12 weeks' treatment. Two of 34 trachoma patients with acute pannus and corneal complications were given 2 gm. of terramycin for three days; acute symptoms subsided on the fourth and fifth day. Twenty-eight patients were given ointment and eye wash therapy. Pain and photophobia subsided after 24 hours, cornea complications subsided after 6 days, and in most of these patients vision improved. Four patients with pannus hypertrophy recovered in from 10 to 15 days.

Five of 13 hospital patients, aged 13 to 62, had trachoma and corneal complications, 1 had trachoma and acute flux, 1 had florid trachoma, the other 6 had corneal ulcers and infiltration, and 1 had in addition follicular conjunctivitis. With the exception of one patient subjective symptoms began to subside after from 24 to 48 hours after the initial treatment, and in from 3 to 5 days there was marked improvement. Of six dispensary patients that were followed up, three children, 1, 3, and 4 years old, had acute trachoma and severe ophthalmia in both eyes; acute trachoma in two patients, 17 and 28 years old, was complicated by follicular conjunctivitis and flux; and a 50-year-old patient had advanced trachoma and flux. Four of these six patients were given terramycin ointment for one eye and eye wash for the other eye; the patients that were given ointment recovered sooner. With the exception of one patient with hypopyon corneal complication, the hospital patients recovered in from 9 to 24 days. Dispensary patients recovered in from 3 to 5 days. In 11 dispensary patients with trachoma complicated by severe ophthalmia very good results were obtained with terramycin ointment therapy. Terramycin was well tolerated by all patients.

Turkish Red Crescent's Health Program.—To further the health of the nation, the Turkish Red Crescent Society has set aside from its budget 4 million Turkish pounds for the year 1952. According to Prof. Ekrem Hayri Üstündag, the minister of health and social assistance, who also is a member of the society's central committee, 300,000 pounds are to be spent for the construction of a 100 bed preventorium-sanatorium for university students, as an annex to the Istanbul-Erenköy tuberculosis sanatorium, 400,000 pounds for the construction of a 100 bed maternity hospital as an annex to the Izmir Children's Hospital, 300,000 pounds for the construction of a 100 bed tuberculosis hospital in Adana, 400,000 pounds for the construction of a 50 bed hospital in the town of Mus, population 45,349, and the eastern province near Lake Van, and 200,000 pounds for establishment of each health center providing medical care and free medicaments in nine eastern frontier districts. There will be 450,000 pounds spent for the construction of a cancer research institute in Ankara, and 320,000 pounds are to be spent for the purchase of 12 mobile units for operation in the eastern and southeastern trachoma campaign areas. The Red Crescent will participate in the trachoma campaign by supplying terramycin and other antibiotics. There are still 250,000 persons who have trachoma. When these constructions have been completed, the Turkish Red Crescent Society will turn them over to the government for administration by the ministry of health and social assistance.

CORRESPONDENCE

CHRONIC URETHRITIS IN GIRLS

To the Editor:—I should like to take issue with Dr. Hodges' statement in THE JOURNAL, June 21, 1952, in his article "Chronic Urethritis in Girls." On page 756, he states, "One does not need the services of a urologist to diagnose the ordinary case of chronic posterior urethritis. The history of recurring episodes of urinary frequency, enuresis, fever, and pyuria is highly suggestive."

It is the opinion of this urologist that recurring urinary frequency, fever, and pyuria in a child are not suggestive of urethritis but of upper urinary tract infection, and a complete [illegible]cal study is strongly indicated.

[illegible]e were to heed Dr. Hodges' advice, many a case of hydronephrosis or other types of renal disease would progress to serious proportions before necessary steps for proper therapy would be taken.

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REACTIONS TO ISONIAZID AND IPRONIAZID

To the Editor:—Isoniazid (isonicotinic acid hydrazide) and iproniazid (1-isonicotinyl-2-isopropylhydrazine) are being given to many patients with tuberculosis throughout the United States and in many other countries. Toxic reactions to both drugs have been reported and, undoubtedly, more will occur. However, it is unlikely that any single investigator will observe enough of these toxic reactions in the next few months to warrant publication. The Committee on Therapy of the American Trudeau Society is anxious to collect reports of these toxic reactions, so that a consolidated report can be made available to the medical profession as soon as possible.

Any physician who is willing to submit such reports to the committee is asked to send a brief case report to the Committee on Therapy, American Trudeau Society, in care of Dr. D. T. Carr, 102-110 Second Avenue Southwest, Rochester, Minn. The report should include the patient's name or initials, his age, sex, race, diagnosis, weight, the drug administered and its dosage, the duration of treatment, and a list of other drugs being given at the same time. The toxic reaction should be described fully together with information as to its recurrence if the same drug was given later.

Copies of the consolidated report will be sent to all contributors, but will not be published in such a way so as to preclude individual publications.

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GRAVITY RESISTING ABILITY OF THE CIRCULATION

To the Editor:—Some new drugs for the treatment of high blood pressure cause a decrease in the pressure on standing to the point of dizziness and fainting, i. e., orthostatic hypotension. This is to be avoided. The recommendation of makers of such drugs for study and selection of cases is good. It is suggested that a method for estimating the gravity resisting ability of the circulation, i. e., contra-orthostatic-hypotension ability, introduced many years ago, may be utilized for this purpose. This test consists in taking both the blood pressure and the pulse rates with the patient standing and lying.

The fall of the blood pressure and the rise of the pulse rates on standing indicate weakness. This was reported in 1904; it was picked up by Schneider at Mineola in 1916, and the two items were included in his six-point test. This test will show the presence and degree of orthostatic hypotension. It is not generally known that blood pressure can fall from 30 to 100 points on standing, and that a man can and sometimes does have high blood pressure in bed, and low blood pressure standing. The prognosis is far better for the man who has no postural fall in pressure, as the records show.

Thus we have a method of discovering and recording the gravity resistance ability of the individual. Although hitherto somewhat neglected, this method will give a clue to the efficiency of the splanchnic vasotone. This test may aid not only in therapy and clinical management but also in better understanding of the disease and, what is more, of the patient.

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FOREIGN GRADUATES AS INTERNS

To the Editor:—During the past year, with the cooperation of the Office of the High Commissioner of Germany, the State Department, and cooperation of doctors and hospitals in New Jersey, sixty-five carefully screened recent graduates of foreign medical schools have been placed in A. M. A.-approved rotating internships in New Jersey. Upon the completion of the internship they must return to their homeland.

Word of mouth reports on this experiment have apparently gotten around fairly well in medical centers abroad so that almost daily we receive applications for residencies here in America. Only today we received a request from a surgeon in India who has a master of science degree in surgery from the University of Michigan asking us to attempt to place him as a resident in surgery at a second year level. A member of the United Nations Permanent Secretariat from India recently urged us to interest ourselves in some of his fellow countrymen whom he would personally guarantee as going back into excellent teaching positions where they could have a definite impact on the political, cultural, and medical conscience of India.

If any hospitals or doctors are interested in this experiment and have any approved residencies open, we will be glad to pursue these opportunities further.

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COUNCIL ON MEDICAL SERVICE

This is the third in a series of studies made by the Committee on Indigent Care of the Council on Medical Service concerning local plans for medical care of the indigent. A general introduction to the series and the first of the Committee's studies (Erie County, New York) appeared in the May 10, 1952, issue of The Journal, *pages 188-191. The second study (Madison, Wisconsin) appeared in the July 26, 1952, issue, pages 1247-1249.*

MEDICAL CARE FOR THE INDIGENT IN NEWARK, NEW JERSEY

This report concerns the medical care available for indigent residents of Newark in Essex County, New Jersey. The county has approximately 905,000 residents, of whom 439,000 live in Newark, the state's largest city. Newark is highly urban and industrialized. It is located in the eastern part of New Jersey, within the New York City metropolitan area.

ELIGIBLE POPULATION

General assistance programs are administered by Newark itself, while the public assistance programs are county-wide in scope. During the year 1950, approximately 13,150 persons in Newark, or about 3% of the city's population, received aid under the city programs. About 8,730 residents, or about 1% of the county population, received aid from Essex County public assistance agencies. The number of city residents receiving public assistance aid is not available; however, if the percentage of the population receiving aid is assumed to be the same for city and county, a total of 4% of the city's residents or approximately 17,692 persons received aid under the combined programs. Table 1 shows the distribution of the recipients of aid among the various assistance programs. Because of the differences in administration and population covered, general assistance and public assistance programs will be discussed separately.

Table 1.—*Distribution * of the Recipients of Aid Among the Assistance Programs, 1950*

General Assistance (Newark residents only)	
Indigent (mean of relief rolls for year)	8,704
Medically indigent (cases treated)	4,446
Public Assistance (whole of Essex County)	
Old Age Assistance	5,000
Aid to Blind	209
Child Welfare (1,517 families)	3,521

* No allowances have been made for duplication of coverage.

GENERAL ASSISTANCE

Administration.—In Newark, medical care for the indigent (those actually on relief rolls) is provided through the department of public welfare and for the medically indigent (persons able to provide the basic necessities for their own subsistence but unable to pay for medical care) through the Newark Health Department. Both departments are part of the city's division of public affairs, headed by the director of public affairs, who is one of the five city commissioners. Both departments report to this director, and all budgets and emergency appropriations must be approved by him and by other city commissioners.

The director of welfare and the health officer exercise immediate supervision of their respective departments. However, policies and procedures of the welfare department are subject to the approval of the local assistance board, appointed by the director of public affairs. The welfare department is under the general supervision of the New Jersey State Department of Institutions and Agencies and submits reports to this body as well as to the division of public affairs. Each department is in charge of planning, determination of eligibility, authorization of treatment, special procedures, and review for its own medical program.

The welfare department maintains a central clearance division, which provides information concerning eligibility to the city hospital, the medical social service department, the health department, and the Medical Service Administration (the agency through which payments are made to private physicians). The two latter agencies are continually advised of status changes as they affect the clients' eligibility for services provided. All clients applying for medical assistance are cleared through this division, and any assistance given by the county welfare board to Newark residents is also noted and passed on to the investigators concerned.

Indigent cases are reviewed every two weeks to determine continuing eligibility, unless the client is permanently disabled. These cases are reviewed monthly by the department's case workers. The health department has a small staff of medical social workers who check on the medically indigent patient's ability to pay. New patients who are not on the welfare department's relief rolls are personally interviewed; in addition, spot-check home visits are made, especially for home care by physicians. If the physician suggests that the patient is able to pay, the department investigates further and bills those clients who are ineligible for free care.

Bills from private physicians are examined carefully; if a charge seems exorbitant, payment is withheld and the bill is reviewed by the Essex County Medical Society. Indigent patients may appeal to the state welfare agency; medically indigent patients must appeal to the Newark Health Department.

Services Available.—Indigent and medically indigent patients are eligible for the same medical care, through the same facilities. Patients may receive home care from a private physician of their choice, provided the physician has agreed to work with the program. However, since ambulatory patients are generally treated at the health department clinic, the free choice system functions primarily for home visits. The clinic provides all special medical services as authorized by the department from which the patient receives aid.

Other special services are provided by 24 neighborhood clinics, used for well-baby stations, tuberculosis examinations, and prenatal and dental clinics; nine neighborhood dental clinics for treating both adults and children; and a main dental clinic, which provides treatment for children three days a week and adults two days. The health department maintains an allergy clinic, to which patients are referred from the dispensary clinics and from other allergy clinics when they cannot afford the usual clinic fees. Neuropsychiatric treatment is available at the city dispensary.

The welfare department also maintains a medical center, which does not provide treatment but was initiated so that case workers could maintain current adequate knowledge of the medical status of each client. Cases are discussed individually at weekly conferences between the medical director and the case supervisors. The medical staff is composed of a nurse and four physicians loaned by the health department and one psychiatrist; non-medical personnel are provided by the welfare department. Authorizations for special medical service for welfare clients must be approved by this center.

Patients may receive treatment at the outpatient departments of the private hospitals at their own expense, at a fee of 50¢ to $1.00 per visit. Hospitalization, when recommended by a physician, is provided at Newark's City Hospital. If the city hospital has no vacancies, patients are sent to private hospitals in the city. The hospitalized patient does not have free choice of physician. Clients may receive home nursing care from the visiting nurse association or convalescent care from Newark nursing homes, when recommended by a physician.

Prescriptions are filled at the health department pharmacy. When this dispensary is closed, emergency prescriptions may be filled at private pharmacies at the authorizing department's expense. Medicines recommended by the physician and not available through the city dispensary are purchased by the department involved. When recommended by physicians, special diets are provided for the ill through an accredited dietitian who determines the food needs of the patient. Hospital surgical appliances, orthopedic shoes, eyeglasses, and other appliances are provided when necessary for the health or rehabilitation of are paid for prescriptions filled for clients of the programs, although physicians are requested to avoid prescribing expensive proprietary drugs unless necessary for treatment. In the child welfare program, the pharmacist may either bill the board of child welfare directly or charge the mother or foster mother, who will then be reimbursed by the board.

Costs and Financial Support.—Funds for all programs are provided by state and county appropriations and by federal subsidy, except that no federal funds are received for the care of children who are wards of the state. New Jersey supplies 87% of the funds for the old age assistance program; in the Child Welfare programs state and county pay an equal share of the cost.

Hospitalization as a part of the New Jersey old age assistance program is provided only in Essex and Gloucester Counties, where the boards of freeholders make appropriations directly to the county welfare board for such service. In the other counties, old age assistance patients are hospitalized through general assistance programs, as are aid to the blind clients in Essex County. Dependent children are hospitalized through board of child welfare funds.

Appropriations are made annually and emergency appropriations can be made if insufficient funds are provided. The county welfare board, in addition, has the right to insure anything of value belonging to an old age assistance client and put the insurance policy into trust. This money can be used for burial expenses and for partial reimbursement of the program. Detailed cost figures are available for the old age assistance program only. The expenditures for this program in 1950 are shown in the following tabulation:

Physicians	$ 66,622.90
Visiting Nurse Association	7,194.68
Pharmacists	64,412.99
Clinics	12,688.01
Other medical expenses (dental, eye, chiropody, etc.)	15,477.81
Total medical expenses	$167,196.39
Hospital (may include convalescent care)	235,000.00
Total cost	$402,196.39

The aid to the blind program had a monthly medical expense of approximately $249.73, or $2,973.00 per year. The child welfare program does not have cost figures available for Essex County.

Using these figures as a basis, the annual cost of physician's care per person eligible for old age assistance was $13.32 in 1950 and the total cost was $80.44 per person. Annual medical expenses of aid to the blind per person eligible was $14.22. Since these figures refer to those eligible for care rather than to the actual number of patients treated, no analysis of cost per case is possible.

Medical Society Relationship.—The Medical Society of New Jersey is studying methods of facilitating the payment of private physicians for care to old age assistance clients. The state has requested the society to develop some plan, similar to the Medical Service Administration, which will enable the state to reduce the bookkeeping and supervision required in making separate payments to individual physicians.

The Essex County Medical Society has entered into active cooperation with all three programs. It attempts to stimulate physician participation and acts as a board of review for all bills submitted by physicians and for complaints concerning alleged abuses by physicians.

The county society has also entered into an agreement with the state board of child welfare, through the society's child welfare advisory committee, to facilitate the care of dependent children. Before 1948, the state negotiated with the individual physicians who provided this care; beginning with that year, the society has provided and kept up to date a list of all members who agree to treat child welfare patients. The list is distributed by the district office to all child welfare clients. The society's child welfare committee also acts as an advisory or liaison committee to the state board; it reviews bills, hears complaints against physicians, and provides professional consultation and advice at the request of the district supervisor of the program.

SUMMARY

Indigent and medically indigent residents of Newark receive medical care through a variety of programs and a variety of agencies. Medical care for general assistance clients is provided under the supervision of city departments, while public assistance is provided through county-wide programs by county offices under state supervision. Benefits, payments, and method of payment differ somewhat from one program to another but, in general, much the same services are provided in all programs.

No single agency is responsible for over-all supervision of medical care in Newark, but the central clearance division of the Newark Welfare Department maintains records on all recipients of medical aid, whether from city or county offices, and notifies city departments of the status of each client. In the absence of a unified program, this office serves to provide some coordination of aid for city residents.

The major point of interest in the Newark plan is the Medical Service Administration, which serves as an intermediary between the private physician and the departments providing general assistance. This agency makes possible free choice of physicians for home care, with a reasonable reimbursement for the physician, yet keeps control of this care firmly in the hands of the medical profession. The agency has provisions for the review of bills and for hearing complaints, whether from the physician or the welfare authorities. It keeps involvement of the physicians in red tape to a minimum.

The Medical Service Administration is designed to serve as a state-wide agency, though at present it is operating only in the Newark area. The state old age assistance authorities have suggested to the state medical society that a similar plan be formulated to handle payments for care of the aged.

An important factor in providing proper care in Newark is the guarantee that a physician will receive payment for his first visit to a patient, whether or not the patient is eligible for free care. This rule assures immediate treatment of emergency cases without the delay necessary to check the welfare status of the patient.

Provision is made for utilizing all types of medical facilities in the city, although in the general assistance and child welfare plans primary emphasis is placed on use of the City Hospital and Dispensary. The provision for convalescent care of old age assistance patients in New York nursing homes when Essex County homes have no vacancies is particularly noteworthy.

The Essex County Medical Society, through advisory and liaison committees, supervises the quality of medical care and reviews bills to determine whether fees are fair to physicians and to the welfare programs. Participation in the programs is, however, uneven. All physicians are willing to treat old age assistance clients, while only 17% of the county's physicians have agreed to handle child welfare cases, and only 7% of Newark's physicians handle the city's general assistance cases.

The program as a whole has an obvious disadvantage in its lack of unified administration. General assistance is provided at a city level, through two city agencies; public assistance is provided at a county level, through two county agencies and the state authorities. At least three separate investigating staffs are maintained to determine eligibility; authorization for care, depending on the program, may be at a city, county, or state level, as is payment of bills. The Medical Service Administration, organized for state-wide service, is used only in Newark and only for general assistance payments, although it appears to be an efficient agency to handle payments to physicians for all programs.

There also appears to be some confusion in the benefits offered by the various programs. One program allows the use of private clinics only, another allows the use of both private clinics and the Newark City Dispensary, while another allows private clinic care only at the patient's own expense. Some patients will find it necessary to receive aid from both general and public assistance programs in order to obtain complete medical care.

In general, however, despite the multiplicity of agencies, the indigent resident of Newark seems to have available all medical services necessary for treatment and rehabilitation, with a minimum of delay between request for care and first treatment.

COUNCIL ON NATIONAL EMERGENCY MEDICAL SERVICE

The Council on National Emergency Medical Service has authorized publication of the following statement.

C. Joseph Stetler, *Secretary.*

WILLIAM L. WILSON PROMOTED

On July 24, 1952, Col. William L. Wilson, Assistant Administrator, Federal Civil Defense Administration, was promoted to the rank of brigadier general.

In his position as head of the Health and Welfare Division of the Federal Civil Defense Administration, General Wilson has, for more than a year, worked diligently at the difficult task of educating the country and the medical profession in the medical aspects of civil defense. His technical knowledge, his personal enthusiasm for his work, and his cooperation with the Council on National Emergency Medical Service have been largely responsible for the encouraging progress that has been made in medical civil defense preparedness throughout the country.

On behalf of the medical profession, the Council on National Emergency Medical Service would like to congratulate General Wilson on his promotion and express the hope that the Council and the profession will continue to have the benefit of his knowledge and his efforts in medical civil defense work.

MEDICAL MOTION PICTURES

Rheumatoid Arthritis of the Spine: (PMF 5163) 16 mm., black and white, sound, showing time 34 minutes. Produced in 1951 by the U. S. Army. Procurable on loan from the Area Surgeon of the Army Area in which the request originates.

This film begins with a discussion of the various terms used in describing rheumatoid arthritis and then describes in excellent fashion, by means of animated diagrams, the pathological lesions that exist. This is followed by good pictures of the roentgenogram findings, and then the physical findings are demonstrated. Finally the management of the type that begins with an acute onset is described carefully and satisfactorily. There is adequate attention to the employment of physical therapeutic procedures, medication is discussed briefly, the importance of rest is stressed, and different physical therapeutic procedures are carefully outlined and graded during the various stages of recovery. The value of x-ray therapy is mentioned and proper diet and supportive measures discussed. The use of transfusions to overcome anemia is mentioned, and the place of cortisone and corticotropin (ACTH) in management of rheumatoid arthritis of the spine is also described briefly. Next the discussion turns to the diagnosis of rheumatoid arthritis of the spine that is of gradual onset. The signs of onset of this gradual type are described, and the differential diagnosis is discussed. Again the employment of physical therapeutic measures is well discussed, x-ray therapy is mentioned, the value of home programs of physical therapy is stressed, and the need for orthopedic consultation is brought out.

This is a good general discussion of the whole subject of rheumatoid arthritis of the spine. The photography is good, the narration is satisfactory, and the only criticism of the filming is that some of the scenes are superfluous. By more judicious cutting, the film might be shortened by 5 or 10 minutes. Presumably, this picture was made for medical officers in the service who are treating these patients under conditions where time and expense is no consideration to the patient. It would be of interest to general practitioners, physical therapists, and nurses.

BUREAU OF LEGAL MEDICINE AND LEGISLATION

MEDICOLEGAL ABSTRACTS

Prenatal Injuries: Right to Recover Damages For.—The plaintiff, as administrator of the estate of Coleen Ann Drabbels, deceased, sued for damages for Coleen's death, allegedly due to the defendant's negligence. From a judgment for the defendant, the plaintiff appealed to the Supreme Court of Nebraska.

The plaintiff's petition alleged that the defendant, the Skelly Oil Company, is engaged in the selling of bottled gas under the trade name of Skelgas and that the defendant, Frey, is the agent of the Skelly Oil Company at Gordon, Neb., for the handling, sale, and delivery of Skelgas to the public. It is alleged that Virgil W. Drabbels purchased a container of Skelgas from the defendant, Frey, which, because of the defective condition of the container, exploded and caused the death of the decedent. The explosion occurred on July 2, 1948. The decedent, the unborn child of Audrey Drabbels, wife of Virgil W. Drabbels, was born dead on July 5, 1948. The petition also alleges that at the time of the explosion the pregnancy of Audrey Drabbels had advanced to a period of approximately eight months and that the unborn child was viable and capable of separate and independent existence. The defendant demurred to the plaintiff's petition on the ground that the decedent was dead when born, that the child was never an existing person, that there was never a person or estate for which the plaintiff could be appointed the administrator, and that the plaintiff had no legal capacity to sue.

The wrongful death statute of Nebraska, said the Supreme Court, is plain in stating that the right of action created by it exists only in cases wherein the injured person could himself have maintained an action for damages had he lived. It is clear, therefore, that the plaintiff can maintain no action for damages on account of the death of the child unless the child, had it lived, could have maintained an action against the defendants for the injury inflicted on it before its birth. The cases hold, the court continued, from a numerical standpoint at least, that damages for prenatal injury may not be recovered either by the injured child if it be born and lives or by its personal representative in the event of its death before birth from such injury. As was said in an Illinois case, "That a child before birth is, in fact, a part of the mother, and is only severed from her at birth, cannot, we think, be successfully disputed. The doctrine of the civil law and the ecclesiastical and admiralty courts, therefore, that an unborn child may be regarded as in esse for some purposes, when for its benefit, is a mere legal fiction, which, so far as we have been able to discover, has not been indulged in by the courts of common law to the extent of allowing an action by an infant for injuries occasioned before its birth. If the action can be maintained, it necessarily follows that an infant may maintain an action against its own mother for injuries occasioned by the negligence of the mother while pregnant with it. We are of opinion that the action will not lie." Following reference to that case and other cases, the Supreme Court of Nebraska held that a child born dead cannot maintain an action at common law for injuries received by it while in its mother's womb, and, consequently, the personal representative cannot maintain it under a wrongful death statute limiting such actions to those that would, if death had not ensued, have entitled the party injured to maintain an action and recover damages in respect thereof. An unborn child is a part of the mother until birth and, as such, has no juridical existence. Since no cause of action accrued to the child born dead, for injuries received before birth, the court concluded, none survived to the personal representative under the wrongful death statute. It would appear therefore, that an action of this character may not be maintained unless and until the right to bring it is afforded by legislative enactment. Accordingly, the judgment of the trial court in favor of the defendant was affirmed. *Drabbels v. Skelly Oil Co., 50 N. W. (2d) 229 (Nebraska 1951).*

MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A.M.A. Arch. Surgery, Chicago

64:535-732 (May) 1952

Meditations upon the Status Quo. E. R. Schmidt.—p. 537.
Acute Intestinal Obstruction in Infancy. C. W. McLaughlin Jr. and J. D. Coe.—p. 541.
Appendicitis During First Two Years of Life: Report on 21 Cases and Review of 447 Cases from Literature. W. H. Snyder Jr. and L. Chaffin.—p. 549.
*Acute Appendicitis in Infants: 15-Year Study. C. D. Benson, J. J. Coury Jr. and D. R. Hagge.—p. 561.
Pancreatic Calcifications. K. E. Lemmer and E. R. Schmidt.—p. 571.
Intestinal-Biliary Reflux After Anastomosis of Common Duct to Duodenum or Jejunum: Experimental Study. J. E. Musgrove, J. H. Grindlay and A. G. Karlson.—p. 579.
Acute Cholecystitis, Its Pathogenesis and Repair. C. G. Thomas Jr. and N. A. Womack.—p. 590.
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Arterial Blood Supply of Human Stomach. J. R. Brown and J. W. Derr.—p. 616.
*Clinical and Pathological Review of Nodular Thyroid. J. E. Kearns and H. Davis Jr.—p. 622.
Management of Head and Neck Cancer by General Surgeon. H. M. Morfit.—p. 631.
Application of Combined Hemimandibulectomy and Neck Dissection to Oral Carcinoma. W. W. Carroll.—p. 647.
Internal Fixation of Jaw Fractures: Further Report. F. McDowell and J. B. Brown.—p. 655.
Cardiovascular Responses to Acute Ligation of Portal Vein. M. E. Peck and R. F. Grover.—p. 665.
*Phlebectomy in Treatment of Acute Thrombosis of Saphenous Varices or Veins. L. G. Herrmann.—p. 681.
Small Lesions of Colon: Importance of Cooperation Between Surgeon and Roentgenologist in Diagnosis and Treatment. F. L. McMillan and R. M. Potter.—p. 686.
Carcinoma of Rectosigmoid and Upper Part of Rectum: Recurrence Following Low Anterior Resection. E. S. Judd Jr. and N. J. Bellegie.—p. 697.
Carcinoma of Colon: Study of End-Results of Surgical Treatment. H. K. Ransom.—p. 707.
Potassium Depletion in Surgical Patients. R. Patton, E. H. Ellison, E. T. Boles and R. M. Zollinger.—p. 726.

Acute Appendicitis in Infants.—Most infants with appendicitis are hospitalized after peritonitis has developed. This is due to (1) too liberal use of cathartics, (2) inability of the infant to localize his complaints, (3) inability of an underdeveloped omentum to assume a protective role, and (4) high position of the cecum, which tends to obscure the disease. Benson and associates review observations on 39 infants who were treated for acute appendicitis and its complications during the years 1936 to 1951 at two Detroit hospitals. The patients were 24 months of age or younger, but only three infants were under 12 months of age. More than two-thirds were boys. The symptoms had been present prior to hospitalization for from 6 hours to 14 days. The average rectal temperature in the infants with acute nonperforated appendicitis was 100.9 F; in infants with acute perforated appendicitis it was 102.8 F. An elevated leukocyte count is significant, but a low leukocyte count does not rule out appendicitis or its complications when the clinical picture is evident. Seven of the infants had acute appendicitis, 13 had appendicitis with perforation and peritonitis, and 19 were admitted with a perforated appendix and a mass in the right lower quadrant or pelvis. All six deaths occurred during the first 5 years of the 15 year period. Thus the mortality was reduced from 60% during the five year period 1936-1941 to zero during the 10 years 1941-1951. This is attributed to the fact that parents and physicians are more aware that appendicitis may occur in infants, and realize that surgical intervention is important. The availability and use of penicillin, streptomycin, and chloramphenicol (chloromycetin®) also have played an important role. The triad of abdominal pain, vomiting, and fever in infants must be considered to be due to appendicitis until proved otherwise.

The Nodular Thyroid.—The thyroids of 155 patients were examined because of the presence of a "single nodule." Studies were made also on 27 patients with cancer of the thyroid. Except for bizarre carcinomas and sarcomas, the histological picture was not related to survival. Gross blood vessel invasion as described by Graham is important in diagnosis of thyroid cancer, but not the microscopic picture often interpreted as such. The thyroid is abundantly supplied with blood vessels and tightly encapsulated. If there is hypertrophy, pressure within the capsule results in invagination of cells into the smaller vessels, a condition often wrongly interpreted as blood vessel invasion. The glands of 101 of the 155 patients who were operated on because clinically a single nodule was evident contained multiple nodules on examination. The incidence of cancer was not related to the incidence of the so-called "single nodule." Most of the patients with carcinoma were between 20 and 40 years of age; all three who were under 12 years of age had carcinoma. Seven of 14 male patients and 10 of 141 female patients had carcinoma. The presence of nodules necessitates surgical exploration in women over 18 whose glands show a rapid change in size in the absence of pregnancy or of hyperthyroidism, and in children and men immediately on discovery. Lobectomy is required for adequate study of a tumor and often is easier than simple excision of a nodule. In the 27 patients with cancer of the thyroid, 8 had been previously operated on for a nontoxic nodular goiter. This suggests that subtotal thyroidectomy does not protect against cancer. The authors show that diseased tissue can be completely excised with minimal deformity. They recommend total thyroidectomy rather than resection of only the affected lobe and isthmus, because there may be a tumor or a satellite in the opposite lobe. Radioactive iodine is of little value in the preoperative diagnosis, since a tumor may lie beneath normal thyroid tissue and thus show the same activity after a tracer dose of radioactive iodine as the normal thyroid. Radioactive iodine therapy in thyroid carcinoma is difficult to evaluate, because patients with untreated carcinoma of the thyroid can survive 20 to 35 years. In a few instances the use of ionizing radiation has produced excellent results.

Phlebectomy for Acute Thrombosis of Saphenous Veins.—Herrmann discusses a method of phlebectomy that has been employed for more than 15 years at the Cincinnati General Hospital as the definitive treatment for acute thrombosis of saphenous varices and veins. The author and his associates believe that the danger of this type of superficial venous thrombosis is in the spread of the thrombosis to the deep veins. Anticoagulants do not alter the thrombosis that has already taken place; if they are to be effective in limiting the spread of the thrombosis, they must be administered many weeks. The expense, as well as the difficulty of keeping the coagulability of the blood at the required level constitute problems. Complete excision of the vein and blood clots (phlebectomy) is a simple, safe, logical surgical procedure. When there is perivenous edema, the veins can be "shelled out" with minimum effort and least trauma to the surrounding tissues. If the operation is delayed until organization of the blood clots and cellular reaction about the veins becomes widespread, then complete extirpation of the veins and the blood clots becomes more difficult. The author believes that phlebectomy should be employed more

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Titles marked with an asterisk (*) are abstracted.

widely, for it reduces the length of disability to a few days, relieves pain promptly, reduces the total cost of the illness, and eliminates the serious complications that occasionally follow superficial venous thrombosis in the extremities.

American Journal of Medical Sciences, Philadelphia

223:469-588 (May) 1952

Analysis of Autopsy Records of 157 Cases of Carcinoma of Pancreas with Particular Reference to Incidence of Thromboembolism. C. M. Thompson and L. R. Rodgers.—p. 469.
Addison's Disease in the Negro. H. St. G. Tucker Jr., I. Taliaferro, R. H. Kirkland and R. Irby.—p. 479.
Rheumatic (Valvular) Heart Disease in Madison, Wisconsin: Survey of Seventh Grade School Children. R. W. Quinn and C. K. Kincaid. —p. 487.
*I^{131} Uptake in Treated Hyperthyroidism. F. K. Bauer.—p. 495.
Effects of Radioactive Colloidal Gold (AU^{198}) on Guinea Pigs. E. R. Fisher, J. C. Neering, J. B. Hazard and R. A. Hays.—p. 502.
Fluid, Electrolyte, and Protein Depletion Secondary to Bronchorrhea of Pulmonary Adenomatosis: Complication Heretofore Unreported. W. J. Levinsky and R. A. Kern.—p. 512.
Comparison of Precordial Electrocardiograms Using Indifferent Electrodes V, CR, and RS. E. Feldman, P. H. Wosika, E. S. Chesrow and G. A. Pipilis.—p. 522.
*Nitrogen and Potassium Metabolism: Reaction Pattern in Poliomyelitis. A. G. Bower, F. M. Morgan and A. L. Chaney.—p. 532.
Lymphopenia in Diagnosis of Acute Abdomen. A. E. Hirst Jr. and V. J. Johns Jr.—p. 548.
Current Uses of Cortisone and ACTH. T. Greiner.—p. 553.
Generalized Decreased Bone Density. D. M. Gould.—p. 569.

Radioactive Iodine Uptake in Treated Hyperthyroidism.—Doses of 1 or 2 μc of carrier-free I^{131} were administered intravenously or orally to 88 healthy persons. Uptake determinations were performed 30 minutes, 1 hour, 3 hours, 6 hours, 24 hours, and 48 hours after administration of the tracer dose. Results showed that the iodine could be administered orally as well as intravenously. Repeated I^{131} uptake readings were then made at the same intervals in 60 patients with hyperthyroidism or a previous history of hyperthyroidism. Previous observations by other investigators on the initial rapid rate of accumulation of I^{131} by the thyroid were confirmed. In all patients more than two-thirds of the total of I^{131} accumulated in 24 hours was present in the gland within 6 hours. The total amount of I^{131} accumulated in the glands of treated hyperthyroid patients in remission was less than in untreated patients, but the treated patients retained their rapid rate of uptake. The thyroids of several patients suspected of having hyperthyroidism accumulated a normal maximum amount of I^{131}, but with a rapid rate of uptake. These patients are being followed. The importance of obtaining repeated uptake readings within 24 hours after administration of the tracer dose of I^{131}, particularly at 3 and 6 hours, is stressed.

Nitrogen and Potassium Metabolism in Poliomyelitis.—Results of biochemical studies in 17 patients acutely ill with poliomyelitis with respiratory involvement indicated that in general the disease has an acute catabolic, a subacute catabolic, and an anabolic phase. Deviations from this general pattern were observed in a number of cases. The pronounced negative nitrogen balance that occurs in the acute phase of poliomyelitis may be utilized as a prognostic aid in predicting the ultimate degree of neuromuscular involvement. The necessary biochemical determinations are measurement of the nitrogen excretion in 24 hours or, less preferably, of the urinary nitrogen concentrations when urine volume is adequate. This negative balance is accompanied by a precipitous drop in serum albumin, elevation of the globulin fractions, and a negative potassium balance. The rapid drop in serum albumin is an indication for administration of serum albumin or irradiated plasma for prevention of peripheral vascular collapse. Hypopotassemia, which was frequently present and may be detected by serum determinations, may be prevented by administration of 3 gm. or more of potassium daily. Hyperpotassemia with shock and respiratory acidosis may be observed, in which case therapy should be directed toward restoration of the pulmonary ventilation, diffusion, and improvement of peripheral vascular tone while potassium is not given orally and parenterally. Excessive sodium intake should be avoided, particularly during the acute catabolic phase, when it may predispose to hypopotassemia and metabolic alkalosis. Average requirements at this time are approximately 3 gm. and decrease to 1 gm. during the subsequent anabolic phase. During the course of the disease, occurrence of complications such as extension of the disease process, pulmonary edema or atelectasis, respiratory acidosis, metabolic alkalosis, and hemolytic reactions with hepatic involvement may be detected early by recurrence of increased nitrogen and potassium excretion.

Am. J. Roentgenol. & Rad. Therapy, Springfield, Ill.

67:535-714 (April) 1952

Cranial Clues to Intracranial Abnormality. W. Penfield.—p. 535.
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Symmetrical Calcification in Cerebellum: Report of Two Cases. A. B. King and D. M. Gould.—p. 562.
Dilatation of Azygous Vein: Roentgen Sign of Venous Engorgement. F. G. Fleischner and S. W. Udis.—p. 569.
Roentgenologic Findings Accompanying Carcinoma of Pancreas. J. W. Beeler and B. R. Kirklin.—p. 576.
Pseudocyst of Pancreas Associated with Hydrothorax. R. J. Gross, R. Null and W. Loeb.—p. 585.
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Evaluation of Intracardiac Angiocardiography. G. C. Sutton, G. Wendel, H. G. Wedell and D. C. Sutton.—p. 596.
Focal Myocardial Calcification: Report of Case. W. M. Sennott and J. R. Mitchell.—p. 602.
Treatment of Carcinoma of Skin. G. S. Sharp and F. C. Binkley.—p. 606.
Mortality in Swine and Dose Distribution Studies in Phantoms Exposed to Supervoltage Roentgen Radiation. J. L. Tullis, F. W. Chambers Jr., J. E. Morgan and J. H. Zeller.—p. 620.
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Journal of Applied Physiology, Washington, D. C.

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Comparison of Blood Volume by Simultaneous Measurement of T-1824 in Arterial, Peripheral Venous and Mixed Venous Blood in Man. J. W. Nicholson III.—p. 771.
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*Jejunal Absorption of Amino Acid Mixture in Normal and in Hypoproteinemic Subjects. J. D. Hardy and J. Schultz.—p. 789.
Considerations Regarding Invert Sugar and Dextrose. D. V. Frost, J. P. Miller and R. K. Richards.—p. 793.
Effect of Acetate on Tm_{PAH} in Man. D. S. Baldwin and P. G. McLean. —p. 797.
Spectral Reflectance of White and Negro Skin Between 440 and 1000 Mμ. H. F. Kuppenheim and R. R. Heer Jr.—p. 800.
Observations on Dogs Exposed to Ambient Pressure of 30 mm. Hg. A. Edelmann and F. A. Hitchcock.—p. 807.
Spectrophotometric Determination of T-1824 in Whole Hemolyzed Blood. J. W. Nicholson III, G. G. Nahas and E. H. Wood.—p. 813.

Absorption of Amino Acid Mixture in Hypoproteinemia.—The jejunal absorption of an amino acid mixture (casein hydrolysate) was studied in six normal and five hypoproteinemic subjects, the aim being to determine whether or not there was any impairment in the jejunal absorption of an amino acid mixture in patients with hypoproteinemia as compared to that in subjects with normal serum protein levels. It was thought possible that impaired amino acid absorption might be a factor in the hypoproteinemia manifested by some patients who do not have obvious evidence of gross hepatic, renal, or intestinal disease. The normal subjects absorbed an average of 36.8% and the hypoproteinemic subjects an average of 27.9% of the nitrogen introduced. This difference in absorption is statistically significant. The reason for the decreased absorption of nitrogen from the casein hydrolysate solution by the hypoproteinemic subjects is not clear. Some degree of generalized edema was present in all except one of them, and in this patient the absorption of nitrogen was comparable to that of the controls. It is possible that in the edematous subjects edema of the intestinal mucosa was a factor in the decreased rate of absorption. Support for such an explanation is supplied by Elman, who found that patients with hypoproteinemia and resulting edema were unable to utilize orally administered protein hydrolysates (amigen®) normally.

Journal of Clinical Investigation, Cincinnati

31:341-432 (April) 1952

In Vitro Metabolism of Estrogens by Liver Studied by Means of Colorimetric Method. S. Lieberman, H. J. Tagnon and P. Schulman.—p. 341.
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Distribution of Thiosulfate Ion with Respect to Normal Human Serum and Red Blood Cells and Its Application to "Extracellular Fluid" Determination. H. J. Kowalski and D. D. Rutstein.—p. 370.
Amino Acid Studies and Clinical Findings in Normal Adults and Rheumatoid Arthritis Patients Treated with ACTH. A. L. Borden, E. C. Brodie, E. B. Wallraff and others.—p. 375.
Lung Volume and Its Subdivisions in Normal Boys 10-17 Years of Age. M. Morse, F. W. Schlutz and D. E. Cassels.—p. 380.
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Diurnal Variation in Diuretic Response to Ingested Water. S. Papper and J. D. Rosenbaum.—p. 401.
Potassium and Sodium of Red Blood Cells in Sickle Cell Anemia. D. C. Tosteson, E. Shea and R. C. Darling.—p. 406.
Studies of Total Body Water with Tritium. T. C. Prentice, W. Siri, N. I. Berlin and others.—p. 412.
Postoperative Potassium Deficit and Metabolic Alkalosis. Pathogenic Significance of Operative Trauma and of Potassium and Phosphorus Deprivation. L. P. Eliel, O. H. Pearson and F. C. White.—p. 419.

Journal of Nervous and Mental Disease, New York

115:283-376 (April) 1952

Comparative Twin Study on Genetic Aspects of Male Homosexuality. F. J. Kallmann.—p. 283.
Clinico-Experimental Investigation into Psychologic Aspects of Multiple Sclerosis. M. V. Baldwin.—p. 299.
Effect of Mephenesin on Psychotic Patients. W. E. Holt.—p. 343.
Intramedullary Spinal Cord Metastasis. M. B. Cantor and J. M. Stein. —p. 351.
Toxic Psychosis Following Nitrogen Mustard Therapy. B. Roswit and J. E. Pisetsky.—p. 356.

115:377-470 (May) 1952

*Organic Sequelae of Electric Convulsive Therapy Including Facial and Body Dysgnosias. A. Gallinek.—p. 377.
Psychiatric Aspects of Vagotomy: III. Problem of Diarrhea After Vagotomy. T. S. Szasz.—p. 394.
Amphetamine and Addiction. P. H. Knapp.—p. 406.
Patterns of Sensory Disturbance in Trunk and Face in Polyneuritis. N. Savitsky and E. Savitsky.—p. 433.
Effect of Rapid Intravenous Metrazol on Electroencephalogram. J. Smith, R. Anderson, M. M. Healy and M. Greenblatt.—p. 443.

Organic Sequelae of Electric Convulsive Therapy.—Gallinek is not concerned with the immediate postshock confusion, Korsakoff syndromes, memory defects, or the general reactions of blurring or blunting, characteristic of organic reactions, which are known to every shock therapist. The reactions described here are usually of a fleeting character and occurred in only a few patients. Three of the seven patients reported on had manic and paranoid syndromes. The manic syndrome in one became manifest two days after termination of the treatment, and the author gained the impression that neither memory impairment nor confusion is a factor in this psychosis. On the other hand, paranoid syndromes were observed only in association with considerable confusion. No relationship between the original psychosis for which the patients were treated and the induced psychosis could be found. These syndromes almost never lasted more than 10 days. Dysgnostic phenomena were observed in patients who were treated for depression, including one in whom depressive and schizophrenic features were coexistent. Nothing in the past histories of these patients seemed to predispose them to dysgnostic or pseudoillusional difficulties. The syndromes were observed in association with mild confusion and memory difficulties. The chief distortion of gnostic function was that faces of strangers gave the impression of being familiar, inducing one patient to address strangers. The second disturbance is the opposite of the first one. Familiar faces, like the face of husband, mother, or baby, may impress the patient as strange, the strangeness being indefinable, never interfering with recognition. The third disturbance is that faces of people are perceived as grotesque and caricature-like. Occasionally this impression provoked hilarious laughter. The phenomena usually do not arise before most of the original symptoms have disappeared, and they persist for a short time after complete disappearance of the underlying psychosis. They do not have the characteristics of a hallucination or an illusion, since their pathological nature is clearly recognized by the patients.

Journal of Urology, Baltimore

67:565-778 (May) 1952. Partial Index

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Polypoid Rhabdomyosarcoma (Sarcoma Botryoides) of Bladder in Children. F. K. Mostofi and W. H. Morse.—p. 681.
Primary Sarcoma of Bladder in Children: Report of Two Cases. R. A. Way.—p. 688.
Bold Excision of Strictures of Urethra with Small Resectoscope. E. E. Angle and LaV. F. Pfeifer.—p. 695.
New Surgical Procedure for Cure of Scrotal Hypospadias: Grafting of Male Human Urethra Taken from Fresh Cadaver. J. P. Bourque. —p. 698.
Carcinoma of Penis. J. E. Colon.—p. 702.

Aluminum Gels for Control of Renal Calculi.—Shorr's regimen for control of phosphatic renal calculi was used in 37 patients who had a tendency to formation of this type of calculi, and in whom the tissue damage, obstruction, and infection present in the urinary tract should have enhanced the rate of stone growth. Twelve patients had had unilateral nephrectomy (11 for calculi and one for tumor), so that 62 kidneys were studied. When the regimen was begun, 35 kidneys in 26 of these patients contained calculi, and in 11 patients the kidneys were stone-free. Seventeen kidneys were hydronephrotic. All but eight patients had pyuria. The aluminum hydroxide gels administered for prophylaxis were amphojel,® usually in doses of 40 cc., or basaljel,® in doses of 30 cc., after meals and at bedtime. For individual patients the dosages were varied slightly. The patients followed a diet supplying approximately 1,300 mg. of phosphorus and 700 mg. of calcium per day. Each was urged to maintain a daily fluid intake of approximately 3,000 cc. Antimicrobial drugs and urologic measures were employed according to the usual indications. Comparison of the clinical courses of these patients before institution of the regimen with their courses during the prophylactic program showed that the aluminum gel and dietary therapy reduced stone formation. For instance, 78 operations for removal of calculi had been carried out before the prophylactic regimen was begun, but during the program only four operations were required, for removal of stones that had been present before the start of the regimen or had developed following lapse in therapy. Five patients provided individual control studies by voluntarily discontinuing therapy, and in three of these calculi promptly recurred. This study includes several of the cases reported by Shorr and Carter, and substantiates their clinical findings. The experimental work of Vermeulen and his associates and the clinical observations of Satterthwaite corroborate this study.

Laryngoscope, St. Louis

62:333-448 (April) 1952

Further Observations Concerning *Toxic Effects* of *Streptomycin* and Quinine on Auditory Organ of Guinea Pig. L. Ruedi, W. Furrer, F. Luthy and others.—p. 333.
Resuscitation of Heart in Practice of Otolaryngology. M. Lineback.—p. 352.
Cardioesophageal Junction—Histologic Study. G. D. Strauss and J. M. Lubitz.—p. 359.
Diagnosis and Treatment of Acute Subdural Abscess Complicating Suppurative Frontal Sinusitis. W. R. Pitts.—p. 367.
*Present Status of Globus Hystericus. R. G. Rigby.—p. 401.
Bacteriologic Aid in Rhinologic Diagnosis and Treatmnt: Improved Criteria. J. L. Goldman.—p. 406.
Bleeding Following Tonsillectomy. S. L. Fox.—p. 414.
Treatment of Hyperplastic Sinusitis. L. E. Silcox.—p. 426.

Globus Hystericus.—Rigby believes that so-called "globus hystericus" is an important disease and that too little time has been spent in its diagnosis and treatment. He discusses the difficulty in differentiating functional disturbances and organic disease at the level of the upper esophagus and reports his experiences with 98 patients referred to him because of a "lump in the throat." These patients should be put at ease, and, in taking the history, attention should be given to emotional disturbances. For most of the patients a diagnosis of a mild type of hysteria had been made. The others were found to have organic disease, which was treated. X-ray examinations of the neck revealed two characteristic phenomena: (1) marked hypertrophy of the lingual tonsil, which is seen by indirect examination of the pharynx, and is confirmed by lateral x-ray films of the neck, and (2) marked lipping or osteoarthropathy of the anterior borders of the cervical vertebrae. Marked lipping of the cervical·vertebrae with the so-called inflammatory reaction in the soft tissues anterior to the lipping may set off the trigger mechanism that produces the globus hystericus. To the author this seems as logical as such explanations as hyperirritability of the mucous membrane, spasm of the cricopharyngeus muscle, or irritability caused by enlarged lymph nodes. He found psychotherapy effective in treating these patients. He concludes that "globus hystericus" or a "lump in the throat" can be a symptom of functional disorder or of organic disease. The diagnosis and treatment is based on a complete history and physical examination.

Maryland State Medical Journal, Baltimore

1:175-212 (April) 1952

Committee for the Study of Pelvic Cancer. B. C. Compton.—p. 176.
No Longer Our Ivory Towers. N. E. Day.—p. 180.
The Physician Against Atomic Attack. W. L. Wilson.—p. 182.
Cortisone (Cortone) in Treatment of Acute Subdeltoid Bursitis. M. Friedman.—p. 186.
Thirty Years of Phytopharmacology or Applications of Plant Physiology to Medical Problems. D. I. Macht.—p. 188.

Neurology, Minneapolis

2:81-176 (March-April) 1952

Sensory Return in Partial and Recovery Spinal Cord Lesions. B. Boshes, M. Brown and R. L. Crouch.—p. 81.
Neuromyelitis Optica with Xanthochromia and Spinal Block. H. D. McIntyre and A. P. McIntyre.—p. 96.
Perioral Reflexes. K. A. Ekbom, B. Jernelius and E. Kugelberg.—p. 103.
Behavior Deficits Following Diencephalic Lesions. J. S. Meyer and J. Hunter.—p. 112.
*Relief of Pain in Trigeminal Neuralgia by Crystalline Vitamin B_{12}. W. S. Fields and H. E. Hoff.—p. 131.
On Certain Aspects of Sensory Organization of Human Brain: III. Patterned Response to Three Simultaneously Applied Cutaneous Stimuli. R. Cohn.—p. 140.
Psychologic Tests in Diagnosis of Organic Brain Disease. E. D. Hoedemaker and M. E. M. Murray.—p. 144.
Action of Adrenaline and Noradrenaline on Tremor in Parkinson's Disease. H. Barcroft, E. Peterson and R. S. Schwab.—p. 154.

Vitamin B_{12} to Relieve Pain in Trigeminal Neuralgia.—Relief of neuropathy in pernicious anemia by administration of vitamin B_{12} suggested its employment in other nervous disorders. It relieved the pain of alcoholic, diabetic, and other peripheral neuritides, but in early tests it failed to alleviate the pain of trigeminal neuralgia. Nevertheless when solutions of greater potency became available, therapy was undertaken in 13 cases of trigeminal neuralgia begining in May, 1950. The first six patients were given 1,000 µg doses biweekly or triweekly for four to eight weeks, and the pain was markedly relieved within the first week, usually after the second injection. In four patients the sharp lancinating type of pain was replaced by nonradiating burning paresthesia in the same area. This paresthesia disappeared with continuance of therapy, although it was more resistant to treatment. In two cases, when therapy was interrupted for about one week while the burning sensation was still present, the lancinating pain returned but was relieved after the first of six daily injections of 1,000 µg each. Because symptoms returned when therapy was interrupted and burning sensations persisted, daily injections for 10 days were given to the last seven patients. At the time of this report there have been no recurrences in this series. None of these patients has requested other therapy. Five of the patients had received other therapy in the past, such as nerve section or alcohol block. Relapses after these treatments made these patients reluctant to undergo further surgical intervention. Four of these proved to be the most resistant patients in the series, but the ultimate degree of relief was the same as in others. After a year of experience with massive doses of vitamin B_{12} it is felt that this substance offers promise of prompt relief of the most distressing aspects of the pain, with ultimate complete or virtually complete relief of the secondary burning paresthesia.

New England Journal of Medicine, Boston

246:677-720 (May 1) 1952

"Hysteria" in Men: Study of 38 Patients so Diagnosed and 194 Control Subjects. E. Robin, J. J. Purtell and M. E. Cohen.—p. 677.
Conduction of Visceral Pain. J. C. White.—p. 686.
*Chronic Ulcerative Colitis and Pregnancy. M. Patterson and E. J. Eytinge.—p. 691.
Rupture of Uterus During Pitocin Infusion. J. H. Ferguson, G. T. Schneider and H. K. Miller.—p. 694.
Significance of Cholesterol in Pathogenesis of Vascular Lesions: Discussion of Intracellular Accumulation of Cholesterol in Intima and of Extracellular Precipitation of Cholesterol in Arterial Tissue. S. J. Thannhauser.—p. 695.
Acute Intestinal Obstruction Caused by Macaroni: Report of Case. D. Pecora, E. Pepe and P. Cooper.—p. 702.

Chronic Ulcerative Colitis and Pregnancy.—Patterson and Eytinge reviewed the cases of 16 women in whom ulcerative colitis and pregnancy concurred. The diagnosis of chronic ulcerative colitis was made by proctoscopy and barium enema examination of the colon. Eight of the 16 patients became pregnant while the colitis was moderately active. These eight patients had 18 pregnancies, with 16 viable children (1 pair of twins), 1 fetal death that seemed unrelated to the ulcerative colitis, 1 therapeutic abortion, and 1 spontaneous abortion. Four patients thought that pregnancy improved the colitis, three thought that the colitis was aggravated during and after pregnancy, and one said that pregnancy had no effect. Three patients who became pregnant at a time when their colitis was inactive had four pregnancies with three viable children, and one therapeutic abortion. Two of these three patients thought that pregnancy had no effect on their condition and one believed that it aggravated her symptoms. Five patients in whom ulcerative colitis developed during pregnancy were acutely ill. In three cases viable infants were delivered. In one patient the process became quiescent, and she has since had two children and is now pregnant for a third time. One patient died shortly after a miscarriage. One patient had two pregnancies but no viable children. In the other two patients the disease continued to be active; one required ileostomy two months after delivery. Pregnancy that occurs during an inactive stage of ulcerative colitis is usually well tolerated. Pregnancy in a patient with mild or moderately active symptoms may produce improvement or aggravation. If possible, pregnancy should be delayed until the colitis is quiescent, but regardless of symptoms the pregnancy can usually be continued to term with birth of viable children. Ulcerative colitis that develops during pregnancy is virulent and may be fatal.

Psychiatric Quarterly, Utica, N. Y.

26:177-352 (April) 1952. Partial Index

Contribution to Study of Phenomenon of Resistance. L. Eidelberg —p. 177.

*Clinical Evaluation of Use of Decamethonium Bromide (C_{10}) in Convulsive Shock Therapy. M. Brody.—p. 205.

Incidence of Mental Diseases as Measured by Census Investigations Versus Admission Statistics. O. Oedegaard.—p. 212.

Participant Teaching of Psychotherapy by Senior Physicians: Hospital Program and Clinical Illustrations. L. B. Hill and F. G. Worden. —p. 228.

Some Values of Use of Multiple Therapists in Treatment of Psychoses. M. L. Hayward, J. J. Peters and J. E. Taylor.—p. 244.

Psychodynamic Study of a Patient During Experimental Self-Regulated Re-Addiction to Morphine. A. Wikler.—p. 270.

Decamethonium Bromide in Convulsive Shock Therapy.—Drugs that produce muscular relaxation, prevent fractures, minimize muscular soreness, and reduce the strain on the heart and lungs are of obvious advantage to the patient undergoing therapeutic convulsions. This report is concerned with the clinical evaluation of decamethonium bromide as a muscle relaxant and is based on the study of 117 consecutive patients who required a total of 1,302 electric shock treatments, of which 869 were modified by decamethonium bromide, 326 by *d*-tubocurarine, and 107 by dimethyl ether of *d*-tubocurarine chloride. Brody concludes that curare and decamethonium are dissimilar drugs, although both produce muscular relaxation. The weakness produced by curare simulates myasthenia gravis, while the action of decamethonium simulates the paralysis of botulism. Because of their dissimilarity, there will be situations in which one is preferred to the other. In shock therapy, the advantages of decamethonium bromide seem to outweigh the disadvantages. The chief advantages are the shorter action of the drug and the relative sparing of the tongue and larynx; the disadvantages are the lack of antidote and the inability to predict the first dose. These disadvantages can be handled by the method of fractional administration of decamethonium bromide. All drugs mentioned are satisfactory relaxing agents, but Brody prefers decamethonium in shock therapy.

Stanford Medical Bulletin, San Francisco

10:65-134 (May) 1952. Partial Index

Hypertension and Relation Between Kidney Weight and Body Weight. E. Braun-Menendez.—p. 65.

Electroencephalogram in Functional Psychiatric Disorders. H. W. Newman and R. Lawrence.—p. 76.

Bleeding in Late Pregnancy and Puerperium. R. W. Noyes.—p. 78.

*Bedside Method for Semiquantitative Sodium Analysis. G. D. Talbott and W. King.—p. 82.

*Parenteral Use of Sodium Para-Amino-Salicylic Acid in Critical Tuberculosis: Preliminary Report. E. G. Roberts and C. Brown.—p. 87.

New Staining Technique for Ringworm Diagnosis: Preliminary Report. R. C. Lofgren and E. E. Batts.—p. 96.

Mucous Cysts Causing Vesical Neck Obstruction. H. M. Weyrauch, C. S. Harrod and P. O. Mustacchi.—p. 99.

Diagnostic Value of Biopsy of Nonpalpable Retroclavicular Paratracheal Lymph Nodes. L. S. Baer.—p. 104.

Anticonvulsant Action of Methylparafynol (Dormison®). R. W. Schaffarzick and B. J. Brown.—p. 106.

Bedside Method for Sodium Analysis.—Most methods for measurement of sodium in urine or other excretory fluids have been indirect and not entirely satisfactory. The method described requires only one reagent, a uranyl zinc acetate solution. The simple procedure is a new application of an established method for chemical analysis of sodium, Kolthoff having recognized in 1927 that uranyl zinc acetate would react with sodium to produce an insoluble salt. The sodium is thus precipitated, and the amount of precipitate is read as 1+ to 5+, each degree representing a definite range of sodium ion concentration expressed in milliequivalents per liter. The preparation of the reagent solution and the equipment required are described. The test procedure consists of placing one drop of clear (if necessary, filtered) fluid to be tested into a test tube, adding eight drops of reagent, shaking gently, and reading in eight minutes. The value of this determination lies in its simplicity. It may be carried out in the wards, on home visits, or be used in the office. It permits detection of the low salt syndrome or sodium retention in cardiac failure. It is also helpful in checking adherence to a low salt diet.

Parenteral Use of Sodium Para-Aminosalicylic Acid.—Several European, particularly Swiss, investigators have administered *p*-aminosalicylic acid solution by continuous intravenous drip to patients with tuberculous meningitis and miliary tuberculosis. This method of treatment is now being tried in this country. The material used is lyophilized sterile sodium *p*-aminosalicylic acid, reconstituted with distilled water to make a 3% solution. Heparin, 5 mg. per 1,000 cc., is added to the intravenous solution. Ordinarily 15 gm. of *p*-aminosalicylic acid are given over a period of four hours, and this dosage is repeated after a rest of two or three hours. Most patients tolerate a daily dose of 30 gm. Blood levels of *p*-aminosalicylic acid are generally 20 to 30 mg. per 100 cc. within an hour or two of administration. Spinal fluid levels are 11 to 15 mg. per 100 cc. within two to three hours after the start of treatment. Parenteral *p*-aminosalicylic acid has been used in tuberculous meningitis, miliary tuberculosis, as an adjuvant before and during surgery, especially in patients whose bacilli are streptomycin-resistant, and in patients who do not respond to standard therapy. The authors discuss the histories of 3 of 14 patients to whom *p*-aminosalicylic acid was given by the parenteral method. Six of the 14 had tuberculous meningitis, 4 had miliary tuberculosis, 1 had a combination of the two, and 3 had not responded to standard treatment. Two of the patients with tuberculous meningitis died. Four are greatly improved, three having been discharged to their homes. The authors believe that the treatment was begun too late in the patients who did not respond.

Western J. Surg., Obst. & Gynecology, Portland, Ore.

60:193-240 (May) 1952

Use of ACTH and Cortisone in Homografting: Effect of Preoperative Administration of ACTH to Donor and Recipient on Survival of Skin Homografts in Humans. R. C. Bishop, J. M. Beal and W. P. Longmire Jr.—p. 193.

Problem of Splenectomy in Diseases of Spleen. S. P. Lucia.—p. 199.

Intestinal Antisepsis: Present Status in Surgical Conditions. E. J. Poth. —p. 205.

*Tumors of Brain Complicating Pregnancy. D. L. Reeves.—p. 211.

Solitary Cecal Diverticulitis. R. E. Ahlquist and M. F. Kepl.—p. 219.

SYMPOSIUM: CERTAIN ASPECTS OF OFFICE TREATMENT IN OBSTETRICS AND GYNECOLOGY

Trichomonas Vaginitis Vaginalis. J. R. Upton.—p. 222.

Monilia Vaginitis. E. F. Anderson.—p. 224.

Senile Vaginitis. J. N. Ewer.—p. 226.

Sterility. E. W. Overstreet.—p. 230.

Brain Tumors Complicating Pregnancy.—Reeves reviews six cases in which brain tumor complicated pregnancy and shows that each of these cases presented a different problem and required individual study and treatment. When intracranial pressure increases, operative intervention cannot be postponed. Furthermore, because of the increased intracranial pressure associated with spontaneous delivery, cesarean section is safer than spontaneous delivery in women with brain tumor. If an electroencephalographic tracing of a pregnant woman indicates the presence of an expanding intracranial lesion, additional diagnostic procedures and treatment should be considered. While any surgical intervention in a pregnant woman increases the chance of abortion, the first case here presented indicates that even convulsions are tolerated without resultant abortion. That the neurosurgeon is faced with complex and difficult problems if brain tumor occurs during pregnancy becomes evident from cases reported in the previous literature as well as from the six here described.

Wisconsin Medical Journal, Madison

51:441-536 (May) 1952

The Challenge of General Practice. P. T. Bland.—p. 468.

Use of Progesterone and Vitamin K in Treatment of Rh Sensitized Pregnant Women: Preliminary Report. L. J. Paquette and J. T. Schmitz. —p. 473.

Early Diagnosis and Effective Treatment of Depressive Reactions. L. Danziger.—p. 475.

Male Infertility. J. W. Sargent.—p. 477.

Respiratory Symptoms Due to Esophageal Foreign Bodies in Infants. A. C. Tompsett Jr.—p. 481.

BOOK REVIEWS

Posture and Pain. By Henry O. Kendall, Director, Physical Therapy Department, Florence P. Kendall, Assistant Director, Physical Therapy Department, and Dorothy A. Boynton, Physical Therapist, Physical Therapy Department, Children's Hospital School, Baltimore. Cloth. $7. Pp. 204, with 159 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1952.

The relation of faulty posture to the production of pain, as presented in this book, is based on the authors' many years of experience as physical therapists in the physical therapy department at Children's Hospital School in Baltimore.

The book deserves special commendation for its attractive format and excellent illustrations. The first chapter considers standard posture, which, according to the text, refers to an "ideal" posture rather than an average posture. It is further indicated in the text that "the authors have not seen an individual who matches the standard in all respects." The second chapter, which is about postural faults, considers deviations from this "standard." It seems peculiar that deviations from a "standard" that has not been observed to exist should be considered as postural faults. It would seem more proper that postural faults be considered as deviations from an acceptable average posture.

The third, fourth, and fifth chapters contain a simple yet practical analysis of muscle action and imbalance as related to faulty posture. The importance of muscle strength and tightness or loss of extensibility as a contributing factor to poor posture is discussed. A systematic and practical procedure for posture analysis is discussed in the sixth chapter. In the section of the text on the treatment of faulty posture and pain associated with faulty body mechanics this statement is most properly included: "In discussing pain in relation to postural faults, there are those, however, who are dubious. They often ask the very pertinent question why it is that many cases of faulty posture exist without symptoms of pain. They also question why seemingly mild postural defects give rise to symptoms of mechanical and muscular strain." There follows a logical discussion of this controversial subject. The opinions expressed in the text are worthy of consideration, even though some physicians may not agree. The physician may find many of the suggestions regarding the use of various physical therapeutic agents useful in prescribing physical therapy and supports for the treatment or prevention of faulty posture and the treatment of painful conditions of the neck, back, and extremities.

Infant Development: The Embryology of Early Human Behavior. By Arnold Gesell, M.D. Cloth. $3.50. Pp. 108, with illustrations. Harper & Brothers, 49 E. 33rd St., New York 16, 1952.

This book is beneficial to pediatricians and others confronted with the evaluation of physical and mental development of infants and children. This volume presents an informal orientative introduction to growth and forms of behavior at advancing maturity levels from birth to the 10th year. It attempts to show that there is a significant continuity of functional and physical development in both the prenatal and postnatal phases of human growth. A study of premature infants throws light on growth changes that ordinarily take place in the prenatal period. There is a discussion of comparative anatomy in lower animals emphasizing the embryonal development of the eyes and eye and hand behavior. A pictorial chapter has 50 photographs from their feature length film "The Embryology of Human Behavior" to demonstrate the progression of the growth cycle. These excellent action photographs clearly illustrate developmental patterns at various age levels. A chapter on examination methods and diagnostic procedures helps one to evaluate both the normal and abnormal child. An appendix includes sketches and charts of growth patterns of the 28-week-old infant, a bibliography, and a list of silent and sound movies by the Yale Films of Child Development.

Brain Tumors of Childhood. By Henry M. Cuneo, M.D., Assistant Clinical Professor of Neurological Surgery, University of Southern California, School of Medicine, Los Angeles, and Carl W. Rand, M.D., Clinical Professor of Neurological Surgery, University of Southern California, School of Medicine. Publication number 104, American Lecture Series, monograph in Americal Lectures in Surgery, edited by Michael E. De Bakey, M.D., Professor of Surgery and Chairman of Department of Surgery, Baylor University College of Medicine, Houston, and R. Glen Spurling, M.D., Clinical Professor of Surgery, University of Louisville, Louisville, Ky. Neurosurgical Division, editor: Barnes Woodhall, M.D., Professor of Neurosurgery, Duke Hospital, Durham. Cloth. $5.75. Pp. 224, with 61 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto 2B, 1952.

This compact volume is in the American lecture series. In it, the authors summarize their experiences with brain tumors in children at the Children's Hospital in Los Angeles during the past decade. Eighty-three verified cases of brain tumor and/or granulomatous lesions resembling tumors are discussed.

Each of the first 12 chapters is devoted to one pathological type of tumor. In the final chapter, the authors discuss the general symptomatology and differential diagnosis of these lesions as they occur in childhood. A brief quotation from some authority in this field at the beginning of each chapter sets the stage for the discussion that follows. A few case reports are included; these are brief and to the point, with excellent illustrations. Selected references, mostly in English, are given at the end of each chapter.

The authors' experiences with cerebellar astrocytoma and medulloblastoma emphasize the importance of biopsy and histological verification of the tumor type before administration of x-ray therapy. The failure of ventricular estimation to differentiate between cerebellar and third ventricle tumors is properly emphasized and leads the authors to advise routine ventriculography for children who have symptoms of posterior fossa tumors.

One wonders at the inclusion of cases of spongioblastoma polare in the chapter on glioblastoma multiforme rather than in the chapter on astrocytoma, a tumor with which it is more closely related. This minor pathological point is compensated for by the inclusion of discussion of the granulomatous lesions, not ordinarily considered under the heading of brain tumors in childhood.

The similarity of their results with those reported in the monograph by Bailey, Buchanan, and Bucy in 1939, emphasizes the enormity of the therapeutic problems. This review will be especially valuable to pediatricians and to neurologists and neurosurgeons who do not see many patients in this age group; however, it can be read with profit by all physicians who are interested in neoplastic diseases of the brain.

A Review of Nursing with Outlines of Subjects: Questions and Answers. By Helen F. Hansen, R.N., M.A., Inspector, Schools of Nursing, State Department of Public Health, California, Sacramento. Seventh edition. Cloth. $5.75. Pp. 844. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; 7 Grape St., Shaftesbury Ave., London, W.C.2, 1952.

The author of this review of nursing has extensive experience in educational administration. Her positions as chief at the bureau of registration of nurses in California and executive secretary of the board of nurse examiners in California were excellent background for the preparation of this book.

The material is presented in outline form, and each section is followed by a battery of tests on the preceding subject. Correct answers to these tests are found at the back of the book. In this edition, the author has attempted to include more comprehensive situation type questions to stimulate additional reading. True-false and completion type questions have been reduced in number.

The fields covered are chemistry, anatomy, and physiology, psychology, nutrition, public health, pharmacology, and thera-

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The reviews here published have been prepared by competent authorities and do not represent the opinions of any official bodies unless specifically stated.

peutics, and microbiology. In patient care, the areas included are medical care, communicable disease, and surgical, obstetric, pediatric, and psychiatric nursing. Also reviewed are nursing history, professional relationships, and sociology.

In this new edition, the chapter on pharmacology and therapeutics has been revised. Public health aspects have been enlarged to include international health problems and the World Health Conference. Also added is material on the role of poisons in industrial health, the hygiene and nursing care of patients receiving x-ray therapy, and the anatomy and physiology of the male reproductive system. There are no illustrations, plates, or bibliographies.

This book should serve a useful purpose for those who wish an outline to guide their review of the field of nursing.

Textbook and Guide to The Standard Nomenclature of Diseases and Operations. By Edward T. Thompson, M.D., F.A.C.H.A., Senior Surgeon, Public Health Service, and Adaline C. Hayden, R.R.L. Cloth. $8. Pp. 619. Physicians' Record Company, 161 W. Harrison St., Chicago 5, 1952.

This is a new book designed, as its title implies, to serve as a guide to the use of the recently published fourth edition of the "Standard Nomenclature of Diseases and Operations." In it, the authors have succeeded in answering practically all questions that may arise in the use of the nomenclature, either for physicians or medical record librarians. It will serve an invaluable function as a textbook in the training of medical record librarians and as a reference book in any institution using the "Standard Nomenclature." Probably few physicians will read this book from cover to cover, but medical record librarians can use it with great effectiveness in discussing certain problems of classification with the professional staff members of their institutions by pointing out appropriate paragraphs.

There are few criticisms; however, it is perhaps unnecessary to discuss eponyms in two places (pages 189 and 213). The book contains a list of disease entities that are not classified but does not state what to do with them when such diagnoses arise. The authors are to be complimented on the preparation of this difficult and useful book, which is presented with good type and in attractive form.

Manual for Medical Records Librarians. By Edna K. Huffman, R.R.L., Medical Records Consultant. Third edition. Cloth. $7.50. Pp. 484, with 125 illustrations. Physicians' Record Co., Publishers, 161 W. Harrison St., Chicago 5, 1952.

This book was planned as a day to day guide for problems arising in medical record libraries. Most of the previous edition has been completely rewritten to bring material up-to-date. The chapter on organization and management now contains job descriptions for the various tasks assumed by medical record library personnel. One chapter has been devoted to the medical audit. This subject is timely and has attracted much attention in the last decade. Some of the old illustrations have been completely omitted and/or replaced by new ones.

The material is discussed in a clear, concise style, and, after a careful review of the volume, there can be no doubt in the mind of the medical record librarian as to what procedure she should follow in maintaining an efficient medical record department. The book is printed in clear type on good paper and is attractively bound. It is wholeheartedly recommended to all hospital personnel who deal, even to the slightest degree, with medical records.

Health Instruction Yearbook 1951. Compiled by Oliver E. Byrd, Ed.D., M.D., F.A.P.H.A., Professor of Health Education, and Director, Department of Hygiene, School of Education, Stanford University, San Francisco. Foreword by Bernice Moss, M.A., Ed.D., President, American Association for Health, Physical Education, and Recreation, Salt Lake City. Cloth. $3.50. Pp. 236. Stanford University Press, Stanford University, California; Oxford University Press, Amen House, Warwick Sq., London, E.C.4, 1951.

This is another book in the series that was begun eight years ago, making this the ninth "Yearbook." Previous editions have been reviewed in THE JOURNAL, and there is no new comment to be added with regard to this edition. It is a useful reference book for the desk of anyone engaged in public health or health education.

Experiments in Biochemistry. By Max S. Dunn, Ph.D., Professor of Chemistry, University of California, Los Angeles, and William Drell, Ph.D., Research Fellow, California Institute of Technology, Pasadena. Cloth. $5. Pp. 197, with 12 illustrations and 46 data sheets for experiments. McGraw-Hill Book Company, Inc., 330 W. 42nd St., New York 18; Aldwych House, Aldwych, London, W.C.2, 1951.

This book, designed as both a reference work and laboratory guide, accomplishes to a large extent the purpose for which it was written. It contains 46 experiments, which have been chosen to cover as wide a field as possible and at the same time to serve as an introduction to the use of the various laboratory techniques that are employed in investigative biochemistry. The experiments include syntheses, analyses, isolations, and purification and/or identification of proteins, amino acids, fats, carbohydrates, purines, pyrimidines, vitamins, and enzymes. The analytical experiments are chosen to include animal, microbiological, and instrumental as well as chemical procedures.

Each chapter is prefaced with a brief discussion of the subject of the experiment. These discussions, although brief, are made broad by including numerous references to original literature, reviews, and books. These references are included as a bibliography at the end of the book. Many of the experiments contain elaborate explanatory notes. The volume also contains an appendix, which gives details with respect to apparatus employed, reagents used, and special techniques required in various experiments. First aid hints, instructions to students, and suggestions to instructors are also contained in this section. Data sheets, included at the end of the book, can be conveniently filled out by the student and cut from the book. An adequate index is also included.

Case Record from a Sonnetorium: Poems. By Merrill Moore. Cartoons by Edward St. John Gorey. Cloth. $1.50. No pagination. Twayne Publishers, 42 Broadway, New York 4, 1951.

Dr. Merrill Moore is said to have written more sonnets than any other poet. His sonnets are original in form to the extent of being referred to by the poet himself as illegitimate. They are not necessarily medical. In fact, one may find on opposing pages sonnets referring, respectively, to eels and to love. Along with these sonnets goes a running series of cartoons. The cartoon has no necessary relationship to the sonnet with which it shares the page, but the cartoons themselves are supposed to constitute a series, which is, to say the least, highly fanciful. Physicians who like poetry will find this an amusing volume if they choose to read it in that vein, or if they wish to think more deeply they can find material here for that.

Eleventh Semiannual Report of the Atomic Energy Commission. United States Atomic Energy Commission. Paper. Pp. 211, with illustrations. Govern. Print. Off., Washington 25, D. C., 1952.

The three main divisions of this book are (1) a summary of the major activities in atomic energy programs from July to December, 1951, (2) a condensed Atomic Energy Commission annual financial report for the fiscal year 1951, and (3) a review of the applications of atomic energy to plant science. The last 76 pages consist of appendixes giving concrete details to document the preceding material.

Every page of the book is of interest to the physician either as a citizen concerned about the multifarious activities of the government or as a scientifically trained person watching for possible applications of atomic energy in the healing arts. It is therefore hard to single out passages of particular importance, except perhaps the section (pages 34 to 42) on biology and medicine, which describes the almost inconceivably extensive plans for laboratory facilities for the biologic and medical research program. It is stated that the total value of such facilities completed or approved for construction will approximate 30 million dollars. The status of the Argonne Biological Laboratory, Argonne Cancer Hospital, Brookhaven Biological Laboratory, Hanford Aquatic Laboratory, and Los Alamos Biomedical Laboratory is briefly summarized. Among the projects dealing with protection from injury by radiation is one (page 36) on chelating agents, chemicals that can be taken internally to hasten the excretion of radioactive elements that have become fixed in the animal body. The book is recommended to everyone interested in the possible relations of radioactivity to disease in man.

QUERIES AND MINOR NOTES

POISONOUS GARDEN PLANTS

To the Editor:—*I would like reliable information about hazardous or toxic plants commonly found in gardens. What are the toxic constituents, and what antidotes should be used for poisoning by them? I have had inquiries concerning the danger for children of eating oleander berries.*

Robert N. Philips, M.D., Chico, Calif.

Answer.—Only a few garden plants are poisonous by contact, but many others are poisonous if eaten, especially those in the family Solanaceae and the rhododendrons, mountain laurel, Christmas rose, and monkshood. A complete list of poisonous garden plants will be found in Taylor's "Encyclopedia of Gardening" (ed. 2, New York, Houghton Mifflin Company, 1948) in the section on poisonous plants, in which the part of each plant that is poisonous is specified.

The seeds and juice of oleander (Nerium oleander) are dangerously poisonous, as are many other plants in the family Apocynaceae. The poison in oleander is the glycoside oleandrin. Many other garden plants contain dangerously poisonous alkaloids; the best references for these are Henry's "Plant Alkaloids" (ed. 3, Philadelphia, The Blakiston Company, 1939) and the "Dispensatory of the United States of America" (Osol, A., and Farrar, G. E., Jr., editors, ed. 24, Philadelphia, J. B. Lippincott Company, 1947). Patients should be urged to keep part of the suspected plant for identification, as this may be the only clue to the alkaloid or glycoside causing the poisoning. Treatment should be based on this identification. Reference should be made to Sollman's "Manual of Pharmacology and Its Applications to Therapeutics and Toxicology" (ed. 7, Philadelphia, W. B. Saunders Company, 1948) and, especially, to "The Pharmacological Basis of Therapeutics" (Goodman, L., and Gilman, A.: New York, The Macmillan Company, 1941).

HYPERTENSION IN HIGH SCHOOL BOYS

To the Editor:—*In routine physical examinations, a number of high school boys with a systolic blood pressure higher than 150 mm. Hg and a pulse rate faster than 110 per minute were found. At what level of blood pressure is it safe to accept a high school boy for full athletic activity? What are the proper pulse rate ranges for acceptance and rejection of these boys? I maintain that it is not wise to accept a boy with a relatively high blood pressure or pulse rate, even if no organic disease is demonstrated, for such a boy is potentially hypertensive.*

M.D., Massachusetts.

Answer.—It is unusual to encounter "a number of boys" with systolic blood pressure readings over 150 mm. Hg, although blood pressure in the adolescent and young adult age periods is known to be labile. One group of observers found this degree of hypertension in less than 1% of 16-year-old boys (Graham, A. W.; Hines, E. A., Jr., and Gage, R. P.: Blood Pressures in Children Between the Ages of 5 and 16 Years, *Am. J. Dis. Child.* 69:203 [April] 1945). Emotional tension was apparently considered and ruled out as a factor causing the hypertension and tachycardia in the cases described in the query. Possibly a technical error was made in the determinations; for example, the use of a narrow "pediatric" cuff (4 to 9 cm. in width) would produce a false high reading, as would failure to wait for the appearance of sounds synchronous with each heart beat before recording the reading for systolic pressure. A recent statement by the American Heart Association is relevant to this point. (Recommendations for Human Blood Pressure Determinations by Sphygomanometers, *J. A. M. A.* **147**:632 [Oct. 13] 1951.) The finding of a pulse rate of 100 or more per minute among high school boys is also rare, and it supports the validity of the blood pressure determinations. Diastolic pressures of the subjects were not mentioned. Elevation of diastolic pressure is of equal or greater importance than the systolic pressure.

The normal blood pressure is best described as a range. The normal range for systolic pressures of adolescents and high school age youths is probably 105 to 135 mm. Hg and the normal diastolic range is 60 to 85 mm. Hg (Master, A. M.; Dublin, L. I., and Marks, H. H.: The Normal Blood Pressure Range and Its Clinical Implications, *J. A. M. A.* **143**:1464 [Aug. 26] 1950). Youths with blood pressures persistently higher than these values should be thoroughly examined to rule out possible causes of elevated blood pressure, including nephritis, coarctation of the aorta, polycystic and hypoplastic kidneys, pheochromocytoma, and Cushing's syndrome. Those youths with no demonstrable cause for elevated systolic pressure of 135 to 145 mm. Hg or diastolic pressure of 85 to 90 mm. Hg and who do not have cardiac enlargement could be permitted to take part in competitive athletics, provided there is medical supervision. It is reasonable, however, that, even when results of examination are normal, those boys with systolic pressure over 145 mm. Hg or diastolic pressure over 90 mm. Hg should not be permitted to engage in competitive sports. Cardiac enlargement and intracranial hemorrhage, the complications to be feared, are more likely to occur with the increased hypertension brought about by the emotional and physical stress of participation in competitive sports. These students should, however, be permitted to participate freely in a supervised program of physical education, in which there are opportunities for rest and the stress of competition is lacking. The mean pulse rate at this age is about 75 beats per minute. The upper limit of resting pulse rate for acceptance for competitive sports probably is 95 beats per minute.

Reported experience in the Army supports the opinion that transient hypertension of 150 mm. Hg or more and transient tachycardia of over 100 beats per minute appreciably increase the chances of hypertension developing in later years (Levy, R. L.; White, P. D.; Stroud, W. D., and Hillman, C. C.: Sustained Hypertension: Predisposing Factors and Causes of Disability and Death, *J. A. M. A.* **135**:77 [Sept. 13] 1947). When those two conditions occur together the likelihood of sustained hypertension later in life is even greater.

RHEUMATOID ARTHRITIS

To the Editor:—*Are gold salts and cortisone or corticotropin (ACTH) considered to have synergistic action? Is their simultaneous use advisable in treatment of rheumatoid arthritis?*

Robert J. Ralston, M.D., Holyoke, Colo.

Answer.—To date, opinions among rheumatologists regarding the combined use of cortisone or corticotropin (ACTH) and gold salts have been variable and tentative. Such a program of treatment has been termed "hopeful," "encouraging," "of no advantage," "disappointing," and even "possibly harmful." Use of the combination has been based on the premise that the relatively prompter, though generally temporary, antirheumatic effects of cortisone or corticotropin might be sustained by the slower acting gold salts; however, by definition, such an effect is supplementary rather than synergistic. In any event, the desired result is remission of the active, inflammatory reactions of the rheumatoid arthritis.

In the investigations of the possibility of remission from combined use of cortisone or corticotropin and gold salts, treatment with the gold salts has been started sometimes several weeks before, sometimes several weeks after, and sometimes concomitantly with, administration of the hormone and has then been continued beyond the period of hormonal treatment. A few instances have been cited that suggest that the hormonally induced remission has been prolonged by gold salts, but, in gen-

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The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

eral, no additive effect has been obtained in most patients receiving treatment with the combination. About 85 rheumatoid patients have received this combined treatment from at least seven investigators, according to the literature. Most of the data concerning these patients have been cited in discussions or other brief references. According to these reports, about 20% (18 patients) have had some prolongation of the hormonally induced antirheumatic effects, which may be attributable to the associated administration of gold salts; however, interpretation of the results in these patients is complicated by multiple and variable factors of both the disease and details of treatment. More experience with the combined use of these substances is required before such use could be considered advisable in the treatment of rheumatoid arthritis. In the meantime, if such a program is undertaken, it should be considered to be on an investigative basis.

TUBERCULOSIS, NEUROSIS, AND PREGNANCY

To the Editor:—*A 32-year-old housewife wants to have a baby. She has had minimal bilateral tuberculosis that has been arrested for over a year and a psychoneurosis for four years, which is greatly improved but still produces occasional annoying anxiety attacks. She is happily married and has one child, 5 years old. Her first attack of tuberculosis, at the age of 19, produced a small lesion confined to the right apex. This was arrested in six months, and she continued to be in good health until the birth of her child. Because of poor living conditions and malnutrition, which are not present now, tuberculosis spread to the left apex. The disease was arrested with bed rest. The patient is anxious about her health but is an intelligent woman and is capable of handling her household duties. The child is in good health, and the husband is intelligent and cooperative. Should the woman be allowed to become pregnant now, should she wait until a later date, or is a pregnancy inadvisable at any time?* *M.D., Pennsylvania.*

This inquiry was referred to two consultants, whose respective replies follow.—Ed.

Answer.—The pulmonary lesions of the patient under consideration should be inactive for at least two years before she becomes pregnant. Inactivity is defined by the National Tuberculosis Association as follows: "Lesions as observed in serial roentgenograms must be stable except for extremely slow shrinkage, and there must be no roentgenologic evidence of cavity. Symptoms of tuberculous origin must be absent. Sputum, if any, must be found negative for tubercle bacilli repeatedly, not only by concentration and microscopic examination but also by culture or animal inoculation. When the patient is not raising sputum or when there is any question concerning the authenticity or adequacy of expectorated sputum specimens, the fasting gastric contents or pulmonary secretions which have been aspirated from the tracheobronchial tree should be examined by culture or animal inoculation." Advice in the case in question must necessarily be influenced by the economic and social situation as well as by the degree of psychoneurosis involved.

Answer.—The problem is twofold, consisting of tuberculosis and mental instability. The tuberculosis should be thoroughly resolved before any thought is given to another pregnancy. Although detailed history and roentgenograms are not available, it seems that this is one of those smoldering types of disease that drags on for years and flares up on provocation. All foci should be adjudged arrested before pregnancy is permitted even if it requires a resection to extirpate the focus and assure an arrest at an earlier date than by ordinary rest treatment. This assurance is almost mandatory in view of the mental history. A complete arrest and cure of tuberculosis is now a relatively simple matter with present facilities for treating the disease. The problem of child-bearing will therefore depend on the severity and nature of the mental aberration. It may be only a temporary process due to transient worries and circumstances. There is mention of malnutrition that conceivably may be a contributing factor. If, after a proper diet and good mental environment for a year or two, there is no evidence of tuberculous activity she should be permitted to have another baby. If the mental disease is more serious or permanent or hereditary, it would be better not to think of pregnancy, even if the tuberculosis is entirely arrested.

IMMUNITY AGAINST COLDS

To the Editor:—*What procedures are there to build up a child's immunity against colds, bronchitis, and recurrent and relapsing respiratory infections?*

Charles L. Concklin, M.D., Corpus Christi, Texas.

Answer.—In an otherwise healthy child, aside from the ordinary rules of hygiene including adequate rest, food, and exercise in the open, there are no medical procedures to build up immunity. Isolation from persons with colds is important. Bronchiectasis may underlie repeated infections of the lower respiratory tract. Obstruction or other structural defects in the upper respiratory tract should be corrected. Enlarged tonsils rarely are a cause, and tonsillectomy has no effect on minor respiratory tract infections except repeated tonsillitis. Possible allergic factors must be eliminated. "Tonics," vitamins, antibiotics, and vaccines are of no value.

ARGYROSIS

To the Editor:—*Last December I instilled a few drops of mild silver protein (argyrol®) solution into a patient's lacrimal duct with a lacrimal syringe. I have done this on numerous occasions on other patients and have never had any trouble. This time as the argyrol was being injected, there seemed to be some extravasation into the soft tissue under the lower eyelid. I immediately stopped the injection. The discoloration under the eye caused by the mild silver protein has not disappeared and causes the appearance of a black eye. Can anything be done to cause the discoloration to disappear?*

M.D., Pennsylvania.

Answer.—Long continued use of organic silver salts in the form of mild and strong silver protein (argyrol® and protargol®) on mucous membrane and skin results in a permanent discoldration, which increases in depth as more silver is added. When introduced subcutaneously, the silver is deposited as an albuminate in collagenous tissues, in which it is reduced and a dirty gray-brown color is produced. The deposit excites no tissue reaction but remains permanently. The depth of discoloration depends on the amount of fluid injected and the proximity to the surface of the skin. Attempts to oxidize the silver and cause its absorption have been only partially successful. M. F. Weymann (*J. A. M. A.* **93**:1367 [Nov. 2] 1929) described a method by which he successfully treated conjunctival argyrosis. Argyrosis of the skin was successfully treated by Stillians and Lawless (*Arch. Dermat. & Syph.* **17**:153 [Feb.] 1928; *J. A. M. A.* **92**:20 [Jan. 5] 1929). The technique is to make up a 12% solution of sodium thiosulfate and a 2% solution of potassium ferricyanide. One part of the sodium thiosulfate solution is mixed with two parts of the potassium ferricyanide solution, and the mixture is injected intradermally, a platinum needle being used to prevent corrosion of the metal.

IRRADIATION IN BREAST CANCER

To the Editor:—*Has postoperative x-ray irradiation for cancer of breast any real therapeutic value?*

M.D., Rhode Island.

Answer.—The question cannot be answered with certainty, since the statistical evidence available is open to so much question. There have been claims of improvement of from 5% to as high as 15% in the five year survival figures in the treatment of cancer of the breast through the addition of postoperative x-ray therapy. The majority of surgeons do use postoperative x-ray therapy when the pathologist has returned the report of metastatic carcinoma of the axillary nodes, since it can be demonstrated that x-radiation is definitely cancerocidal for some cells. It seems the logical plan to follow since the question has not been more accurately settled.

JOURNALS ABSTRACTED IN THE CURRENT MEDICAL LITERATURE DEPARTMENT, MAY-AUGUST 1952

Titles have been listed or abstracts made of important articles in the following journals in the Current Literature Department of THE JOURNAL during the past four months. Any of the journals, except those starred, will be lent by THE JOURNAL to individual subscribers in Continental United States and Canada and to members of the American Medical Association and its student organization. No charge is made to members, but the fee for other borrowers is 15 cents in stamps for each periodical. Three journals may be borrowed at a time for a period not exceeding five days. Requests should be addressed to the Library of the American Medical Association. Thus most of these journals are accessible to the general practitioner.

*A. M. A. American Journal of Diseases of Children. Chicago.
*A. M. A. Archives of Dermatology and Syphilology. Chicago
*A. M. A. Archives of Industrial Hygiene and Occupational Medicine. Chicago.
*A. M. A. Archives of Internal Medicine. Chicago.
*A. M. A. Archives of Neurology and Psychiatry. Chicago.
*A. M. A. Archives of Ophthalmology. Chicago.
*A. M. A. Archives of Otolaryngology. Chicago.
*A. M. A. Archives of Pathology. Chicago.
*A. M. A. Archives of Surgery. Chicago.
Acta chirurgica scandinavica. Stockholm.
Acta haematologica. Basel.
Acta medica scandinavica. Stockholm.
Acta orthopaedica scandinavica. Copenhagen.
Acta paediatrica. Stockholm.
Acta pathologica et microbiologica scandinavica. Copenhagen.
Acta tuberculosea belgica. Brussels.
Acta tuberculosea scandinavica. Copenhagen.
American Heart Journal. St. Louis.
American Journal of Clinical Pathology. Baltimore.
American Journal of Digestive Diseases. Fort Wayne, Ind.
American Journal of Human Genetics. Baltimore.
American Journal of Hygiene. Baltimore.
American Journal of the Medical Sciences. Philadelphia.
American Journal of Medicine. New York.
American Journal of Obstetrics and Gynecology. St. Louis.
American Journal of Ophthalmology. Chicago.
American Journal of Pathology. Ann Arbor, Mich.
American Journal of Physiology. Baltimore.
American Journal of Psychiatry. New York.
American Journal of Psychotherapy. New York.
American Journal of Public Health. New York.
American Journal of Roentgenol. and Radium Therapy. Springfield, Ill.
American Journal of Surgery. New York.
American Journal of Syphilis, Gonor. and Venereal Diseases. St. Louis.
American Journal of Tropical Medicine and Hygiene. Baltimore.
American Practitioner and Digest of Treatment. Philadelphia.
American Review of Tuberculosis. New York.
American Surgeon. Atlanta, Ga.
Anesthesiology. New York.
Angiology. Baltimore.
Annales de dermatologie et de syphiligraphie. Paris.
Annales de medecine. Paris.
Annals of Allergy. Minneapolis.
Annals of Internal Medicine. Lancaster, Pa.
Annals of Otology, Rhinology and Laryngology. *St. Louis.*
Annals of Physical Medicine. London.
Annals of the Rheumatic Diseases. London.
Annals of Surgery. Philadelphia.
Annals of Western Medicine and Surgery. *Los Angeles.*
Antibiotics and Chemotherapy. Washington, D. C.
Aparato respiratorio y tuberculosis. *Santiago.*
Archives of Disease in Childhood. *London.*
Archives des maladies du coeur et des vaisseaux. Paris.
Archives of Physical *Medicine. Chicago.*
Archivos españoles de urología. Madrid.
Archivos médicos de Cuba. Havana.
Archivos de pediatría del Uruguay. Montevideo.
Archivum chirurgicum neerlandicum. *Arnhem.*
Arizona Medicine. Phoenix.
Arquivos de neuro-psiquiatria. São Paulo.
Australian Journal of Experimental Biology and Medical Science. Adelaide.
Australian and New Zealand Journal of *Surgery. Sydney.*
Beiträge zur klinischen Chirurgie. Munich.
Belgisch tijdschrift voor geneeskunde. Leuven.
Blood. New York.
Boletín del colegio médico de Camaguey. Camaguey.
Boletín del consejo general *de colegios* médicos de España. Madrid
Brain. London.
British Heart Journal. London.
British Journal of Cancer. London.
British Journal of *Dermatology. London.*
British Journal of Experimental Pathology. *London.*
British Journal of Industrial Medicine. *London.*
British Journal of Ophthalmology. London.
British Journal of Plastic Surgery. Edinburgh.
British Journal of Radiology. London.
British Journal of Surgery. Bristol.
British Journal of Tuberculosis. London.
British Journal of Urology. London.
British Journal of Venereal Diseases. London.
British Medical Journal. London.
Bulletin of the Johns Hopkins Hospital. Baltimore.
Bulletin of the New York Academy of *Medicine.* New York.
California Medicine. San Francisco.
Canadian Journal of Medical Sciences. Ottawa.
Canadian Journal of Public Health. Toronto.
Canadian Medical Association Journal. Montreal.
Cancer Research. Chicago.
Chirurg. Berlin-Heidelberg.
Circulation. New York.
Clinica pediatrica. Bologna.
Connecticut State Medical Journal. Hartford.
Delaware State Medical Journal. Wilmington.
Deutsche medizinische Wochenschrift. Stuttgart.
Deutsche Zeitschrift für Verdauungs- und Stoffwechselkrankheiten. Leipzig.
Deutsches medizinisches Journal. Berlin.
Diabetes. New York.
Die Medizinische. Stuttgart.
Diseases of Chest. Chicago.
East African Medical Journal. Nairobi.
Edinburgh Medical Journal. Edinburgh.
Electroencephalography and Clinical Neurophysiology. Boston.
Endocrinology. Springfield, Ill.
Fortschritte auf dem Gebiete der Röntgenstrahlen vereinigt mit Röntgenpraxis. Stuttgart.
Gastroenterology. Baltimore.
Geburtshilfe und *Frauenheilkunde. Stuttgart.*
Geriatrics. Minneapolis.
Giornale di clinica medica. Parma.
Giornale italiano *di anestesiologia.* Turin.
Giornale italiano di chirurgia. Naples.
Glasgow Medical Journal. Glasgow.
GP (Journal of American Academy of General Practice). Kansas City, Mo.
Gynaecologia. Basel.
Hawaii Medical Journal. Honolulu.
Helvetica medica acta. Basel.
Helvetica paediatrica acta. Basel.
Hoja tisiológica. Montevideo.
Illinois Medical Journal. Chicago.
Industrial Medicine and Surgery. Chicago.
International Journal of Leprosy. New Orleans.
Irish Journal of Medical Science. Dublin.
Journal of Allergy. St. Louis.
Journal of American Medical Women's Association. Nashville, Tenn.
Journal of Applied Physiology. Washington, D. C.
Journal of Arkansas Medical Society. Fort Smith.
Journal of Aviation Medicine. St. Paul.
Journal of Bacteriology. Baltimore.
Journal of the Bowman Gray School of Medicine. Winston-Salem, N. C.
Journal of Bone and Joint Surgery. Boston.
Journal of the Canadian Association of Radiologists. Montreal.
Journal de chirurgie. Paris.
Journal of Clinical Endocrinology and Metabolism. Springfield, Ill.
Journal of Clinical Investigation. Cincinnati.
Journal of Clinical Pathology. London.
Journal of Endocrinology. London.
Journal of Experimental Medicine. New York.
Journal of the Florida Medical Association. Jacksonville.
Journal of Gerontology. St. Louis.
Journal of Immunology. Baltimore.
Journal of the Indiana State Medical Association. Indianapolis.
Journal of Infectious Diseases. Chicago.
Journal international de chirurgie. Brussels.
Journal of International College of Surgeons. Chicago.
Journal of Investigative Dermatology. Baltimore.
Journal of the Iowa State Medical Society. Des Moines.
Journal of the Kansas Medical Society. Topeka.
Journal of the Kentucky State Medical Association. Bowling Green.
Journal of Laboratory and Clinical Medicine. St. Louis.
Journal of Laryngology and Otology. London.
Journal of the Maine Medical Association. Portland.
Journal of the Medical Association of the State of Alabama. Montgomery.
Journal of the Medical Association of Georgia. Atlanta.

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* Cannot be lent.

Journal of the Medical Society of New Jersey. Trenton.
Journal of Mental Science. London.
Journal of the Michigan State Medical Society. Lansing.
Journal of the Missouri State Medical Association. St. Louis.
Journal of National Cancer Institute. Washington, D. C.
Journal of Nervous and Mental Disease. New York.
Journal of Neurology, Neurosurgery and Psychiatry. London.
Journal of Neuropathology and Experimental Neurology. Baltimore.
Journal of Neurophysiology. Springfield, Ill.
Journal of Neurosurgery. Springfield, Ill.
Journal of Nutrition. Philadelphia.
Journal of Obstetrics and Gynaecology of the British Empire. Manchester.
Journal of the Oklahoma State Medical Association. Oklahoma City.
Journal of Oral Surgery. Chicago.
Journal of Pathology and Bacteriology. Edinburgh.
Journal of Pediatrics. St. Louis.
Journal of Pharmacology and Experimental Therapeutics. Baltimore.
Journal of the Philippine Medical Association. Manila.
Journal of Physiology. London.
Journal of the South Carolina Medical Association. Florence.
Journal of the Tennessee State Medical Association. Nashville.
Journal of Thoracic Surgery. St. Louis.
Journal of Urology. Baltimore.
Journal-Lancet. Minneapolis.
Klinische Monatsblätter für Augenheilkunde und für augenärztliche Fortbildung. Stuttgart.
Klinische Wochenschrift. Berlin-Heidelberg.
Lancet. London.
Laryngoscope. St. Louis.
Lyon chirurgical. Lyon.
Maandschrift voor kindergeneeskunde. Leyden.
Maryland State Medical Journal. Baltimore.
Medical Annals of the District of Columbia. Washington.
Medical Journal of Australia. Sydney.
Medicina clínica. Barcelona.
Medicine. Baltimore.
Medizinische Monatsschrift. Stuttgart.
Mental Hygiene. Albany, N. Y.
Military Surgeon. Washington, D. C.
Minerva medica. Turin.
Minerva pediatrica. Turin.
Minnesota Medicine. St. Paul.
Münchener medizinische Wochenschrift. Munich.
Nebraska State Medical Journal. Lincoln.
Nederlandsch tijdschrift voor geneeskunde. Amsterdam.
Nervenarzt. Heidelberg.
Neurology. Minneapolis.
New England Journal of Medicine. Boston.
New Orleans Medical and Surgical Journal. New Orleans.
New York State Journal of Medicine. New York.
New Zealand Medical Journal. Wellington.
Nordisk medicin. Stockholm.
North Carolina Medical Journal. Winston-Salem.
Northwest Medicine. Seattle.
Ohio State Medical Journal. Columbus.
Pediatrics. Springfield, Ill.
Pédiatric. Lyon.
Pennsylvania Medical Journal. Harrisburg.
Physiological Reviews. Baltimore.
Plastic & Reconstructive Surgery. Baltimore.
Policlinico (Med. Sect.). Rome.
Policlinico (Pract. Sect). Rome.
Policlinico (Surg. Sect.). Rome.
Portugal medico Porto.
Postgraduate Medicine. Minneapolis.
Practitioner. London.
Prensa médica argentina. Buenos Aires.
Presse médicale. Paris.
Proceedings of Royal Society of Medicine. London.
Proceedings of the Society for Experimental Biology and Medicine. Utica, N. Y.
Proceedings of Staff Meetings of Mayo Clinic. Rochester, Minn.
Psychiatric Quarterly. Utica, N. Y.
Psychiatry. Washington, D. C.
Psychoanalytic Quarterly. Albany, N. Y.
Psychoanalytic Review. Albany, N. Y.
Psychosomatic Medicine. New York.
Public Health Reports. Washington, D. C.
Quarterly Bulletin of Sea View Hospital. Staten Island, N. Y.
Quarterly Journal of Medicine. Oxford.
Quarterly Journal of Studies on Alcohol. New Haven, Conn.
Radiology. Syracuse, N. Y.
Review of Gastroenterology. New York.
Revista de la Asociación médica argentina. Buenos Aires.
Revista chilena de pediatría. Santiago.
Revista española de las enfermedades del aparato digestivo y de la nutrición. Madrid.
Revista española de pediatría. Zaragoza.
Revista española de tuberculosis. Madrid.
Revista de la Facultad de ciencias médicas de la Universidad Nacional de Córdoba. Córdoba.
Revista do Hospital das clínicas. São Paulo.
Revista médica de Chile. Santiago.
Revista de medicina e cirurgia de São Paulo. São Paulo.
Revista de obstetricia y ginecología. Caracas.
Revista paulista de medicina. São Paulo.
Rhode Island Medical Journal. Providence.
Riforma medica. Naples.
Rivista di clinica pediatrica. Florence.
Rocky Mountain Medical Journal. Denver.
Schweizerische medizinische Wochenschrift. Basel.
Science. Washington, D. C.
Semaine des hôpitaux de Paris. Paris.
South African Medical Journal. Cape Town.
South Dakota Journal of Medicine and Pharmacy. Sioux Falls.
Southern Medical Journal. Birmingham, Ala.
Southwestern Medicine. El Paso, Texas.
Stanford Medical Bulletin. San Francisco.
Strahlentherapie. Munich.
Strasbourg medical. Strasbourg.
Surgery. St. Louis.
Surgery, Gynecology and Obstetrics. Chicago.
Texas Reports on Biology and Medicine. Galveston.
Texas State Journal of Medicine. Fort Worth.
Thorax. London.
Tidsskrift for den norske lægeforening. Oslo.
Transactions of the Royal Society of Tropical Medicine and Hygiene. London.
Transactions of the Southeastern Section of the American Urological Association. Memphis.
Tubercle. London.
Tuberkulosearzt. Stuttgart.
Ugeskrift for læger. Copenhagen.
United States Armed Forces Medical Journal. Washington, D. C.
Union médicale du Canada. Montreal.
Urologia. Treviso.
Virginia Medical Monthly. Richmond.
West African Medical Journal. Nigeria.
Western Journal of Surgery, Obstetrics and Gynecology. Portland, Ore.
West Virginia Medical Journal. Charleston.
Wiener medizinische Wochenschrift. Vienna.
Wisconsin Medical Journal. Madison.
Yale Journal of Biology and Medicine. New Haven.
Zeitschrift für Geburtshilfe und Gynäkologie. Stuttgart.
Zeitschrift für Hygiene und Infektionskrankheiten. Heidelberg.
Zeitschrift für Immunitätsforschung und experimentelle Therapie. Jena.
Zeitschrift für Krebsforschung. Munich.
Zeitschrift für Orthopädie und ihre Grenzgebiete. Stuttgart.
Zeitschrift für Urologie. Leipzig.
Zentralblatt für Chirurgie. Leipzig.
Zentralblatt für Gynäkologie. Leipzig.

SUBJECT INDEX

This is an index to all the reading matter in THE JOURNAL. In the Current Medical Literature Department only the articles which have been abstracted are indexed.

The letters used to explain in which department the matter indexed appears are as follows: "BI," Bureau of Investigation; "E," Editorial; "C," Correspondence; "ab," abstracts; the asterisk (*) indicates an original article in THE JOURNAL.

This is a subject index and one should, therefore, look for the subject word, with the following exceptions: "Book Notices," "Deaths," "Medicolegal Abstracts" and "Societies" are indexed under these titles at the end of the letters "B," "D," "M," and "S." Matter pertaining to the Association is indexed under "American Medical Association." The name of the author, in brackets, follows the subject entry.

For the author index see page 1745.

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Acad.—Academy
Am.—American
A.—Association
Coll.—College
Commn.—Commission
Comm.—Committee
Conf.—Conference
Cong.—Congress
Dist.—District
Div.—Division
Found.—Foundation
Hosp.—Hospital
Indust.—Industrial
Inst.—Institute
Internat.—International
M.—Medical
Med.—Medicine
Nat.—National
Pharm.—Pharmaceutical
Phys.—Physicians
Soc.—Society
Surg.—Surgery
Surgs.—Surgeons
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